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Depression, Volition, and Death: The Effect of Depressive Disorders on the Autonomous Choice to Forgo Medical Treatment

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DEPRESSION, VOLITION, AND DEATH: THE EFFECTS OF DEPRESSIVE DISORDERS ON THE AUTONOMOUS CHOICE TO FORGO MEDICAL TREATMENT

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TABLE OF CONTENTS

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In doubt his Mind or Body to prefer, Born but to die, and reas'ning but to err;
Alike in ignorance, his reason such, Whether he thinks to little, or too much:
Chaos of Thought and Passion, all confus'd; Still by himself abus'd, or disabus'd;
Created half to rise, and half to fall; Great lord of all things, yet a prey to all;
Sole judge of Truth, in endless error hurl'd: The glory, jest, and riddle of the world!

-Alexander Pope
An Essay on Man

It is a wonderful feeling to recognize the unity of a complex of phenomena that to direct observation appear to be quite separate things.

-Albert Einstein
Letter to Marcel Grossman

The mind is inherently embodied. Thought is mostly unconscious. Abstract concepts are largely metaphorical. These are three major findings of cognitive science. More than two millennia of a priori philosophical speculation about these aspects of reason are over. Because of these discoveries, philosophy can never be the same again…

Philosophical sophistication is necessary if we are to keep science honest. Science cannot maintain a self-critical stance without a serious familiarity with philosophy and alternative philosophies. Scientists need to be aware of how hidden a priori philosophical assumptions can determine their scientific results. This is an important lesson to be drawn from the history of first-generation cognitive science, where we saw how much analytic philosophy intruded into the initial conception of what cognitive science was to be. On the other hand, philosophy, if it is to be responsible, cannot simply spin out theories of mind, language, and other aspects of human life without seriously encountering and understanding the massive body of relevant ongoing scientific research. Otherwise, philosophy is just storytelling, a fabrication of narratives ungrounded in the realities of human embodiment and cognition. If we are to know ourselves, philosophy needs to maintain an ongoing dialogue with the sciences of mind.

- George Lakoff and Mark Johnson
Philosophy in the Flesh

GENERAL INTRODUCTION

The Nature and Purpose of this Dissertation

Since its inception, medical ethics has concerned itself with balancing several key concepts – the patient's best interest, both psychosocial and medical; the patient's legal rights and autonomy; the authenticity of the patient's decision, i.e., narrative concerns that the patient's choice be reflective of her values, etc. As is the case with any pluralistic system, these concepts are complementary at times and conflicting at times. Significant efforts to determine just how to proceed in any given case result, both in academic circles, in which theories clash and value structures rise and fall, as well as in clinical cases, in which academic language gives way to clinical context and lives hang in the balance.

These values have been stressed in different manners at different times – before the advent of medical ethics as a field in itself, the physician-patient relationship was defined in paternalistic terms. The physician, being the source of medical knowledge and prognostic wisdom, had an air of authority which one dared not question. Decisions were top-down, in the manner of a general commanding his soldiers, and were rarely questioned. While the patient's concerns were counted, what was in their medical best interest carried the day.

This perspective evolved over time, and a different model of physician-patient interaction emerged. Patient advocacy found legal and philosophical recourse in autonomy models which stressed the ability of a patient to choose the treatment she wanted, and patients' rights became more than an ephemeral concept. Decisions proceeded on egalitarian terms, in which the patient and the treatment team reached an accord – the patient's medical best interest was now simply one factor of many to consider, along with religious principles, social consequences and contexts, and legal rights to refuse undesired treatment. The general was stripped of his rank –

choice and responsibility were no longer top-down considerations. A new era of patient autonomy was ushered in, changing the face of medical decision-making.

This gave rise to ethics committees and consultations – a treatment team would find itself conflicted as to how to proceed, and brought in individuals with special knowledge of philosophy, theology, and law to facilitate a decision. The case would be evaluated, the patient and/or her family would be interviewed, and non-binding recommendations were made which helped the treatment team progress. Sometimes they clarified communication problems in prognosis, sometimes they led to palliative care, sometimes they resolved a family conflict. The welfare of the patient was at the forefront, but was no longer understood as being solely a medical issue. Instead, a shift occurred – the patient became an evolving story, and the choices made were meant to reflect the pattern of that patient's life.

These concepts of autonomy and authenticity have dominated ethical thought for several decades, and have been given significant, if not complete, weight in many theories. Autonomy is seen by many as a deontological norm – an absolute right and duty in some models, a *prima facie* duty in others. Its value and moral weight are understood as being *a priori* – it is not contingently valuable or worthy simply as a means to some other end.

The purpose of this dissertation is to explore this concept of autonomy, and to see how it is modified by knowledge from multiple fields. Philosophy certainly offers compelling accounts and definitions, but a fundamental question arises: what does the concept mean in light of what we have learned from fields like cognitive psychology and psychiatry? Philosophy and ethics have debated 'modifiers of the voluntary' for a long time, but these concepts of coercion generally are predicated on conscious awareness and experience. A more complete model of cognition notes that significant thought processes occur at levels which we are only beginning to

understand. These influences are non-conscious: they stem from a collection of processes outside of our conscious awareness. How, therefore, can we exercise control over or appreciate the influence of elements of which we aren't aware? Many models fiat the ability of the moral agent to choose amongst alternatives – these models seem to be less compelling in light of what we know and understand from other disciplines. In fact, the more we learn about the brain, the more homuncular they seem – it is almost as if they argue for a little man sitting in our brains, selectively choosing what will influence us to act. These models are untenable – any conception of autonomy must include an appreciation for cognitive elements outside cognition, which potentially bias us in ways that are inauthentic. In essence, the concern of this dissertation is whether it is possible for depressive disorders – a spectrum of cognitive dysfunction which is chronically underdiagnosed in contemporary medicine – to so influence a person as to skew their decision-making process. In upholding choices that may be skewed, we undercut any meaningful sense of autonomy. We destroy that which we would protect in a decision which may be the last choice the patient ever makes. If we claim to be concerned about the welfare of our patients, this cannot stand.

Philosophical Arguments Against Homuncular Autonomy

The first avenue of approach to understanding autonomy concerns two related philosophical doctrines: reductionism and determinism. Reductionism in this sense refers to the modeling of the structural basis of cognition. It is almost a truism to argue that cognition needs a platform upon which to operate – there must be some physical *stuff* there that is employed in thought. To the best of our available scientific findings, there is no disembodied thought. Whether cognition occurs in terms of neurons and neural pathways or in terms of silicon chips and copper wiring, there is *something* there which allows us to think, emote, rationalize,

contextualize, and perform myriad other processes which guide and influence our everyday experience. As such, any cognitive model must include *physical* elements – the question is still open as to whether we radically reduce thought to neurochemistry and cell interactions, or whether we account for cognition in pluralistic terms – proposing that there are multiple levels of cognition that are mutually influential. Several philosophical arguments are examined, and elements of them are championed or rejected, yielding a sense of reductionism which is pluralistic and materialistic. But this has necessary consequences and raises further questions.

If we accept a materialistic account of thought and volition – introduced by the experiments of Benjamin Libet – we necessarily raise the question of determinism. Physical systems have laws they must follow; laws of cause and effect are axiomatic in the sciences, and are requisite assumptions in accounting for the phenomena of will and volition. We *feel* that we choose between options; we *deliberate* in making a choice; we *explore* the consequences of given alternatives prior to exercising our choice. These experiences, however, have neurological bases and follow certain laws, governing possibilities and thought pathways. Further, there are elements of volition that *demonstrably* are not within our control or conscious awareness. In fact, *choice* occurs fractions of a second *before we are even aware of having chosen*. As in the discussion of reductionism, several ideas are considered, critiqued, and upheld or rejected. What emerges is a sense of underdetermination – the idea that there are several causal factors in choice that individually may not determine action, but are essentially additive. When a threshold is reached, the individual causal factors force a decision – the process of which we are not necessarily aware. These additive causal factors in cognition stem from automatic psychological processes, the essence of chapter two.

Psychological Automaticity, Backstage Cognition, and Cognitive Heuristics

The second prong of the argument against homuncular autonomy concerns the automatic elements of human cognition. These processes by definition are outside of our awareness, and may only be made evident in discussion with others. Information is taken in and processed in a variety of ways, employing a variety of heuristics – cognitive 'rules of thumb'. Deliberation and decision-making occur in light of these heuristics, which causes some concern. Cognitive *heuristics* by definition are not *algorithms* – they are not detailed plans that necessarily yield correct decisions or interpretations of situations or data.

Further, these heuristics are very common – the phenomena of being on 'autopilot' is not limited to specific circumstances. Automaticity is a generalized phenomena; it can occur in a variety of situations or contexts, influencing not only how we drive to work but how we understand information, how we act, how we behave towards strangers, friends, family, and loved ones. Automaticity factors into the thought processes we have all the time – they are refined and strengthened over time unless challenged. As such, our biases become deeply ingrained, and may prevent us from seeing situations objectively or accurately. Autonomy is only meaningful if we can genuinely choose between options – as such, cognitive biases must be identified, addressed, and challenged if they are mistaken. Preserving choice for the sake of choice is meaningless unless some effort is made to prevent error.

This discussion yields a deeper problem, however – at issue is whether there is even such thing as a 'rational' process, unless we simply define the term as acting out the determined pathways of normal cognition. Normal, however, is just as problematic – what defines normal and abnormal? Unless one is discussing clear cases of psychosis and dysfunction, the issue of normalcy is not so black and white. If we cannot clearly define normal and abnormal – not

necessarily the point of this chapter, but an important ancillary issue – how are we to determine rational and irrational? Further, we chronically underdiagnose psychiatric disorders like depression – these are causal factors of critical importance, as they can have biological bases which directly impact cognition at several reductive levels. The prevalence of these disorders is the focus of chapter three.

The Spectrum of Depressive Disorders and Comorbidity in Common Medical Conditions

Chapter three notes the diagnostic criteria for commonly occurring depressive disorders, exploring their biological basis. We can understand psychiatric dysfunction in both psychosocial as well as biochemical terms. We do not look for neurotransmitter dysfunctions when someone loses his job or spouse – these have principally environmental or *exogenous* etiologies, and while pharmacological interventions can help, in general we seek to offer talk therapy approaches and help the patient develop coping skills. There can be, however, biological dysfunctions, leading to *endogenous* depressions – monoamine deficiencies, structural abnormalities in the frontal cortices, limbic system, hypothalamic-pituitary-adrenocortical pathways, hippocampal degeneration, etc.

The chapter then shifts to discussions of five common medical conditions: Alzheimer's disease, stroke, multiple sclerosis, Parkinson's disease, and cancer. All of these have pathologies potentially affecting important structures in cognition and affective (emotional) regulation – they may produce significant rates of depression that go undiagnosed, due to unfamiliarity with the diagnostic criteria or symptom masking (the physical signs of depression are explained by the physical signs of the underlying medical illness). These are not the only conditions that give rise to depression – rather they simply note the five conditions I experienced while on clinical rounds at St. Francis Health System and while working on the adult and geriatric behavioral health units

at Mercy Hospital North Shore Campus. Other conditions are mentioned that have significant rates of depression. The overall point of the chapter is that psychiatric comorbidities are much more common than is presently diagnosed, and that these can fundamentally affect cognitive processing, due to their effects at structural and psychosocial levels of cognitive reduction. In fact, depressive disorders have produced their own heuristics, discussed in chapter four.

Depressive Heuristics and Homuncular Autonomy Models

Chapter four crystallizes the argument thus far – autonomy and cognition are seen to be easily influenced if not determined outright by the physical condition of the cognizing agent. New cognitive pathways can be introduced by the depressive disorders – new heuristics that can automatically affect the agent's cognition, outside of her awareness, rendering her decisions inauthentic and non-autonomous. There is some debate concerning this, however. Alloy and Abramson have championed a heuristic called 'depressive realism', in which the mild to moderately depressed person actually makes more realistic decisions than individuals at their baseline or severely depressed. Ironically, a little depression may be a good thing, if we are looking at purely objective decision-making. However, this view has been challenged by Beck's argument for a depressive bias – an overly negative and pessimistic view of the world that is more unrealistic than everyday cognition and choice.

Autonomy models in the philosophical and ethical literature have a tendency to avoid these issues – not by answering them or accounting for them, but rather by dismissing or ignoring them. They offer a homuncular sense of autonomy, and elect not to consider the significant arguments that our vaunted 'rationality' and 'autonomy' may not necessarily be what they are cracked up to be. As in previous chapters, several popular arguments will be explored and critiqued. What emerges is a model of autonomy not just informed by cognitive psychology,

but dependent upon it. Ultimately, autonomy, it is argued, ought not be such an absolute principle – it seems that in an effort to move away from the paternalistic model so prevalent in historical medicine, we have overcorrected, and have improperly deified a nebulous sense of autonomy. A proper sense of autonomy is much more deterministic and less 'rational' than modern models suggest. As such, greater care is necessary in assessing competence to forgo treatment – quite simply, current models allow for more bad decisions with fatal consequences, a reality antithetical with the stated and implied purposes of ethics in medicine. A means of addressing this reality is proposed in the final chapter.

Ethical Casuistry in Decision-Making

The final chapter presents nine case studies, creating a metric by which patient presentation can be judged and assessed. These cases are drawn from clinical experiences and narrative accounts of depression; they are designed to offer the clinician clear instances of compromised autonomy and authentic autonomy, along with a continuum of cases in between, so as to facilitate decision-making, both for the clinician and the patient.

Overall, it is hoped that this dissertation will serve not only academic and theoretical purposes, such as integrating germane elements across disciplines into a unified model of physician-patient decision-making, but also clinical purposes, such as helping clinicians in both recognizing the prevalence of depressive disorders outside of psychiatric floors as well as assisting treatment teams and ethics consult teams in assessing whether it is appropriate for a patient to forgo treatment. The issue is quite complex, and given its severity, ideologically simplistic models must necessarily be rejected. If we genuinely care for our patients, we ought to help them reach meaningful choices, instead of fiating an empty and ill-defined autonomy.

CHAPTER ONE: WE'RE NOT AS FREE AS WE THINK WE ARE – REDUCTIONIST AND DETERMINIST ONTOLOGIES IN HUMAN COGNITION

The discussion of cognition and our experience of the phenomenon begins in philosophy. Epistemology and ontology have wrestled with these foundational issues since at least the time of the ancient Greeks and it remains a fertile area of discussion. It is a truism to suggest that we all have unique perspectives on the world, and that we are the sum total of – and possibly more than – our collective experiences, memories, beliefs and desires. But the fundamental question is where do these phenomena come from? Is our sense of self based on anything substantial, or is it an accidental epiphenomenon of a complex neural network? If it is something expressible in terms of simpler relationships, is the nature of our cognition fundamentally determined by the architecture of our individual neural network?

These are critical questions in epistemology, ontology, and ethics. As the overall purpose of this dissertation is to explore the interaction of depressive disorders and our thought processes in the choice to forgo medical treatment, it is necessary to understand just how much of our conscious experience is genuinely up to us. Autonomy becomes a very different concept if it is essentially an acting out of underlying deterministic forces, instead of the more radical agentic choice – an almost homuncular view of a little executive entity picking and choosing amongst causal forces. The model that emerges from the literature suggests that both reductionism and determinism are more accurate foundations of our subjective experience than their alternatives, and each will be explored in turn. Let me establish at the outset that this dissertation is not simply a defense of determinism – in the next two chapters, I will repeatedly argue that human cognition and choice are heavily influenced by deterministic elements which are mediated or modified by conscious processes. I will reject outright models that propose a radical indeterminism as factually incorrect in light of what neuroscience and cognitive psychology

demonstrate; such models are frequently found in 'folk models', which will be explored as they become relevant. The model of human agency that I will defend throughout suggests that cognition begins as a deterministic, non-conscious process, which, if not *consciously challenged and explored*, will simply act out the responses determined by environmental and cognitive stimuli. This is an example of what the literature refers to as 'underdeterminism' – that a causal threshold can be reached, yielding determined action, if there is no voluntary break in the process. As a further necessary caveat, and to avoid potential equivocation, I will use the terms 'non-conscious' and 'un-/sub-/preconscious' interchangeably, as they both refer to processes outside our normal conscious experience. Ultimately, the stage will be set for an exploration of cognition in chapter two – we will see that agency and autonomy are not necessarily as free as we make them out to be.

REDUCTIONISM

Before discussing reductionism, it is useful to place it in context. The concept of mind and agency is influenced by the classic philosophical debate concerning the mind and body. At its heart is the question of whether there is some non-physical element to human existence and cognition – whether our 'mind' is something fundamentally different from our body.[1] John Searle sums up the crux of the issue:

> We think of ourselves as *conscious, free, mindful, rational* agents in a world that science tells us consists entirely of mindless, meaningless, physical particles. Now, how can we square these two conceptions? How, for example, can it be the case that the world contains nothing but unconscious physical particles, and yet that it also contains consciousness? How can a mechanical universe contain intentionalistic human beings – that is, human beings that can represent the world to themselves? How, in short, can an essentially meaningless world contain meanings?[2]

[1] It is well beyond the purpose of this dissertation to properly address the mind/body debate. Such an effort would require significantly more space than is available. As such, I will limit the discussion to the comments of John Searle, C. Wade Savage, and Michael Rugg.
[2] John Searle, *Minds, Brains, and Science* (Cambridge: Harvard University Press, 1984), 13.

Searle argues that all mental phenomena have underlying neural processes.[3] However, he continues that the model we ought to have of the brain is not purely an element of 'microproperties' (i.e., properties of individual particles or neurons); rather, we ought to understand that there are underlying microproperties and overarching 'macroproperties' (i.e., emergent properties or features that only apply to the larger structure).[4] Macroproperties, for instance, would be the liquidity of water – we cannot "reach into this glass of water, pull out a molecule and say: 'This one's wet.'"[5] Consciousness or self-concept are these kinds of macroproperties – they are not properties of an individual neuron, they can only be understood at a higher level of organization. Thus, we can account for mental properties, but we recognize their contingency upon the underlying structure. We cannot have 'wet' in isolation, just as we cannot have 'consciousness' in isolation. Further, these mental properties can have causal force, but are so in light of their underlying structures (microproperties).[6] Understanding of micro- and macroproperties should not be understood as dualism in the physical/nonphysical sense, however – mental states are still the result of physical states.[7]

A recurrent challenge to materialism and reductionism concern dualist claims of the innate separation of the mind and body. Stemming from Descartes, the arguments essentially claim that there is something non-physical which unites conscious experience into memory, self-awareness, and identity. Patricia Churchland argues that this dualistic claim rests upon quite precarious grounds, for several reasons. First, the dualist has a significant burden to prove – how does the non-physical interact with the physical? Unless there is some kind of physical medium

[3] Searle, 19.
[4] Searle, p. 20-1
[5] Searle, 22.
[6] Searle, 26.
[7] Searle, p. 26-27

through which it operates, there is no real means by which a non-physical entity can influence a physical object.[8] Descartes' efforts to answer this question by the soul influencing animal spirits in the pineal gland come up wanting – after all, these animal spirits were physical objects, too, so the same conundrum occurs. Second, personal phenomena like cognition, consciousness, moods, moral and religious feelings are suggested to be autonomous subjective properties. The problem, however, is that each of these are affected by neuropharmacology, suggesting a physical basis for each.[9] The phenomena of subjective experience is discussed below. Third, any claim that a dualist makes regarding the unifying properties of the non-physical mind can also be made by the materialist or reductionist. Both positions are faced with a conundrum, however, in that neither position is iron-clad. To date, neither the materialist nor the dualist position can claim an absolute accounting of the unifying properties of mind – there is a standoff, which will only be resolved by research.[10] Despite her noted objections, Churchland does stress that we ought not reject the dualist hypothesis outright; however, as she cautions that future research may provide a means of the non-physical influencing the physical.[11] We will return to explicit challenges to reductionism following a few definitional and explanatory comments below.

C. Wade Savage takes this issue further, arguing that nonphysical causes ought not be a part of science – the argument against such non-physical causes illustrates the conceptual difficulties in physical/non-physical interaction: one can neither prove nor disprove the existence of non-physical causes or objects (e.g., the 'mind', a deity, etc.).[12] Such arguments cannot provide empirical data – how do we prove that God caused a natural disaster, rather than plate

[8] Patricia Smith Churchland, *Neurophilosophy* (Cambridge: The MIT Press, 1998), 318.
[9] Churchland, *Neurophilosophy*, 319.
[10] Churchland, 322.
[11] Churchland, 320.
[12] C. Wade Savage, "An Old Ghost in a New Body," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 125-53, p. 150-1.

tectonics? How do we demonstrate that an immaterial 'mind' caused a specific neural pathway to activate? Both questions are fundamentally unanswerable. In light of this, this dissertation argues along materialist lines – we can see neurons firing and following pathways, we can trace patterns of electrical activity, in short, we can see the physical evidence supporting materialist theories. Michael Rugg argues that materialist theories allow for functional explanations of cognition – accounting for cognition in terms of interactive structure is an empirically testable and verifiable model, and allows us to interpret and integrate future physiological findings.[13] These models are necessarily reductive, a concept to which we will now turn.

Definitions

As the overall argument of this dissertation is that depressive disorders can potentially give rise to conscious and unconscious cognitive bias in decision-making, it is necessary to discuss the means by which a depressive illness can affect a patient. At the heart of the matter is how mental events are to be understood. 'Choice' ostensibly refers to our reflection upon and selection between different options based upon our internal state, values, and beliefs. All of these concepts are in some manner related to physical structure – while as of this writing we have only a general idea about the neural mechanisms underlying our higher cognitive functions, it remains an axiom of cognitive science that there must be *some* physical structure underlying them. In essence, we have to have a suitably complex physical structure that *actually does the thinking*. As such, it is necessary to explore the interaction of the mental and the physical – whether, in fact, there is actually a divide between them.

As was noted in the outset of this dissertation, the underlying assumption of cognition and autonomy is reductionistic, but this in itself is unclear – there are a variety of understandings

[13] Michael D. Rugg, "Introduction," in *Cognitive Neuroscience*, ed. Michael D. Rugg (Cambridge: The MIT Press, 1996), 6.

of the term 'reductionism'. The first part of this chapter is a discussion of reductionism as a concept, as this phrase has come to mean many things over the course of intellectual history. Patricia Churchland notes that:

> The word 'reduction' has a bewildering range of applications in the literature. 'Reductionism' has come in some quarters to be used as a general term of insult and abuse. Sometimes it is used as a synonym for 'behaviorism' (which is a case of the vague hounding the vague), or as a synonym for such diverse sins as 'materialism,' 'bourgeois capitalism,' 'experimentalism,' 'vivisectionism,' 'communism,' 'militarism,' 'sociobiology,' and 'atheism.' With such diversity, equivocation is inevitable, and often as not opposing sides in a debate on reductionism go right by each other because they have not agreed upon what they disagree about."[14]

On the cognitive level, reductionism is meant to refer to the ability to represent a cognitive process with a correlating neurological process – for example, a given neuron or neural system firing which corresponds to a perceived mental phenomenon. It seems beyond question that all mental processes must have some physical correlation, whether gross or fine in nature. While the debate is still ongoing about whether consciousness is merely an epiphenomenon of neural activity, it seems unscientific and almost regressive to suggest that there is a non-physical basis for thought and reasoning processes. Each heartbeat is triggered by unconscious neural activity – we do not have to think about every systolic and diastolic cycle. In a like manner, we experience conscious, subconscious, and preconscious events through neural processing pathways – we think about things at a variety of levels, some accurate, some inaccurate (these types of heuristics and cognitive distortions and clarifications will be discussed in later chapters).

Reductionism ought not to be understood as explaining *everything cognitive* in pure terms of neuroanatomy and physiology; it seems unrealistic that there would be a cognitive structure for each and every event, emotion, and cognitive experience we have. Rather, we should view cognitive function as the interplay between levels of explanation. Our goal is to explain

[14] Churchland, *Neurophilosophy*, p. 278.

cognitive mechanisms in terms of appropriate simplicity – some explanations will be more complex than others. Wimsatt notes:

> The point of reduction is not to get an 'infinite regress' explanation for 'eventually everything' in terms of 'essentially nothing,' but only to make sure that everything gets explained – at *some* level or other. This in fact allows for the possibility that some things may require explanation at *higher* levels…Also, while we may explain away false theories, or explain why we believed them, we do not literally explain something false or something that did not happen. So the full explanatory maxim which takes account of both of these factors is: *Explain everything that occurs and nothing that doesn't*.[15]

As such, we ought not limit ourselves solely to explanations in terms of anatomy, structure, and signaling; however, we ought not simply throw up our hands in despair and fiat non-physical causative elements (the 'ghost in the machine').

In his discussion of reductionistic arguments in molecular biology, Sarkar argues that contemporary reductionism is essentially a modern incarnation of traditional mechanism, in which living organisms are recast as complex machines.[16] He argues that there are both epistemological and ontological issues present, noting that "the former include questions about what exists in a system, whether the laws and mechanisms at one level determine phenomena at another, etc. The latter include the questions about whether reductionist explanations of phenomena can be offered and whether research programs should be based on reductionist strategies."[17] As such, our discussion of reductionism is not simply a manner of representing data and neural interactions, but also our understanding of what it means to be human and how we should study ourselves.

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[15] William C. Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 205-67, p. 225.

[16] Sahotra Sarkar, *Molecular Models of Life: Philosophical Papers on Molecular Biology* (Cambridge: The MIT Press, 2005), p. 70-71. Hereafter, 'Sarkar'.

[17] Sarkar, p. 106.

Organisms exhibit remarkable biological complexity – processes that seem everyday or mundane are actually the culmination of myriad chemical and physical interactions with specific pathways, dependencies, and outcomes. An explanation of cognitive function, perhaps the most intricate process in the human body, would seem to be irreducibly complex. Surely the phenomena of consciousness, memory, will, identity, emotion, ambition, and desire could not be explained in simple terms. This is, however, not necessarily the case. Sarkar and Paul Churchland both caution that arguments of irreducibility ought not be daunting – we should view the study of cognitive processes not as impossible ventures, but simply as very complex problems.[18] Even in the face of complexity, we must recognize that there is some dependence on physiology.

The point being, every mental event has some physical correlate, and hence, should be explicable in neurological terms. This is not to suggest that there is a strict one to one ratio between emotional and neurological state – it seems reasonable that a given emotional state may correspond to several different neuroanatomical states. Just as there are myriad reasons why one's car might not start, so too could there be multiple reasons why an agent is depressed, angry, agitated, etc. Later chapters will note a variety of etiologies for depressive disorders – there are several competing theories, all of which can explain a depressive state to varying degrees. The task at hand is to explore the most likely representation of the physical bases underlying cognition. I will not claim that mental phenomena are only to be understood in terms of individual neurons or neural systems. Paralleling Sarkar, for reasons which will become

[18] Sarkar, p. 121; Paul M. Churchland, *The Engine of Reason, the Seat of the Soul* (Cambridge: The MIT Press, 1995), p. 189.

clearer in later sections, I will argue for less radical forms of reduction, such as arguments of neural networks and interplay between levels of organization.[19]

Concepts/Types of Reduction

Parallels in Molecular Biology

In framing his discussion of reductionism, Sarkar argues against simpler reduction of molecular biology to genetics. While a detailed discussion of his views on this form of reductionism are interesting and fecund in a general sense, they are beyond the purview of this dissertation. As such, this dissertation will offer a simplified treatment of his rejection of the 'hereditarian program' as a parallel to the present discussion of mental phenomena. To paraphrase Sarkar, mental phenomena may be reductionistic, but they are not simplistic.[20] Even in a discussion of molecular biology, however, and despite his cautionary language, Sarkar does note that we must recognize the interplay between the biological constitution of the organism (at the genetic level) and its cognitive processes:

> Proponents of the Human Genome Project exploited that hope to initiate massive blind DNA-sequencing projects: the full sequencing of entire genomes without prior concern for the functions of the sequences. The same hope led to numerous, often irresponsible, claims that complex human behaviors (including male sexual orientation, schizophrenia, alcoholism, autism, reading disability, bipolar affective disorder [or manic depression], neuroticism, adolescent vocational interests, spatial and verbal reasoning, alleged differences in intelligence, etc.) had genetic etiologies. From this perspective, phenotypic traits are being explained from a genetic basis: the framework is one of genetic reductionism. Not one of these claims of genetic etiology has survived further experimentation and scrutiny…though it would also be irresponsible to argue that inherited biological constitution has no role in the etiology of human behavior.[21]

Having conceded some interplay between biology and cognition, Sarkar argues that suggestions of simple gene-to-phenotype expression (the idea that having the genes to code for a specific

[19] Sarkar, p. 112.
[20] Sarkar, p. 3.
[21] Sarkar, p. 13.

characteristic automatically and with necessity leads to that characteristic's actual expression) is overly facile. There are myriad other influences on phenotype. As he states, "Molecular biology has done much to demonstrate that genetic reductionism itself is sterile by showing how complex the path is from DNA sequence to phenotype, even for ordinary morphological phenotypes, let alone complex behavioral ones. From the molecular perspective, simple genotype-phenotype determinations are exceptional; phenotypic plasticity is ubiquitous."[22] As such, we see an implicit argument for a complex diasthesis-stress model of gene expression (diasthesis referring to genetic predispositions, stress referring to environmental stressors that lead to phenotypic expression of the gene, whether in physical or cognitive terms). In fact, the argument being made is not simply one of individual genes – many traits are polygenetic, and as such, require more than simply the presence of a given amino acid sequence. Rather, the complex whole of the genome and the proteins for which it codes (hence genomics and proteomics as fields of study, respectively) are more relevant to discussions of cellular and organism phenotype. Proteomics offers a reductionistic, but not simplistic, account of cellular function and the behavior of the overall organism.[23]

All of this has been discussed in an effort to note that reductionism is not simply relationship between objects or theories. Reductionism takes many forms, and can allow for different methodologies – that is to say, there are many ways in which we can understand how the reduction takes place. Sarkar notes three elementary forms of reductionism: theory reduction, explanatory reduction, and constitutive reduction.[24] In general terms, theory reduction refers to efforts to relate one theory to another – a theory is shown to be represented in the terms of a simpler or more fundamental theory (e.g., we can understand digestion [a higher level

[22] Sarkar, p. 14.
[23] Sarkar, p. 34.
[24] Sarkar, p. 55.

process] in terms of biochemistry [a lower level process]). Explanation is not always required with this form of reduction, but this frequently occurs.[25] Because of this occasional rift between theory reduction and explanation, a second type of reduction is necessary, hence explanatory reduction. In this type of reduction, an explanation of phenomena in one theory is understood in terms of another. The third type of reduction is constitutive reduction, in which it is asserted that "that upper-level (intuitively larger) systems are composed of lower-level (intuitively smaller) systems and conform to the laws governing the latter. Unlike the cases of the previous two categories, models from this category of reductionism necessarily involve the separation of their domains into levels of organization."[26] These categories are not mutually exclusive – reductions can be represented in any or all of the three categories, or may be shown not to be reducible at all.[27]

Theory Reductionism

At its heart, theory reduction is the claim that theories can be reduced to more fundamental theories, that is, a particular theory is derived from some other, more basic theory. Sarkar notes that the most plausible argument of theory reduction derives from the work of Nagel and Schaffner. Nagel's model makes an epistemological claim about context-dependent conditional or biconditional truth between two theories (i.e., that for a given context or situation, the overall truth of the reduction will be contingent upon the conditional ('if…then…') or biconditional ('if and only if') truth of the relation between theories).[28] In essence, the relationship between the two theories makes an epistemological claim: a reduction is true if both the theory being reduced and the reducing theory are true. Schaffner modifies this claim by

[25] Sarkar, p. 58.
[26] Sarkar, p. 58.
[27] Sarkar, p. 76.
[28] Sarkar, p. 60.

noting that the reduced theory is not directly derived from the reducing theory; instead, the reducing theory produces a modified version of the reduced theory.[29] Hence, the final model of theory reduction from these two accounts is that a reducing theory both modifies and is corrected by the modified theory (hence biconditionality).

Wimsatt notes that there are objections to theory reductionism from logical and semantic perspectives. Philosophers have charged that while there may be some similarity between the reduced and reducing theories, there are meaning changes present that prevent a reduction from one to the other. Hence, they argue, reductionistic arguments commit fallacies of equivocation.[30] Wimsatt rejects this argument, and suggests that in the event of meaning change, we must simply understand it as another aspect of the reduction to explore and explain. He notes that "the assumption of a reduction of this type is generally that the new theory is true, but that the old theory to be derived from it is *literally false*. So if the argument form is valid, there had better be an equivocation somewhere!"[31]

Wimsatt suggests that there are fundamentally different types of theory reduction: interlevel and intralevel. Interlevel reduction occurs between systems of organization. For instance, there is a fundamentally different level of organization between an entire organism and the cells of which it is composed. An interlevel reduction would attempt to explain social functioning (a higher level phenomenon) in terms of biochemistry and physics (lower level phenomena). An intralevel reduction, by contrast, offers explanations of complex phenomena at

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[29] Sarkar, p. 60.

[30] A fallacy of equivocation is a logical error in which words take on different meanings, producing invalid arguments. For example, in the syllogism "A 747 is a plane. A plane is a carpenter's tool. Therefore, a 747 is a carpenter's tool" the fallacy of equivocation stems from an inappropriate usage of the term 'plane'. Contextually, each of the premises contains a correct usage of the term 'plane' – however, the conclusion does not follow, as it attempts to use one meaning of the term entirely out of context (i.e., it attempts to use the context from premise 1, when the conclusion should properly use the context from premise 2).

[31] William C. Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 218.

a given level of organization in light of more basic theories of explanation at that level. For example, "[r]elativistic mechanics may reduce to classic mechanics (etc.) but it clearly replaces (rather than reduces to) Aristotelian physics."[32] Interlevel reduction resists relation between theories, but intralevel reduction might not.[33] The key concern in reduction is the question of translation – how easily one theory can translate into the terms of another. The easier the translation, the more replaceable the reduced theory. By contrast, the more difficult the translation, the more essential the reduced theory – in fact, with enough difficulty in translation, it follows that a theory might not be reducible at all![34]

Wimsatt supports Schaffner's revised biconditional model, but notes that changes and corrections take place in theories at all levels of organization, but tend to be visible only in the upper levels. These changes affect the 'fit' of the model at that organization level, which then also has varying effects on models at other levels. In intralevel reduction, there is some wiggle room; interlevel reductions, however, requires significantly more precision:

> Considering only two levels, in an idealized reduction, this means that the theories at these two levels undergo a *co*evolution – they are the major factors producing change in each other: A lower-level model is advanced to explain an upper-level phenomenon which it doesn't fit exactly. This leads to a closed look at the phenomenon, and perhaps results in some change in the way in or detail with which it is described. This will also lead to changes in the lower level model and may suggest new phenomena to look for. These changes usually produce an improvement in 'fit' in some respects, but may involve a poorer fit in others, or suggest new areas in which fit must be obtained. But as argued above, interlevel explanation requires *exact* fit in all relevant dimensions, so the cycle will be repeated as many times as necessary to produce it.[35]

As such, it is easier for us to argue for reductionistic theoretical models in intralevel terms, but interlevel claims requires a higher onus of proof.

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[32] Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem," 219.
[33] Wimsatt, p. 221.
[34] Wimsatt, p. 222-3
[35] Wimsatt, p. 231.

A final note regarding Wimsatt's treatment of theory reduction – there are multiple levels of organization, but it is not necessarily clear that there is any particular order. Absent a clear order, it is not clear that we can definitively state what reduces to what – levels of organization are interrelated and defined in light of each other (i.e., the body is defined in terms of cells, organ systems, social existence, etc., and each of these levels can be explained in terms of other organizational levels). This creates quite a conundrum:

> Is reduction impossible in such complicated situations? If orderability of levels fails, this means that we can no longer say what is composed of what. On a view of reduction that emphasized ontological simplicity, or regarding upper level things as 'logical constructs' or aggregates of lower level things, this would seem to be a crucial failure. The fact that anatomical organs can be viewed as made up of physiological processes and physiological systems as made up of anatomical components suggests that neither view is complete and also that neither kind of entity is really totally made up of the other.[36]

As a consequence, it is necessary to establish what constitutes higher and lower order processes (if such an ordering is possible). This is especially difficult in light of the complex interaction of levels of organization involved in cognition. Wimsatt notes that the reduction of mental phenomena to physical phenomena is not necessarily a quick and easy process. In fact, when complete the reduction may not be perfect or crystal clear:

> If reduction of the mental to the physical is possible, it may be with this residual indeterminacy: It may be unclear exactly what psychology is being reduced to, and unclear exactly what is being reduced to neurophysiology, but the malaise may disappear, leaving only the conviction that the explanatory task is complete, and without ever having invoked forces which were inexplicable at lower levels. Probably that is the most a reductionist can ask, and probably also it is enough.[37]

While some indeterminacy may remain, this dissertation will assume that there is at least some degree of order in levels of organization in cognition.

Explanatory Reductionism

[36] Wimsatt, p. 255.
[37] Wimsatt, p. 256.

Explanatory reductionism is the easiest of the models of reduction to explain – it simply suggests that phenomena are explicable in simpler terms, theories, or rules. There are, however, challenges implicit in this approach. Sarkar argues that the issues of reduction and explanation ought to be considered separately, and he suggests that there are at least two compelling reasons to do so.[38] First, there is no generally agreed upon explanation of explanation! That is to say, in an effort to reduce one theory to another, we may inadvertently commit an error in translation that will prevent the reduction from being acceptable. This error in translation may, in fact, have nothing implicitly to do with the theory being reduced – it may be a purely methodological error which prevents the reduction from occurring. Hence, the *model of the reduction* may be in error. Second, explanation routinely involves approximation, and there is not presently a completely objective (i.e., context-independent) definition of a 'good approximation.' There are other considerations aside from definitional ones: for instance, how a question is posed, the rigor of the scientific field, etc. This he argues, "further underscores the importance of keeping the issue of explanation separate from that of reduction even while construing reduction as a form of explanation."[39]

Sarkar suggests that maintaining the distinction between reduction and explanation also allows for more fecund exploration of a given reduction, because these seem to capture the essence of real reductions:

> There are two intuitions that motivate this interest. *First*, scientific explanation is often 'messy' and involves that use of semiempirical rules and the invocation of mechanisms that do not form part of any fully explicated theory. At best, in such cases, explanation involves the use of a fragment of a theory. Reduction, in such circumstances, can only be construed as a relation between such rules, mechanisms or fragments, not as a relation between theories. However, even in such cases, certain kinds of explanation are regarded as being reductive, and a general account of types of reduction needs to be able to incorporate these.

[38] Sarkar, p. 65-6.
[39] Sarkar, p. 65-6.

> *Second*, the Nagel and Schaffner models, applied to interlevel reductions and explanations, do not explicitly incorporate the basic idea that a reductive explanation is the explanation *of a whole in terms of its parts*.[40]

As with theory reduction, there seems to be an element of indeterminacy or 'messiness' in scientific reductions. However, indeterminacy in reduction does not necessarily translate into an absence of cognitive modeling that is reductionistic in nature – it is still possible to note essential versus accidental elements of cognition. Cognition, for instance, can involve many levels of organization with complex interactions, and yet it is still explicable, in Sarkar's words, as a whole in terms of its parts.

Constitutive Reductionism

Sarkar argues that constitutive reductionism necessarily requires that higher level phenomena be explicable in terms of lower order phenomena – this 'research program' constitutes an ontological claim about the organism.[41] Further, he notes that it requires that "all biological processes occur in such a way that they are consistent with physical law."[42] In essence, these two conditions require that upper level phenomena (e.g., complex phenomena like consciousness, memory, cognition, etc.) cannot be changed without a corresponding change in lower level phenomena (e.g., neural network structure, neuromodulators, etc.), and vice versa. This relationship is called *supervenience* (i.e., higher order phenomena are supervenient on lower order phenomena).[43] Sarkar argues that the 'research program' exploring the proposed reduction might fail, either because it was pursued improperly or prematurely, or because the research program might be in error – no such reduction might be possible. Sarkar cites the reduction of mental phenomena to physical phenomena as a paradigmatic example:

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[40] Sarkar, p. 63.
[41] Sarkar, p. 69.
[42] Sarkar, p. 119.
[43] Sarkar, p. 69.

> [I]t could be the case that the research program is itself fundamentally mistaken. However, this would probably bring into question whether even any model of constitutive reduction is itself applicable. The only plausible example where so fundamentally mistaken a research program might be being pursued is the case of the attempted reduction of the psychological realm to the physical and, here, it is easy to see that, should the program fail, the ontological assumption involved, that is, constitutive reductionism itself, is exactly what would have to be questioned.[44]

The difficulty, however, is how we can account for changes in cognitive processing, indeed changes in personality, following neurological insult or injury. However, it is likely that Sarkar is simply proposing a hypothetical, rather than explicitly rejecting the thesis that mental phenomena are contingent on and expressible in underlying physical terms.

Computational Models

A popular concept of cognitive processing involves comparisons with computation. Schweitzer notes that intentional states are the cornerstone of cognitive science, and that they are traditionally bound with other philosophies like functionalism (see below); but the essential feature of this approach is the characterization of cognitive events in terms of computational profiles:

> The computational paradigm is standardly welded to some brand of *functionalism*, according to which mental states derive their identity from the abstract causal roles they play in a complex economy of internal states mediating environmental inputs and behavioral outputs. But whatever the details and fine-grained variation, functionalism and/or classical computationalism share the notion that decomposition in terms of input/output profiles and sequences of internal processing states is the salient model for understanding cognitive phenomena. Thus according to the classical view, the level of relevance to the science of mind is the *program*, the sequence of abstract state transitions that yields intelligent responses to external stimuli. This mind/program analogy is one of the basic conceptual legacies of orthodox cognitive science and AI.[45]

While some choose to identify cognitive science solely with the means by which information is processed, Schweitzer argues that there is necessarily a fundamentally materialist underpinning.

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[44] Sarkar, p 69-70.
[45] Paul Schweitzer, "Realization, Reduction, and Psychological Autonomy," *Synthese* 126 (2001): 384.

Instead of adopting the traditional assumptions of cognitive science, which he neatly summarizes as "the abstract formal procedure, not the bowl of porridge in which it happens to be implemented,"[46] he argues that "the primary reason for invoking computation should be precisely the goal of providing a *reduction* of the mental to the physical, and that without this aim, the move to computationalism becomes scientifically unmotivated."[47] In fact, he argues, "[o]ne of the fundamental components of the cognitive science paradigm is the idea that the brain as a physical system serves to *realize* a particular abstract computational structure."[48] Hence, it is necessary to explore the physical underpinnings of a computational model of cognition, in an effort to explain our memory, cognition, sense of self, and other higher order processes.

There is reason to believe that our self-concept has a neuroanatomical correlate. Ira Black, for instance, suggests that part of our self-concept – the cognitive recognition of our physical selves in opposing areas of physical space, as well as our ability to recognize abnormalities in ourselves are "localized to specific regions of the brain. Although the precise neural subsystems have yet to be defined, a physical substratum for the most subjective of psychologic entities has been identified."[49] He notes that it is quite likely that further investigation will reveal increasingly complex levels of association in the non-dominant parietal lobe. In a like manner, our critical "emotional-vegetative-social functions of the self" are localized in the frontal cortices.[50] This localization will be important in later chapters, wherein the link between affective disorders and frontal lobe dysfunction will be explored more fully. For the moment, it will suffice to say that disruptions in neuroanatomy can alter personality and

[46] Schweitzer, "Realization, Reduction, and Psychological Autonomy," 384.
[47] Schweitzer, p. 385.
[48] Schweitzer, p. 389.
[49] Ira Black, *Information in the Brain: A Molecular Perspective* (Cambridge: The MIT Press, 1991), p. 168-9.
[50] Black, *Information in the Brain: A Molecular Perspective*, 170.

cognition. Self-concept, Black notes, is a concatenation of distributed processes; the self is, in essence, modular.[51] Our sense of "self in space, the self in time, and the social self" can be localized to the parietal, temporal, and frontal lobes, respectively.[52] As a final note, Black suggests that these modules interact, forming a plastic neural network (a neural architecture capable of change and adaptation) – our sense of self, our understanding of who and what we are, changes both physically and metaphysically.[53] Phillips and Patricia Churchland also propose neural networks as reductionistic models of cognition.

Returning to the idea from theory reduction of levels of organization, Phillips notes that there are a variety of capacities in which cognition can be interpreted.[54] We can examine individual neurotransmitters, the microcircuits within dendritic trees, neurons, neural circuits, cortical pathways, and systems of cortical pathways. He argues that it would be a mistake to analyze cognition only at higher levels – cognition is a process related at many levels of organization. He notes that "[c]ognition and cortex may be comprehensibly related at all these levels. Some cognitive phenomena, such as the effects of certain drugs, pathologies or altered states of consciousness, may be best understood by using knowledge of how specific substances, such as neurotransmitters or neuromodulators, affect processing at the subcellular level."[55] He notes that computational functions are carried out at many different levels of organization, and as such, "no theory that attempts to explain the whole of cognition on the basis of just a few cortical principles is likely to be successful…Different subsystems may well interact with and constrain

[51] Black, p. 170.
[52] Black, p. 172.
[53] Black, p. 179.
[54] William A. Phillips, "Theories of Cortical Computation," in *Cognitive Neuroscience*, ed. Michael D. Rugg (Cambridge: The MIT Press, 1996), 15.
[55] Phillips, "Theories of Cortical Computation," 15.

each other, but that doesn't invalidate the view that different subsystems play different roles in the system as a whole, and solve essentially distinct problems."[56]

Memory is a key area of exploration in the computational model. Humans have different kinds of memory – e.g., long-term memory, working memory, episodic memory, procedural memory – that require the activation of different pathways, different cortical regions, and different neurological structures. The hippocampus, for instance, is an essential element of episodic memory but not procedural memory, while other types of memory rely more on sensorimotor cortices. Phillips argues that it is essential to keep these distinctions in mind.[57] Hence, there is no single reductive model of memory; the phenomena is explicable in physical terms, but not identical physical terms.

Phillips further argues that activity within the network is not linear, which is to say that it does not simply follow a given path. Rather, the entirety of the network is involved, and activity "does not just flow through the network, but flows 'around' the network."[58] As a consequence, the activity of the network will depend on the connections between the individualized units. A given input may not necessarily yield one single output – rather, the net effect of the neural system may be chaotic, allowing some indeterminacy in cognition. Phillips suggests that these recurrent neural nets are engaged in this type of chaotic activity when they "store and recreate temporal sequences of activity."[59] This should not be understood as an argument for indeterminacy at the reductive level, or an argument against the deterministic qualities of cognition (see below), but rather that the product of a neural network is dependent upon its contemporaneous arrangement and strength.

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[56] Phillips, p. 16.
[57] Phillips, p. 16-7
[58] Phillips, p. 32-3
[59] Phillips, p. 32-3

Memory, then, may be a given state of a recurrent neural net – it may be a pattern of activation and strength of connection between individual units. Phillips asks us to envision a three-dimensional map to represent the energy surface of a cognitive system (much akin to maps of gravitation in physics).[60] The deep points – the 'attractors' – on this map represent the low points of energy activation, and the system will act and react in such a manner as to reduce the overall level of activation (the level of activation is understood in terms of disagreement with external stimuli – the higher the degree of disagreement, the higher the system energy). Surrounding these deep points (perhaps best thought of as the downward change in slopes leading to these deep points) are the activation states of these low points of energy attraction, which Phillips describes as the 'attractor basin.' Memories are the attractors, and the activation pathways that lead to them are the attractor basins. When a system is activated by an external event (triggers for a memory), the system will seek out these attractors – the system will bring forth memories relevant to the external stimuli. In practical terms, this may be why people with similar facial features or gaits may remind us of friends or relatives – they have activated a given pathway, and our cognitive system has settled into a specified energy arrangement. Difficulty in retrieving specific words or memories may be a result of difficulty activating a given attractor basin.[61] This cognitive mapping approach is similar to the tensor networks proposed by Churchland.

Phillips argues further that it is not simply memory that is contingent upon the underlying neural mechanisms of cognition. Rather, there are a variety of cognitive capacities that are predicated upon the computational capabilities of the cerebral cortex.[62] There are significant elements of the human experience that we do not yet fully understand in reductionist terms,

[60] Phillips, p. 33-4
[61] Phillips, p. 33-4
[62] Phillips, p. 40-1

either in neuroanatomy or in terms of recurrent neural networks. Theories attempting to explain these complex phenomena are, however, bound to existent neuroanatomy – we cannot simply posit a 'ghost in the shell'.

This has immediate relevance in the context of cognition and choice. In discussing the meaning of cognitive science for psychology, Schweitzer argues for a model in which there are multiple methods by which mental phenomena may be represented. Schweitzer is not entirely smitten with the term 'reductionism', his focus instead is "not the terminology, but rather the theoretical consequences and methodological practices it is used to justify. And what I *would* strenuously object to is the move from the multiple realizability of abstract mental structure to the conclusion that the mind is somehow 'autonomous' in any interesting or significant sense."[63] Simply because cognitive structures can be realized in a variety of different arrangements, it does not follow that the mind is irreducible to the body or that psychological states are not dependent on physical states. Maintaining the computational motif, Schweitzer notes that "organisms with advanced cognitive capacities are still essentially hardware devices, not software implementations. Naturally occurring systems do not have a pure 'software' level that can be nearly detached from the physical mechanism."[64] Operating within this allusion, I could not type this dissertation abstractly – there is no freely existing Microsoft Word – I have to have a medium on which to run it. In a like manner, there is no freely existing thought – that activity is inextricably bound to and determined by the material doing the thinking. As Schweitzer notes, "[f]rom a truly physicalist perspective, the mind simply *is* a state of the hardware."[65] We cannot study psychology in a vacuum – we require input from neuroscience, neurology, etc., and other fields examining the physical underpinnings of this abstraction. When we change the physical

[63] Schweitzer, p. 390.
[64] Schweitzer, p. 391.
[65] Schweitzer, p. 391.

elements – the hardware in our computational model – we change how the higher processes function – the software. If we adhere to a computational viewpoint, we commit ourselves to a rejection of cognitive autonomy:

> The staunch defender of intentional explanation who insists on mental autonomy must give up the computational paradigm. On the other hand, the *cognitive scientist*, who is committed to computation as the cornerstone of the discipline, must eschew mental autonomy. For the cognitive scientist, the mental level may be indispensable, but it is neither ontologically *nor* theoretically independent of the physical. Psychological structures must be realized in the brain, and hence have empirical dependencies and ramifications for *outside* the field of psychology. The rigorous testing, and thus ultimately the very articulation of psychological laws and regularities, requires systematic accord with the detailed workings of the central nervous system."[66]

The absence of cognitive autonomy is one of many competing definitions of deterministic philosophy, a theme which we will return to shortly.

Functionalism

The functionalist school of thought defines the essential nature of a system by its underlying function – i.e., how it does what it does. A functional description of cognition would focus on the neural structures that are activated when a given task is performed, i.e., the individual neurons, neural pathways, neuroanatomical structures, etc., just as a functional exploration of computation would focus on the interaction of circuitry and processors. Higher cognitive functions are not non-physical phenomena – as Patricia Churchland notes, "**t**he differences between functional descriptions and hardware descriptions turn out to be differences in what *level* of description is appropriate, not in whether what is described are physical states or nonphysical states."[67] Physical structures play a significant part in structures like memory, an important element in self-concept and decision-making. Jonides and Smith argue that memory is a critical element of normal cognition – in fact, they argue that:

[66] Schweitzer, p. 398-9
[67] Churchland, *Neurophilosophy*, p. 340.

> Every intelligent being has some form of memory play a critical role in its higher cognitive function. This is true of any complex computing device, it is true of any animal that is respected for its cognitive skill, and it is true of humans. In fact, the role played by memory in cognition is sufficiently complex that just a single memory will not do. Rather, computers, humans and other animals have all developed (some by evolution, some by design) multiple memory systems that aid in higher level cognition.[68]

Frith and Friston note that neurological experiments have noted specific components of working memory (a central executive and slave systems specific to the information being remembered), and that these components can be disrupted by very different forms of interference.[69] This suggests that there are external elements that can disrupt normal cognitive functioning, and that our normal cognition is dependent upon factors outside of our volition – an argument that we will revisit at length later. Our understanding of the structural elements of central executive processes is not as advanced as that of memory, but "the available evidence is beginning to reveal the complex architecture of executive processes and their representation in the brain."[70]

Challenges to Reductionism

As suggested at the beginning of this chapter, reductionism is not without its detractors. Before proceeding with this dissertation, it is necessary to explore some of the challenges raised, in an effort both to explain the issue as well as to clarify the reductionistic concept assumed in this dissertation. Some of the objections are couched in abstraction – they are objections at the conceptual level about the irreducibility of theories, for instance. Others are empirical, either in appeals to irreducible complexity or the limitations of contemporary research. Still others suggest that mental phenomena are emergent qualities of neural structures – the idea that consciousness emerges as a result of the complexity found in neural structure. Just as light

[68] John Jonides and Edward E. Smith, "The Architecture of Working Memory," in *Cognitive Neuroscience*, ed. Michael D. Rugg (Cambridge: The MIT Press, 1996), 243.
[69] Christopher D. Frith and Karl J. Friston, "Studying Brain Function with Neuroimaging," in *Cognitive Neuroscience*, ed. Michael D. Rugg (Cambridge: The MIT Press, 1996), 183.
[70] Jonides and Smith, "The Architecture of Working Memory.", p. 271-2

emerges from a light bulb due to the completed electrical pathway, so too do our 'minds' emerge from the myriad neuronal interactions.

A recurrent theme is the questionability of the hypothesis that we are 'merely' physical beings – in essence, cogent and compelling arguments are offered suggesting that simple reduction may not account for the variety of mental phenomena we experience. This dissertation does not assume simple reduction; rather, it will argue that mental phenomena are dependent upon the underlying neural structure, and that alterations in lower level phenomena produce changes in upper level phenomena. Further, it will be argued that these levels fundamentally interact, which is capable of producing complex behavioral changes – there does not seem to be a single level of organization which is not influenced by other levels of organization. Case studies and neurological research will clarify this position, and demonstrate that changes in neurochemistry due to a depressive disorder can alter the experience of 'self' in the same manner as neurological impairment or damage. This will be more fully explicated in chapter four.

General objections

Mills suggests that there are many compelling reasons to reject, or at least be skeptical of, materialist and reductionist arguments. He argues that there are significant bases for this rejection – at stake are fundamental elements of humanity:

> The claim that the mind is nothing but the brain is a dogmatic assertion that attributes ontological primacy to physical states over mental processes and properties. In short, the materialist holds a fallacious and simplistic view of causality, denies free agency of the self, and increasingly portrays the human being as a clinical object. The ethical implications of such approaches in medical and social-political practices may potentially threaten the integrity of individuality and collective identity, which may further lead to an invalidation and/or empathic impasse regarding human difference and understanding.[71]

[71] Jon Mills, "Five Dangers of Materialism," in *Taking Sides: Clashing Views on Controversial Issues in Cognitive Science*, ed. Marion Mason (Dubuque: McGraw-Hill/Dushkin, 2005), 10-19, p. 17

Mills raises five general and essential critiques of materialist and reductionist arguments, each of which will be addressed and discussed in turn.

First, there is a significant concern that the individual is reduced to pure mechanism. Mills is concerned that by stripping the psyche of its non-physical elements, we are essentially stripping the mind or self of its ontological status. The self loses its transcendental nature – we reduce humans to mere 'things'. [72] The meaning of being human is lost – our existential questions, our moral dilemmas, all that which would seem to define us and our subjective experience is reduced to biochemical equations and expressions. [73] We will discuss the problem of personal experience in greater detail below.

A second critique concerns a bias towards simplicity. Materialist arguments, Mills suggests, assume that the only phenomena of mental life are those which can be observed. He suggests that we have abandoned complex accounts of personal phenomena due to an implicit parsimony – it is easier for us to explain the mental in terms of the physical, and as such, we have an unwarranted bias in favor of simplicity. However, he notes, "the simplest explanation is not necessarily the most accurate."[74] In response to this claim, however, there is a burden of proof necessarily imposed on someone suggesting more complex models – I could, for instance, claim the same line of reasoning to justify my belief in an impossibly complex model of consciousness involving souls, 'anti-souls', quantum mechanics, and motivating immaterial beetles. This response is obviously a *reductio ad absurdum*, but it seems warranted – an appeal to occasional complexity does not seem sufficient to warrant a conclusion of complexity in any one particular instance.

[72] Mills, "Five Dangers of Materialism", p. 12-3.
[73] Mills, p. 15
[74] Mills, p. 13

Third, Mills argues that materialists must necessarily endorse agnosticism regarding the ontology of consciousness. The essential question is whether materialism is necessary *and* sufficient to explain consciousness. Again, consciousness seems to be essentially immaterial – a psychical construct. He argues that "[m]ind is embodied or instantiated physically, but by virtue of its transcendental and elusive functions and properties, it cannot be spatially localized or dissected. Most materialists want to eliminate this stance as a viable possibility and hold allegiance to a simple economy – that which is *real* is something that is tangible."[75] At this point, one could easily accuse Mills of proposing essentially an argument from ignorance – after all, nonphysical entities are precisely what he feels do not require physical proof, and their absence does not thereby suggest their non-existence. Essentially, this objection insists that just because we can neither prove nor disprove the existence of the non-physical, we cannot rule it out. This seems to be an untenable position – after all, as I tell my introductory philosophy students, they can neither prove nor disprove the existence of an invisible, immaterial magical elf on my left shoulder.[76]

Fourth, there is an implicit danger of resurrecting behaviorist claims about personality and cognition. Cognition in the materialist model boils down to an interaction between physical causation and environmental determinism. In essence, all mental events are caused by physical processes and environmental stimuli, which reduces our agency to a reaction to external events.[77] This seems to be an overly simplistic assessment of the reductionist position – as it is presented and discussed in this chapter, for instance, reductionism does not require belief in a form of radical behaviorism. All that is suggested is influence at multiple levels of cognitive processing.

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[75] Mills, p. 13
[76] Slappy the Magical Elf is frequently invoked my Introduction to Philosophy classes when discussion moves beyond the realm of the logical or empirical, a bias I am happy to admit.
[77] Mills, p. 14

Internal and external stimuli can certainly trigger mental events, but how they are ultimately realized in upper level phenomena, or whether they are acted upon at all is an entirely different question, which amounts to an explicit rejection of behaviorism.

Fifth, there is a concern about the phenomenon of free will. Mills argues that ultimately the materialist is committed to an overt rejection of free will:

> Reliance on material and efficient causal explanations, the over-valuation of simplicity à la Ockham's razor, and consequently, physical reductionism, completely eliminate any possibility of free will. From this standpoint, the human being is not free. This position is summarized by the exclusion thesis, which posits that human beings have no properties or mental powers that no object or physical system can possess. Thus, if free will is a mental process or property, and no physical system is free, then we do not possess free choice and are consequently not free. This simplicity denies the possibility of final causal determinants and transcendental teleology characteristic of free agents. *Agent* is defined here as a subject who is telic, purposeful, and self-directed via choices and deliberation in judgments constituting self-conscious activity. Therefore, thoughts, volitional intentions, and behaviors are the activities of the will: Freedom is ultimately defined as the ability to choose to *be* otherwise. Freedom, however, is not merely restricted to choice; it also encompasses the structural organization of the individual doing the choosing, namely the agent. In short, agency, free will, intentionality, and final causality (e.g., choosing the grounds for the sake of which to behave) are problematic for the materialist, for physical matter is caused rather than freely causal.[78]

While this does seem a bit of a sweeping generalization of all materialist positions, there is a significant concern here which I am inclined to support. Fundamentally thought is dependent upon the physical structure of the cognizing entity – there are no free-floating consciousnesses of which I am aware. As such, and as has been argued in this chapter, cognition is not entirely a voluntary process. We are aware of some higher level phenomena and are capable of some introspection, but there are a great deal of cognitive processes of which we are unaware (see the next chapter). Further, while we can exercise control over some elements of our physiognomy, we cannot directly influence the shape, structure, or connections of individual neurons or neural

[78] Mills, p. 14

pathways. As such, it does seem compelling to note that cognition, and its derivative processes like choice, volition, and agency, are essentially deterministic (the extent of which we will explore in the second half of this chapter). The question, however, is whether this is as dangerous as Mills suggests – perhaps it is indicative of an upcoming paradigm shift in how we view choice in the context of reductionist ontology and epistemology. In later chapters, for instance, we will explore the tenability of several popular conceptions of personal autonomy in light of the neuroscience and neuropsychology discussed in this and later chapters.

Reductionism and the qualia of personal experience

Turning now from general objections to reductionism, the task at hand is to explain the complex phenomena of 'the self' and subjective awareness. Objections have been raised to the idea that our subjective experience is explicable in reductive terms (e.g., Nagel's "What is it like to be a bat?"), and it has been suggested that there is a significant split between what is knowable about the human experience in objective terms (e.g., pure mechanistic explanations of the automation of the human body systems, chemical analysis of cells, bone, and tissue, etc.) versus what is knowable about the human experience in subjective terms (e.g., personal cognitive experience, individuality and identity demarcation, etc.):

> Nevertheless, eliminating materialists, such as Churchland, must still account for the phenomenon of introspection and the 'qualitative feel' of our alleged mental states. The eliminative materialist must account for the difference we claim to perceive between pain, for example, and our understanding of a mathematical problem or our believing of knowing a fact. A strong case can be made that these latter phenomena are best explained under some dualist theory of mind/brain.[79]

These objections rightly claim that there is much about the human experience that is ineffable in current scientific methodology.

[79] Paul C. L. Tang, "A Review Essay: Recent Literature on Cognitive Science," in *Taking Sides: Clashing Views on Controversial Issues in Cognitive Science*, ed. Marion Mason (Dubuque: McGraw-Hill/Dushkin, 2005), 4-9, p. 5.

The exploration of reductionism cannot overlook personal intuition of the self. There is a certain amount of 'folk psychology' at work that does not depend upon models, nor is it beholden to any particular research methodology. There is the intuitive claim that we are the masters of our cognitive fate, that we are not beholden to past experience or subconscious processes in making our choices. Rather, we simply act as we see fit, with no overriding concerns for the determined aspects of our personalities. This is, as Patricia Smith Churchland notes, a potentially self-defeating and incorrect model – once we start looking at our introspective process, we may have to revise or abandon our assumptions.[80]

There are a variety of phenomena which have been used to justify claims of the irreducibility and ineffability of subjective experience. Further these claims are not joined by one single common argument – rather, there is a plethora of arguments which attempt to justify the idea of irreducibility.[81] Churchland notes that there are two essential variations on the irreducibility argument, which she classifies as the 'boggled skeptics', who suggest that the brain is simply too complex to understand, and the 'principled skeptics' who argue from "the nature of subjective experience or from the fact that some mental states have meaning and significance."[82] The boggled skeptic claim can be rejected, as it simply does not offer a defensible position. While current neuroscience has not provided complete answers concerning the essence of the self, it has provided significant inroads and afforded a greater understanding of how the brain works.

The challenges of the principled skeptic, however, can prove to be more difficult. Churchland cites Thomas Nagel's essay "What is it like to be a bat?" as an example of a principled skeptic. Nagel argues that the fundamental qualities of subjective experience are only

[80] Churchland, *Neurophilosophy*, 293.
[81] Churchland, 315.
[82] Churchland, *Neurophilosophy*, 316.

appreciable to that particular being; as an outsider, I can have limited objective knowledge of what it would be like to be someone or something else, but I cannot have complete knowledge without actually being the person or object in question. But there is something specific about actually being the person in question that is not explainable in quantitative terms; Nagel argues that this qualitative aspect of being is irreducible to neuroscience.[83] But this seems to be begging the question:

> The point is this: *if in fact* mental states are identical to brain states, then when I introspect a mental state, I do introspect the brain state with which it is identical. Needless to say, I may not *describe* my mental state as a brain state, but whether I do depends on what information I have about the brain, not upon whether the mental state really is identical to some brain state. The identity can be a fact about the world independently of my knowledge that it is a fact about the world…In short, identities may obtain even when we have not discovered that they do. The problem with the second premise is that the only justification for denying that introspective awareness of sensations *could be* introspective awareness of brain states derives from the assumption that mental states are not identical with brain states. And that is precisely what the argument is supposed to prove. Hence the charge of begging the question.[84]

Nagel assumes a qualitative difference between an objective versus subjective appreciation of one's existence, and in doing so, assumes the existence of the very concept he wishes to prove. But this posited qualitative difference between what we can know externally versus internally can not be dismissed so readily.

Hypothetically, if it were possible to translate subjective human experience into objective language and experience – that is, translate our individuality into quanta that could be understood by anyone – then we would cease to be who we are as individuals. While there is a visceral appeal to the suggestion that I am unique and ineffable, there seems to be a practical and empirical objection to this. First, there are definite elements of my personality that are not shaped by my subjective experience – as John Hospers notes, at the lower level of moral

[83] Churchland, 327.
[84] Churchland, 329.

discourse, my personality is shaped by genetics, my environment as a child, my family experience, my birth order, my race, gender, etc. None of these things are chosen by me, and yet they are essential elements of who I am and how I see the world. It also does not seem to be too far-fetched to note that men can appreciate the worldview of women in objective terms and vice versa (see, for example, Deborah Tannen or Robin Lakoff in sociolinguistics). It further does not seem too far-fetched to appreciate how greater access to resources at an early age can shape appreciation of material goods and thereby attitudes towards economic materialism. These are but two examples of subjective elements which may be understood objectively, without necessarily denigrating the individual's experience of the world. In fact, it has been suggested that appreciation of other sociolinguistic models help to bridge interpersonal gaps – suggesting that this type of understanding may not only be possible but also desirable.

Churchland argues that this irreducible qualia of experience does not necessarily require an immaterial basis. Rather, it may simply refer to a physical information pathway for a particular phenomenon that is not possessed by others.[85] Further, there are empirical claims that can be raised against the argument of non-physical phenomena – there is no reason why a physical system should be affected by them:

> It is of course possible that mental states do have nonphysical features. And it remains possible that one's autoconnected epistemic pathways are precisely what detect them, which is essentially what Nagel is insisting. These ideas are certainly not impossible. Quite the contrary. But their credentials as default assumptions have now evaporated. The mere existence of autoconnected epistemic pathways, which almost every creature possesses, should no longer even suggest the existence of nonphysical features. If they do exist, it is the burden of some other argument to spotlight them. In fact, the situation for Nagel's picture is slightly darker than this, because even if such nonphysical features were to exist, why should one's autoconnected pathways pay any attention to them? Those pathways are themselves entirely physical. How could they interact with any nonphysical goings-on? In any case, it is far more likely that those pathways arose, under the normal selective pressures of biological

[85] Paul M. Churchland, *The Engine of Reason, the Seat of the Soul* (Cambridge: The MIT Press, 1995), 198.

> evolution, so as to integrate all relevant aspects of our internal *physiological* activities, both sensory and motor. Nonphysical properties are not a solution to anything, even where one's self-knowledge is concerned. The existence of one's auto-connected epistemic pathways, their origins, and their current cognitive functions are all intelligible, without remainder, on purely physicalist assumptions.[86]

The onus of proof is thereby upon those who would make the system more complex, as well as the burden of demonstrating how the physical would interact with the non-physical – a conundrum addressed earlier. Churchland argues that the positing of mental phenomena like consciousness is frequently couched in terms meant to foment hostility between reductionist and anti-reductionist camps. This is, however, entirely unnecessary and quite false:

> There is a general lesson to be drawn here beyond the deserved deflation of two anti-reductionist arguments. It is crucial to bring it out. An assumption common to many thinkers, not just to Nagel and Jackson, is that the neuroscientific, computational, physicalistic approach to human cognition is in some essential way hostile to the notion of consciousness, and to the unique first-person perspective that any creature has, onto itself and onto the world at large. Although the assumption is widespread, nothing could be farther from the truth. The explanation of consciousness, both animal and human, is one of the central hopes of current research in cognitive neurobiology…And reconstructing the intricacies of each creature's unique cognitive perspective on the world is part of the lasting explanatory obligation that cognitive neurobiology is eager to accept. How realistic these hopes are is still a matter of dispute. But there should be no disputing that they are now among the hopes that neuroscience holds dear.[87]

Neuroscience is guided by the mysterious; however, as has been a recurring theme in this section, mystery neither requires nor suggests that a phenomenon is inexplicable. Reductionism still faces many challenges, conceptual and practical.

Dialectical Interplay

Wimsatt raises a significant challenge to reductionism by noting that the mental realm of our existence cannot simply be sloughed off in light of reductionism. He notes that while

[86] Churchland, *The Engine of Reason, the Seat of the Soul*, 200.
[87] Churchland, *The Engine of Reason, the Seat of the Soul*, 202.

reductionism may be able to explain lower level phenomena, it does so in light of higher phenomena – in fact, the lower level explanations are a requisite part of that explanation:

> [N]europhysiology cannot make progress at the level of higher units of functional organization without appealing to the mental realm for guidance. The task would be like asking a molecular biologist to give a molecular reconstruction of elephant physiology from what he knows, together with photographs of elephants taken in the wild at a conservatively safe distance. Constant reference to minute details of the upper-level descriptions of elephant anatomy and physiology are at least necessary (though not yet sufficient) for the task.[88]

The crux of his objection is that levels of organization are mutually dependent – while we can explain upper level phenomena in terms of lower level phenomena, we cannot suggest that this ontological support is unidirectional. Rather, there is a constant dialogue between the two – an irreducible dialectic between the two. We have a natural tendency to view ontological support as one-way; but this is, Wimsatt argues, fundamentally mistaken.[89]

This is a difficult challenge for reductionism – there is an empirical and intuitive basis for this claim. After all, when we learn a concept, we are not physically adding new neurons into our existing neural network. Rather, multiple higher level phenomena are interacting which affects our cognitive maps. Wimsatt refers to this as a 'panphenomenalism' which he feels is a necessary understanding to make reductionism palatable to those traditionally opposed to it:

> On this account then we have a kind of 'panphenomenalism' (not to be confused with what is usually thought of as panpsychism) at which entities and things detected by them at different levels are equally real, and none is secondary, in its *reality*, to any other. This is, I think, a necessary move in removing the feeling of austerity that many people appear to fear in reductionism – what, in other words, might be called the 'nothing more than' phobia.[90]

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[88] William C. Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 205-67, p. 236.
[89] Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem.", p. 236
[90] Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem.", p. 242

As an example of the panphenomenalism Wimsatt proposes, a thumbnail sketch of the experience of classroom learning would necessarily involve multiple levels of organization. At a higher level of organization, we would expect to find the phenomena of consciousness, attention, and intention.[91] At a lower level of organization, but above that of individual neurons or neuron pathways, the primary and secondary auditory and visual cortices, language association and comprehension pathways, memory centers (both working and long-term), etc., would be activated and modified by the higher level phenomena experienced, just as they activate and modify the higher level phenomena in turn. At the level of individual neurons and neural pathways, learning creates connections between individual cells that were not present before, which then facilitates future recall. Again, there is a mutual influence between higher and lower levels of organization. One could conceivable discuss effects at the cellular level in the complex chemical interactions between neurons. The classroom learning experience may stimulate addition production of glutamate (a primary excitatory neurotransmitter), which can affect the speed of recall, attention to one's environment, etc., and a corresponding increase in demand for γ-aminobutyric acid (GABA – a primary inhibitory neurotransmitter). What, therefore, reduces to what? Which level is explainable only in terms of a lower level? Does it even make sense to speak of 'reduction' in any traditional sense?

The reductive claims made in this dissertation do not require adherence to a strict definition of reductionism – rather, the plasticity and mutual influence underscore the fundamental claim being made. Given this dialectical influence, a depressive disorder can have

[91] Regretfully, science has yet to prove that learning can occur by osmosis or passive diffusion, hence necessitating that students actually pay attention to the environment around them and be active in their learning process. The bulk of the data do not support the idea that one can sleep through class and learn, or sleep on one's textbook and learn, much to this author's chagrin.

cognitive effects on a variety of levels, each or all of which can affect cognitive processing and consciousness, and hence affect the final decision to forgo medical treatment.

Emergentism

The emergentist objection to reductionism is not a complete rejection of the materialist account of cognition. Rather, it is meant to connote the production of causative mental phenomena from physical structures. Sperry argues that the emergent phenomena of cognition are not meant to invoke dualistic arguments – the emergent processes are products of the underlying physical structures, which then influence the physical structures in turn. [He] argues that:

> On the positive side our present view can be classified broadly as an 'emergent' theory of mind that needs to be distinguished from other emergent theories advanced previously, mainly by the Gestalt school in psychology. It differs from these in several respects: first, the phenomena of subjective experience are not thought to be derived from electrical field forces or volume-conduction effects, or any metaneuronal by-product of cerebral activity. Our view relies on orthodox neural-circuit and related physiological properties. Second, there is no assumption of the need for an isomorphic or topological correspondence between the events of perceptual experience and corresponding events in the brain. I have conceived the mental properties to be *functional* derivatives that get their meaning from the way in which the brain circuits and related processes operate and interact, rather than in terms of isomorphic correlations. Reference to 'spatiotemporal patterning' of brain activity is sage as far as it goes, but this term fails to connote the operational derivation of the conscious properties that I have tried to emphasize. Third, the conscious subjective properties in our present view are interpreted to have causal potency in regulating the course of brain events; that is, the mental forces or properties exert a regulative control influence in brain physiology. The subjective conscious experience on these terms becomes an integral part of the brain process, rather than a correlated phenomenon as conceived by Koehler and others. The mental events are *causes* rather than *correlates*. In this respect our view can be said to involve a form of mental interactionism, except that there is no implication of dualism or other parallelism in the traditional sense. The mental forces are direct causal emergents of the brain process.[92]

[92] R.W. Sperry, "Mental Phenomena as Causal Determinants in Brain Function," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 163-77, p. 165.

As such, there is a 'mutual interdependence' implicit in the model proposed. Consciousness both shapes and is shaped by the underlying physical structures. It emerges as a consequence of the complex interactions between neurons and neural structures. Sperry argues that this position allows for a middle ground between purely materialist positions and dualist positions.[93] He suggests that allowing for this kind of subjective experience to have causal force also influences the determinism debate – subjective causality as an emergent consequent would allow for some control over volition that seems to be absent from many materialist argument.

A key concept added to the debate regarding the explanation of consciousness is the idea of virtual governors. A parallel can be drawn between complex electrical grids and our neural networks – consciousness and mind can be understood as a means of an emergent, automatic self-regulation of cognition, akin to the pattern of self-regulation that appears in interconnected generators. Dewan describes the phenomenon of virtual governors as follows:

> A generator in isolation does not give a very steady 60-Hz output. An electric clock attached to such a generator will become quite inaccurate after awhile. But, in remarkable contrast, when a large number of such generators are interconnected, they behave much more stably; that is, they all lock into step with one another or mutually entrain in a manner which is basically the same phenomenon as the fireflies flashing together or the heart cells 'beating as one.' In the case of the generators, the mechanism is easy to describe. If one generator leads the others in phase, i.e., if it is slightly faster, then its energy will be *absorbed*, not only by the load, but also by all generators which lag behind it. This will increase the load on the generator, forcing it to slow down a bit so that it won't 'get out of step.' If by chance it *lags* in phase, the other generators pump energy *into* it so that it catches up. Thus, generators which go a bit too fast are slowed down while those that lag are speeded up. They *pull together in frequency*. The generalization of feedback to a sort of mutual and shared feedback system should now be easy to see. The stability and accuracy of a *system* of generators is far greater than any single unit. This mutual entrainment is a splendid example of *self-organization*, and it is obvious that such a system can be regarded as a *single unit* so far as its function is concerned. Out of mutual entrainment has emerged what Wiener terms a 'virtual governor' which controls the entire system in a manner which uses feedback. This virtual governor is not located in any one spot in the system, but rather it pervades the system as a whole,

[93] Sperry, "Mental Phenomena as Causal Determinants in Brain Function.", p. 168-9

> so that it does not have a 'physical existence' in the usual sense. It is an *emergent property of the entire system* which goes far beyond what any single unit can accomplish in accuracy and power.[94]

It is plausible to imagine such a virtual governor in cognitive processes – in fact, there is a direct biological correlate. Neuronal transmission is the result of a significant amount of mutual interaction. In general, many other neurons attach to the dendrite of a given neuron, each provide chemical signals. Some of these signals are excitatory, while other are inhibitory. Whether the influenced neuron fires is a matter of additive activation – each excitatory neurotransmitter received moves the charge of the receiving neuron towards its activation threshold (depolarization), while each inhibitory neurotransmitter moves the charge away from its activation threshold (hyperpolarization). A neuron will only fire if it receives sufficient excitatory signaling to exceed its activation threshold, at which point the firing is unstoppable (the 'all-or-none' principle). Sodium rushes into the neuron while potassium rushes out, further depolarizing the cell, which causes an electrical cascade down the axon (this causes sodium and potassium pump activation in sequence), culminating in the release of a neurotransmitter into the synaptic cleft of a receiving neuron, providing excitation or inhibition of the receiving neuron, and so on. In essence, our brains are compact (but ridiculously complex) collections of interconnected chemical and electrical generators. If such self-governance can emerge as a property of inanimate systems, what would prevent them from emerging as a property of animate systems? Dewan suggests that "the 'virtual governors' of a power grid stand in relation to the individual governors in a way which is analogous to the way consciousness and mind stand in relation to the activity of the neuronal units of the brain."[95]

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[94] E. M. Dewan, "Consciousness as an Emergent Causal Agent in the Context of Control System Theory," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 181-98., p. 185

[95] Dewan, "Consciousness as an Emergent Causal Agent in the Context of Control System Theory.", p. 186.

Pluralism

The essence of the pluralistic objection to reductionism is that complexity ought to be welcomed – there are complex phenomena that cannot (presently) be expressed in terms of simpler theories.[96] Sarkar draws a parallel with molecular biology in his support of a pluralistic approach to organization:

> The important methodological point that should be made is that complexity should be embraced, not avoided merely to save the reductionist cause. This may well be the most important lesson to be learnt from the failure of genetic reductionism…Some robust organismic phenomena have stubbornly resisted all attempts at physical reduction. Dominance is a common property of traits. There is, as yet, no satisfactory molecular account, it relies on the topological properties of biochemical reaction networks. Topological properties are not physical properties; consequently, the epistemological weight in such explanations is not borne by the physical interactions involved. Topological accounts provide systemic explanations. The future will show the extent to which they are necessary, even at the molecular level. There is thus no reason for a reductionist triumphalism. Reductionism is an empirical issue and the evidence for or against it is not all in: only the future will show whether all biological phenomena at any higher level of organization will succumb to the lure of physical reduction.[97]

It does not seem to be too much of a stretch to apply the same type of reasoning to mental phenomena and cognition. The same caveats apply – whether mental phenomena are essentially reductive (and if so, to what extent) is ultimately an empirical issue that requires further research. Wimsatt suggests that understanding cognition need not draw significant lines between mental and physical phenomena – the explanation of cognitive phenomena requires input from several different systems at higher and lower levels of organization.[98]

Dupre is a key proponent of pluralistic influences on cognition. He first takes issue with the modular theory of the brain – the idea that our cognition is divided into coherent units that

[96] I include 'presently' in parentheses due to the innate plasticity of science. While the literature does not explicitly endorse the possibility of future research demonstrating definitively the reductionist, emergentist, dualist, etc., philosophies, it seems to me to be a requisite assumption of the possibility that one of these philosophies be ultimately true and triumphant.
[97] Sahotra Sarkar, *Molecular Models of Life: Philosophical Papers on Molecular Biology*, p. 98.
[98] Wimsatt, "Reductionism, Levels of Organization, and the Mind-Body Problem.", p. 263

are then linked together forming larger networks.[99] His skepticism stems from the essentially integrative processes involved in cognition – while cognition may involve 'modules', this approach does not explain how their output is then integrated into a decision of the whole person. Further, even knowledge of these modules does not necessarily offer any insight into the behavior or behavioral tendencies of humans. We are notoriously complex, and our decision-making processes may or may not involve a given module. Further, our behavior is not simply a product of any one given input (e.g., we are not simply the products of our genes). Rather, who we are is contingent upon a variety of inputs. He argues "there are many sources of information that are required in building a human body – genetic, cellular, physiological, and, especially at later stages, cultural. All are necessary, none is sufficient."[100] How we think and behave is influenced by many different variables, and it would be mistaken to assume that one necessarily is reducible to another. On Dupre's argument, focusing on single causes, or on small groups of causes, fails to capture the full complexity and intriguing detail of human volition. [101]

Dupre's second objection echoes that of Mills earlier – the overreliance on the genetic or biological influences on behavior has unpleasant consequences on our sense of morality and responsibility. Dupre specifically takes issue with evolutionary psychology and the general theory that behavior is evolutionary and adaptive. This position, Dupre argues, has the necessary consequence of justifying behavior that would otherwise be condemned:

> [A]lthough evolutionary theorists almost universally deny any such goal, it is hard to see how theories in evolutionary psychology can avoid offering justifications of behavior. If evolution has in fact shaped our behavior, it can only have done so by selecting physical structures, presumably in the brain, that cause the production of such behavior. To say that a certain behavior, which some find morally objectionable, is caused by a physical structure in my brain, is in effect to remove at least part of my responsibility for it…By presenting behavior as in an

[99] John Dupre, "Against Reductionist Explanations of Human Behavior," 153-71., p. 158-9
[100] Dupre, "Against Reductionist Explanations of Human Behavior.", p. 161-2
[101] Dupre, p. 171

> important sense biologically determined its ethical dimension is removed or attenuated.[102]

As with the discussion of Mills argument, I am sympathetic to this concern about the abnegation of free will and moral responsibility. However, as will be discussed in the second half of this chapter, it seems increasingly clear that there must be at least *some* element of determinism in any model of cognition. As we will see, this elemental determinism ought to not to be confused with a completely deterministic model, but should be understood to include strongly deterministic elements which may require conscious challenge and exploration.

Dupre suggests that pluralism has been critiqued historically for mistaken reasons.[103] First, some have criticized it in light of the influence of Cartesian dualism – many view the mind/body debate solely in terms of monism or dualism. As dualism has become less influential, many assume that monism is the only viable remaining choice. This is, he feels, fundamentally mistaken, as pluralism allows for the same explanations as monism (i.e., the characteristics that led to a rejection of dualism are explicable both in terms of monism as in terms of pluralism). The second critique stems from an over-reliance on the physical sciences. Many feel that the realm of scientific inquiry requires a monistic vision of the world, and that while this view has had some success, it has not, in his opinion, had enough success to warrant automatic deference.

Extending his argument, Dupre suggests categorizing reductionism differently than Sarkar and Wimsatt, proposing instead to discuss synchronic and diachronic reductionism. He uses diachronic reductionism to refer to reductions similar to Newtonian mechanics reducing to relativistic mechanics. He uses synchronic reductionism to refer to relationships between "coexisting theories addressed to different levels of organization."[104] Within this categorization

[102] Dupre, "Against Reductionist Explanations of Human Behavior.", p. 166
[103] John Dupre, *The Disorder of Things* (Cambridge: Harvard University Press, 1993), 90.
[104] Dupre, *The Disorder of Things*, p. 94-5.

model, he suggests that supervenience is not quite as tenable as previous theorists had held, and as a consequence, evidence for reductionism is equally tenuous:

> But supervenience is not *self*-evidently true. It is surely imaginable, for instance, that people with identical physical states, including states of the brain, might be thinking different things. This possibility is defended by those who believe that the content of a thought typically depends on facts external to the thinker. *Evidence* for supervenience, it seems, would have to be the kind of evidence necessary for reductionism. It would be evidence that higher-level phenomena are indeed determined by lower-level phenomena, or that identical (or sufficiently similar) lower-level phenomena do indeed produce the same higher-level phenomena. As is the case with evidence for reductionism generally, the problem is that where such evidence exists at all, it is in a narrow range of quite specialized cases, and the legitimacy of extrapolation to a general philosophical thesis is, to say the least, questionable.[105]

If supervenience is questionable, we are by definition questioning the explicit reduction and explanation of one level of organization in terms of another, a theme that has been recurring throughout this chapter. As a consequence, we cannot simply assign behavioral causation to one level over another – pluralism argues that every level of organization has some causative properties.[106] While Dupre does point out that he is rejecting the dualistic notion of a separate mind and body substance, he does not necessarily seem to reject emergentist elements like virtual governors. Concerning levels of organization, Dupre notes that higher order organisms possess a quiddity that lower organisms do not, and hence do not suggest themselves for reductive models. As he notes, "[a] sponge, perhaps, is a multicellular organism that *is* nothing but an assembly of cells. But an aardvark would more naturally be treated as an assembly of organs and other complex systems."[107] Complexity, therefore, lends itself to pluralistic models of behavior.

[105] Dupre, p. 97.
[106] Dupre, p. 101.
[107] Dupre, p. 102.

The difficulty for this dissertation, however, comes from Dupre's treatment of mental phenomena. He argues, counterintuitively in this author's opinion, that only the 'how' of behavior is explicable in reductionist terms.[108] Specifically, he suggests that the 'what' of behavior can only be understood in terms of higher levels of organization (e.g., systemic or social), and that significant work remains to decide what actually ought to be explored in neuroscientific terms. As a field of study, psychology is still concerned with the 'how' and 'what' of behavior, but it ought not to look for answers in terms of biological reduction, as biology is only tangentially related to behavior.[109] Causal explanations, he argues, are best explored in social and individual (i.e., higher) levels of organization:

> The contrary intuition is that whatever mental properties pertain to an individual, they must do on the basis of some structural features of that individual, even if this is so in no stronger sense than that of supervenience. But I hope it is clear by now that there is no need to insist on even such a weak physicalism. Beliefs, often at least, explain actions. Actions, again often, take place in social contexts that have much to do with determining what kinds of actions they are…The reductionist will insist that the social is merely a product of the diverse behaviors of the many individual agents by which it is constituted. But it is equally possible, and perhaps more natural, to think of the social and the individual as each constituting partially autonomous, causally efficacious, domains. Sometimes we can explain social phenomena in individual terms, and sometimes individual behavior should be explained by appeal to social factors. And as with my claims in the previous chapter about phenotypic causes of genetic change, when I speak here of explanation, I do mean *causal* explanation.[110]

As I indicated above, it is unclear why Dupre seems so committed to maintaining behavioral causation at higher levels of organization, especially if he maintains a model in which there are complex interactions between levels of organization. It would seem more plausible to defend a model that allows for mutual influence across levels. Phenotype influences behavior and behavior influences phenotype; one does not have to look too far for examples of this.

[108] Dupre, p. 106.
[109] Dupre, p. 152.
[110] Dupre, p. 157.

Overeating, for instance, is a behavior that influences the organism at many levels. At the social level, it can lead to deterioration of relationships. At the individual level, it can affect self-esteem and body images. At the organism level, it can lead to changes in physical shape, sleep discontinuity (e.g., sleep apnea), stress, hypertension, and elevated risk for other comorbidities. At the cellular level, it can lead to over- or under-production of hormones and other chemicals and chemical messengers. Each of these will affect other levels of organization, and hence, in this author's opinion, each should be seen as causative.

The Shift Towards Determinism

What emerges from the discussion so far is a complex model of cognition with interaction at multiple levels. It is reductive in the sense that there is a physical material upon which it is built, but there are obviously questions as to what is explicable in terms of what. However, what seems to be a fair conclusion is that if levels interact with each other, then disruptions or distortions in overall cognition can come from many different sources and different levels of organization. This would allow for psychosocial stressors like job loss or the death of a spouse to cause depression and influence multiple levels of cognition, in just the same manner as dysregulation of dopamine or serotonin can cause depression and influence multiple levels of cognition. This is germane because our access to the levels of our cognition is limited – while I may be able to control some thought processes (see the next chapter), I cannot necessarily pick what neuron or pathway is activated, let alone how much of a given neurotransmitter I produce. As such, there are necessarily elements of my cognitive process that are out of my control. These will be addressed in more detail in the next chapter. For the moment, it is necessary to explore the philosophical consequences of this lack of control.

It would seem that some credence is given to deterministic models – cognition is dependent upon neuroanatomy, which is influenced by genes, gender, socioeconomics, and myriad other foundational influences (as noted in Hospers' argument). These shape and constrain the options considered by an agent during cognition – determining, as it were, the direction the cognitive process will go. There is an ongoing debate as to how much influence these factors have on behavior; the most credence is given to models that are not fully deterministic, but maintain some semblance of indeterminacy. Instead of a 1:1 cause-effect ratio, there are multiple possible effects. This topic will be explored in later sections on the neuroscience of choice.

DETERMINISM

The Libet Experiments

In making the switch from discussions of reductionism to discussions of determinism, it is useful to consider the experiments of Benjamin Libet. Seeking an understanding of the phenomena of conscious will, Libet constructed a means by which the time of neurological activity (measuring the readiness potential [RP]) versus perception of will to act could be traced. In previous experiments he had found that neural activity preceded movement by more than one second. He concluded that:

> The brain was evidently beginning the volitional process in this voluntary act well before the activation of the muscle that produced the movement. My question then became: *when* does the *conscious* wish or intention (to perform the act) appear? In the traditional view of conscious will and free will, one would expect conscious will to appear before, or at the onset, of the RP, and thus command the brain to perform the intended act. But an appearance of conscious will 550 msec, or more before the act seemed intuitively unlikely. It was clearly important to establish the time of the conscious will relative to the onset of the brain process (RP); if conscious will were to *follow* the onset of RP, that would have a fundamental impact on how we could view free will.[111]

[111] Benjamin Libet, "Do We Have A Free Will?" in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), p. 49.

Indeed, the common perception of cognition is that we consciously will an action before our body begins the necessary processes to bring it about. After all, conscious will should be a catalyst, not an afterthought. This was not, however, what his experiment found:

> For groups in which all the voluntary acts were freely spontaneous, with no reports of rough preplanning of when to act, the onset of RP averaged -550 msec (before the muscle was activated). The W [Will to Act] times for first awareness of wish to act averaged about -200 msec for all groups. This value was the same even when subjects reported having preplanned roughly when to act! If we correct W for the -50 msec error in the subjects' reports of timings of the skin stimuli, we have an average corrected W of about -150 msec. Clearly, the brain process (RP) to prepare for this voluntary act began about 400 msec before the appearance of the conscious will to act (W).[112]

Counterintuitively, Libet found that the decision to act appears before our perception of having willed to act. Volition, then, is an unconscious process – the perception of having 'chosen freely' appears to be an illusion or afterthought. Libet notes that the appearance of will does appear before the muscle contracts, and hence appears as an intermediate stage between the unconscious choice to act, the perception of choosing, and the actual action itself. Because of this intermediate stage, Libet suggests that consciousness acts as a kind of 'veto function' on these unconsciously determined processes – the RP spike appeared when the subject chose to act, but no such similar spike occurred when the subject decided not to follow through on the action willed.[113] This veto function is a form of conscious control, he continues, that does not stem from a corresponding unconscious source.[114] This delay between unconscious processing and action is not affected by the amount of time before deciding – Libet noted that "one may, after all, deliberate all day about a choice but never act; there is *no voluntary act* in that case."[115] Even in these cases, however, the delay between the unconscious initiation and the perception of

[112] Libet, "Do We Have A Free Will?", p. 51.
[113] Libet, p. 51-2.
[114] Libet, p. 53.
[115] Libet, p. 54.

will was present; based on this evidence, Libet argues that this would apply to any sort of decision. Free will, then, is not understood to be an initiator of action in Libet's model – rather, it would necessarily be a control mechanism on an underlying impulse to act. He argues for a model in which multiple urges 'bubble up' from the unconscious, and we then choose to inhibit or not inhibit those impulses.[116]

Gomes and Claxton offer comments on these findings. Gomes notes that despite Libet's suggestion that veto mechanisms are consciously controlled, they do not thereby return *the actual impetus to act* to the agent. Thus free will does not allow for the creation of action, but simply for censoring them.[117] In fact, Gomes argues that perhaps this veto mechanism is not an expression of choice – perhaps, instead, the agent simply lacked the final catalyst to action; everything was present, except for the final variable allowing the action to proceed:

> Retrospectively, one experiences that one 'had almost started' the act and then refrained from carrying it out. Of course, 'having almost started' means not having started…As I have already said, experience of deciding to act is an integral part of the experience of the action itself. And very often the prior experience of an intention is absent: we experience only the suddenly decided and performed action. Indeed, even in the case in which the subject experiences that an impending action has been aborted, it is debatable whether the word 'veto' is a good description of his experience. In some of the spontaneous cases, a more suitable description is perhaps that the final decision to move simply did not occur, although the subject has the experience that it almost occurred. It seems it is more a case of *not* having decided than of positively vetoing an impending event.[118]

Gomes agrees with Libet regarding the unconscious initiation of action, but suggests that we ought not view the readiness potential as the start of volition or will. Rather, Gomes argues that the initial readiness potential is causal in intention and volition, and that following this trigger, a

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[116] Libet, p. 54.
[117] Gilberto Gomes, "Volition and the Readiness Potential," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), 59-76., p. 64-5
[118] Gomes, "Volition and the Readiness Potential,", p. 67.

sequence of events culminates in our conscious experience of the decision to act. He does argue that we exert control over the final decision to act (the final variable mentioned above); after the final neural impulses "comes consciousness of the action as caused by a decision of one's own."[119]

Claxton notes that there are other examples paralleling Libet's experiment. He describes a series of demonstrations by Grey Walter that noted preconscious triggering of choice:

> Patients with electrodes implanted in the motor cortex were invited to look at a sequence of slides, advancing from one to the next, at their own speed, by pushing a button. Unbeknownst to them, however, the button was a dummy. What actually advanced the slides was a burst of activity in the motor cortex, transmitted directly to the projector via the implanted electrodes. The patients reported the curious feeling that the projector was anticipating their decision, initiating a slide change just as they were 'about to' move on, but before they had 'decided' to press the button. In other studies, Grey Walter found that EEG readiness potentials taken from RAF bombardiers, as they were lining up to drop a simulated bomb, preceded the conscious decision to press the bomb release button.[120]

Claxton suggests that the model that emerges from considering Walter's and Libet's experiments are comparable to a racing start: the preconscious brain prepares the body for action, a metaphor Claxton compares to stepping on the brake and accelerator at the same time. Whether we act or not is then decided in our perception of choice. In describing these veto models, he states "thus it is that Libet and others have come to associate volition more with the vetoing of action than its instigation: Richard Gregory's 'free won't' rather than 'free will.'"[121]

What emerges from these arguments is a generalized model of cognition in which our experience of will or volition is a secondary phenomenon – the decision to act is initiated preconsciously, that is, prior to our awareness of it. Our popular conceptions of autonomy are

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[119] Gomes, "Volition and the Readiness Potential,", p. 72.
[120] Guy Claxton, "Whodunnit? Unpicking the 'Seems' of Free Will," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), 105.
[121] Claxton, "Whodunnit? Unpicking the 'Seems' of Free Will," 109.

predicated upon some sense of control – an idea that our actions are the result of our own agency, and not that we are simply responding to stimuli or that something is controlling us. Intuitively, being controlled is antithetical to theses of autonomy – expressed almost tautologically, if we are being controlled, by definition we are not in control of our actions. The difficulty, however, is that this is the model that seems to be borne out by experimentation – there are compelling and causative elements of our volitional process that are not under our control. Hence, as a bare minimum, we must assume at least some deterministic elements in our conception of agency and 'autonomy'. But like reductionism, determinism has multiple meanings, and it is necessary to understand what is meant by deterministic elements.

Why is this meaningful?

What is the essential conflict? Why is the discussion of determinism meaningful? At its heart, the discussion concerns essential questions of human volition, motivation, decision-making capacity and responsibility. We generally believe that human beings are praiseworthy or blameworthy. This model of subjective experience is ubiquitous, and is reinforced every time we have to make a decision, regardless of whether it is a decision as inconsequential as what type of bread to buy or whether we want to undergo another round of chemotherapy. This everyday accounting of subjective experience is a model of folk psychology – a plain-language accounting of phenomena we all experience.

The Folk Model – Our Everyday Experience of Free Will

There is a common experience in cognition concerning the question of free will: we feel consciously aware of the process of choice and deliberation. We persistently feel a sense of ownership and authorship of our actions, and hence corresponding feelings of pride and embarrassment when we excel or err. McCrone summarizes the experience quite cogently:

> Free will seems a straight-forward enough business. I feel the mental effort of making a choice and anyone who tells me my choices are predetermined can quickly be proved wrong – I will simply do the opposite of what's expected. Of course there are a few mysteries. When I crook a finger or raise a hand, it is hard to be sure how I really make these simple actions happen…So there are some complexities to the story. But the ancient tripartite division of the mind into thought, feeling and will seems indisputable. Nestled somewhere in the humid folds of our brains must be a moving soul-stuff or at least some clever neural machinery of volition. Such is the folk psychology view of volition and already many of the standard suppositions about the nature of freewill are apparent. It is seen as a unitary faculty – any differences are of degree rather than kind. It is innate – all humans are born with the power, although its shoots may need nurturing to grow healthy. It is dimensionless – the willing of an act is a point-like event, clearly separate from deliberations that may have preceded it. And free means free – don't you dare call it an illusion.[122]

This folk psychology view is deeply engrained in many people – it is very difficult to challenge and very resistant to change. As Claxton notes, our perception of folk psychology explanations are that they do a good job of explaining our everyday experiences. Unless we consciously look for breakdowns or errors in this process, it has a tendency to be self-perpetuating, and just seems right. However, these common 'seems' can be unreliable and inaccurate.[123] The extent of the inaccuracies will be explored shortly.

What, then, is at stake? What could the basis of objecting to this folk psychology model be? One might approach the issue from a purely philosophical question of the nature of causality. If we accept that effects must have prior causes, then we can challenge the folk psychology model on the basis of adequate versus inadequate exploration of causation. We may not necessarily be aware of all of the elements relevant to or explicitly causing the given action. From a psychological viewpoint, we may note that the cognitive biases of the agent, conscious and un-/sub-/preconscious elements shaped the decision, but that we only were aware of the surface level phenomena. Elements outside of consciousness are avolitional, or at least not

[122] John McCrone, "A Bifold Model of Free Will," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), 242.
[123] Claxton, "Whodunnit? Unpicking the 'Seems' of Free Will.", p. 99-100.

controlled by us – therefore we do not exert the conscious control that we believe we do. From an anatomical basis, we have Libet's own experiments, which demonstrate causative and unconscious activation of specific cortices relevant to the feeling of having 'willed' something – therefore, our ownership of the subsequent action might not be as complete as we believe it to be. From a scientific basis, one could argue that the universe has causal laws that are inherently deterministic (in the sense of being predictable, explanatory, and consistent), and that we ought not to conceive ourselves as being exempt from these laws. These concerns will be addressed in a brief exploration of the arguments of Searle, Libet, and Sappington.

Searle

Searle raises several issues at stake in the debate. There is a concern about taking ownership and responsibility for actions – much in the same manner as we do not hold individuals responsible for actions done under hypnosis, if psychological determinism were true (that our actions are caused by underlying psychological processes), we ought to remove any discussion of morality from discusses of humanity. If we are simply our psychological processes, we are not moral agents. This will be a recurring theme in this chapter, and as such, it is not necessary to belabor this point at the present. It is worth exploring the question of psychological determinism, however, as Searle's question speaks to issues fundamental to this dissertation, and explored more fully in the next chapter. In Searle's argument, psychological models are not incompatible with free will:

> Is all behavior determined by such *psychological* compulsions? If we try to treat psychological determinism as a factual claim about our behavior, then it seems to be just plain false. The thesis of psychological determinism is that prior psychological causes determine all of our behavior in the way that they determine the behavior of the hypnosis subject or the heroin addict. On this view, all behavior, in one way or another, is psychologically compulsive. But the available evidence suggests that such a thesis is false. We do indeed normally act on the basis of our intentional states – our beliefs, hopes, fears, desires, etc. – and in that

> sense our mental states function causally. But this form of cause and effect is not deterministic. We might have had exactly those mental states and still not have done what we did. As far as psychological causes are concerned, we could have done otherwise. Instances of hypnosis and psychologically compulsive behavior on the other hand are usually pathological and easily distinguishable from normal free action. So, psychologically speaking, there is scope for human freedom.[124]

This seems to be counterintuitive, however. Searle fiats the ability to act differently with identical causal psychological states, which seems to be an unfounded statement. It seems likely that perhaps there was an incomplete accounting of causal factors – that while the situations may appear similar, there were other elements present which inhibited the initiation of action. In light of myriad un-, sub-, and preconscious elements in cognition, it seems entirely plausible that we manifest different behavior simply because we are not aware of all of the germane factors affecting agency.

A further irony is that while Searle argues for ownership of action, there is a fundamental paradox: How do we then account for ownership of an action if it doesn't stem from our character (and hence psychological state)? Character, as a recurrent pattern of belief and behavior, is not a consistently conscious and willed phenomenon. Character stems from experiences and causal elements outside of our control (e.g., genes, early environment, parenting, gender, race, economic status, etc.) – determined elements – and is not something about which we consistently think. The availability heuristic, a concept more fully explored in the next chapter, notes that our cognition tends to be shaped by events that are easily accessed in our memory, a deterministic model. My choices are also shaped by previous events about which I have no working memory (i.e., I do not consciously think about them), and as such, I cannot control their influence on my present choice or volitional process. One's psychological state is much more causative and deterministic than Searle suggests.

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[124] Searle, *Minds, Brains, and Science*, 91.

Searle ultimately argues that the psychological model is less worrisome to free will theorists than models stemming from physical models of microelements:

> This is an absolutely fundamental point in this chapter, so let me repeat it. The form of determinism that is ultimately worrisome is not psychological determinism. The idea that our states of mind are sufficient to determine everything we do is probably just false. The worrisome form of determinism is more basic and fundamental. Since all of the surface features of the world are entirely caused by and realized in systems of micro-elements, the behavior of micro-elements is sufficient to determine everything that happens. Such a 'bottom up' picture of the world allows for top-down causation (our minds, for example, can affect our bodies). But top-down causation only works because the top level is already caused by and realized in the bottom levels.[125]

This seems overly dismissive of psychological determinism – after all, as noted above, there are significant causal elements of cognition that are psychological in origin. There is a potential out, however, in that it is likely Searle is referring to hard determinism (a very strict form of determinism discussed below) rather than underdeterminism – which refers to avolitional causative structures that strongly influence the agent, but do not remove all actual agency. Underdeterminism will be discussed more fully below.

Libet

Libet notes that we face a fundamental problem – in the course of studying consciousness and our experiences we feel free, but we cannot reconcile this with our understanding of deterministic processes in the natural world. We 'know' we have volitional control, but we also now that this isn't in accord with natural laws.[126] This conflict is significant; in fact, Libet notes that history has demonstrated it to be beyond the ken of some of the most influential thinkers in philosophy:

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[125] Searle, *Minds, Brains, and Science*, p. 94.
[126] Benjamin Libet, Anthony Freeman, and Keith Sutherland, "Editor's Introduction: The Volitional Brain," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), p. ix.

> Our sense of free will (volitional control) depends upon a balance between reliability and flexibility in relation to cause-and-effect. Without the former, all outcomes would be arbitrary; without the latter, all outcomes would be predetermined. In neither case would there be any way of putting one's will into effect. So much is clear, yet establishing that precarious balance has proved so difficult that Kant himself declared 'freedom of the will' to be one of the only three metaphysical problems which lie beyond the powers of the human intellect.[127]

Two models that recur in the debate are libertarianism and compatibilism – philosophies that attempt to preserve some volitional elements of cognition and choice from underlying deterministic causality (which will be discussed below). Libertarianism generally argues that individuals are moral agents who can create their own goals and motives – actions originate with the moral agent, not with the agent's surrounding environment or external stimuli. Compatibilism generally holds that freedom consists of "being free from outside constraints and abnormal internal compulsions."[128] Libet suggests that compatibilism tends to be a more commonly held opinion among philosophers and the general public. The public, he suggests, would reject theories that argue for more deterministic models, as " a common reaction, after all, to the suggestion that we are *not* ultimate self-originators – that we could *not* have willed otherwise in a given situation – is to suppose that morality, responsibility, justice, fairness, and the social order itself are deprived of a necessary foundation."[129] These same concerns were raised with regard to reductionism, and we will return to these concerns in discussing objections to determinism below.

Ultimately Libet suggests that perhaps it would be better to simply assume that free will and volition are not essentially deterministic, as they are the basis of many social and cultural

[127] Libet, Freeman, and Sutherland, "Editor's Introduction: The Volitional Brain," p. ix.
[128] Libet, Freeman, and Sutherland, p. xiv.
[129] Libet, Freeman, and Sutherland, p. xiv.

mores. Absent hard data proving or disproving free will or determinism, he suggests that we err on the side of caution:

> Present-day physics does not provide for the possibility of free will, but neither does it rule it out – unless one subscribes to the view that presently known physics is final and complete. Given the immense complexity of the brain, it probably is not feasible to demonstrate unequivocally the presence or absence of free will through analysis of neurophysiological processes. But further experiments can give us insight as to the way it presumably works, if it exists…Seeing as our experience is one of agency and free will, and seeing as the entire religious, ethical, cultural and legal system of the western world is based on such an assumption, then it might be much better to assume that this is the position until science tells us, unequivocally, that this is not the case. That way we should all be 'innocent until proven guilty.'[130]

But this raises a necessary question – do we simply adhere to models because they are fundamental to our current worldview? This type of mentality justified a variety of questionable pseudoscientific practices, including belief in spontaneous generation, phlogiston, Lamarckian evolution, geocentrism, etc. As Kuhn argues, scientific progress occurs as the result of paradigm conflicts and resolution – as evidence mounts against a currently dominant paradigm, as more questions are found that cannot be reconciled with the current theory (or can only be reconciled with tortured logic and loose inference), we have more reasons to reject the dominant paradigm and adopt the challenger. Quite simply, there are too many unanswered questions to maintain current beliefs. Libet's experiments note an unconscious volitional precursor, indicating backstage cognitive processes that by their nature are avolitional and deterministic. Cognitive and social psychology note a variety of heuristics that act as automatic filters during decision-making and cognition. These are all compelling arguments that our cognition is not quite as free as we would like to maintain – in short, these are all compelling arguments that the current 'folk' paradigm of psychology and subjective experience ought to be replaced with something that incorporates more deterministic elements.

[130] Libet, Freeman, and Sutherland, "Editor's Introduction: The Volitional Brain," p. xxi.

Sappington

Sappington notes that both libertarians and compatibilists can admit that choices influence behavior, and that there can be causative influences external to us that shape our behavior.[131] Neither position is arguing for a radical freedom uninfluenced by external or past factors. We have personalities, after all, and have previous experiences which can affect how we view situations and circumstances. Sappington notes that the data on personal choice prove neither free will nor determinism, as they do not address the origin of choice or personal goals, or why one goal or value may be given priority over others, or whether genetic factors or past learning history are causative in choice.[132] He suggests that some theorists have been able to demonstrate compatibility between free will and scientific tasks like control and prediction, but they have been unable to demonstrate that there is any reason to view humans as exceptions to the rules governing other natural phenomena. The same types of argument justifying free will in humans can be used to justify free will in other chaotic systems that we do not traditionally view as being free (pendulums, weather systems, leaf distributions, and mathematical equations).[133]

He suggests that free will and determinism can act as meta-assumptions in guiding scientific research. Experiments proceeding from different meta-assumptions will explore different constructs and thereby gather different data. Theories adopting free will as a meta-assumption might explore subjective "purpose or conscious choice"; theories adopting deterministic meta-assumptions would be less likely to explore these elements.[134] Sappington concludes that scientific data will never allow us to answer the question of subjective free will versus determinism. He makes a distinction between subjective consciousness and free will –we

[131] A.A. Sappington, "Recent Psychological Approaches to the Free Will Versus Determinism Issue," *Psychological Bulletin* 108, no. 1 (1990): p. 26.
[132] Sappington, "Recent Psychological Approaches to the Free Will Versus Determinism Issue," p. 26.
[133] Sappington, p. 27.
[134] Sappington, p. 27.

are aware of and can test subjective consciousness and choice; free will is not testable. Fundamentally, he argues, proving either free will or determinism requires an impossibility – proving a negative claim:

> It has been argued previously that the data covered in this article are inadequate for demonstrating the existence of free will. It is now argued that no empirical data can settle the free will versus determinism issue. Logically, to prove that free will exists, it would be necessary to show that at least some choices cannot be explained in terms of factors outside the person. To prove the determinism position would require a demonstration that no choices exist that cannot be explained in terms of factors outside the person. To prove either position would thus require proving a negative, which cannot be done conclusively. Thus, free will is not a scientific construct in the same sense as conscious choice, and the free will versus determinism issue cannot be settled scientifically.[135]

It would seem, then, that we are left in the initial quandary of having a concept that can neither be proven nor tested; whether we actually exert control over the causes of our actions may not be answerable in scientific study – it may not be a question so much of physics as metaphysics.

Key Definitions in the Determinism Debate

Before the conceptual discussion begins, it is useful to lay out a few key differences between the ideas at hand. These differences are not purely semantic – they have strong consequences not only for accounting for human cognition and behavior, but also for issues on the periphery of epistemology and ontology – questions of morality and responsibility. The two key concepts that must be addressed are fundamentally opposed: indeterminism and determinism.

Defining Indeterminism

The traditional alternative to determinist schools of thought is that of indeterminism. Where determinism sees strict causation, indeterminism sees probabilities. Where determinism

[135] Sappington, p. 27.

sees necessity, indeterminism sees possibility. In the experiment discussed above, Libet defined free will in accord with what he called the common view:

> First, there should be no external control or cues to affect the occurrence or emergence of the voluntary act under study; i.e., it should be endogenous. Secondly, the subject should feel that he/she wanted to do it, on her/his own initiative, and feel he could control what is being done, when to do it or not do it. Many actions lack this second attribute.[136]

Despite the prevalence of actions lacking this second attribute, Libet suggests that actions are free, but perhaps not free in the usual sense. After all, the common view of free will is not veto capacity, but actively willing one of many alternatives.

Enç defines indeterminism as "the thesis that starts from the formation of the beliefs and desires of an agent, through a decision, an intention, to the execution of the action [and that thesis] must essentially contain an indeterministic element."[137] In essence, the process must originate with the plans of the agent and must have alternatives available to the agent. These alternatives cannot be simply for show – they must be genuine options that the agent could make, and the choice finally made must stem from the agent instead of some determining element or elements. This does raise a compelling question, however – how do we reconcile the two apparently disparate concepts of agent-based choice and character-based agency. Ultimately who we are is a culmination of a lifetime of experience and subjective assessment; as will be demonstrated in the next chapter, this experience and assessment is not always a conscious process. In fact, compelling arguments have been made suggesting that these deeply formative un-, sub-, and preconscious elements are also causative – our character is shaped by elements outside conscious choice. We cannot selectively filter our unconscious processes – certain things happen automatically and outside our perception which affect how we think and act. Agency,

[136] Libet, "Do We Have A Free Will?", p. 47.
[137] Berent Enç, *How We Act: Causes, Reasons, and Intentions* (Oxford: Clarendon Press, 2003), p. 163.

then, is not entirely a volitional process; it must contain determined elements if it is to accurately portray our actions. Agency that does not admit this backstage cognition is homuncular – it's as if there were a little man selectively deciding what will affect us and what will not, a theme that will be explored in chapter four in the discussion of popular autonomy models. Cognitive and social psychology tell us that we cannot simply fiat these little men; our cognitive processes are not quite so choosy – a theme explored more fully in the next chapter. For the moment, let it suffice to note that this type of agency does not gel well with Libet's experiments; action has unconscious, non-agentic roots.

Defining Determinism

Determinism, however, is perfectly compatible with Libet's experiment – deterministic systems do not fiat such homuncular thought. Instead, un-, sub-, and preconscious processes (hereafter referred to as backstage cognition) are perfectly compatible with the volitional data suggesting our 'choice' to act is caused by elements outside our conscious awareness. Dorato defines determinism as "a scientific-philosophical doctrine according to which the state of a physical system at one instant of time *t* (whatever that means) univocally fixes any other temporal states of that system, past or future."[138] In this model, there is a 1:1 relationship between a state of the universe and it's following arrangement – there are no contingent events or other possibilities. If, he argues, our actions conform to this definition, we cannot have acted any differently in the past, and by extension cannot act except as we are determined to do. This does not equate to fatalism, however, which he distinguishes as arguing that certain events are fated to occur, although the paths we may take to reach them can be variable. Fatalist arguments suggest that certain events must happen, but there are multiple causal pathways that can bring them

[138] Mauro Dorato, "Determinism, Chance, and Freedom," in *Between Chance and Choice: Interdisciplinary Perspectives on Determinism*, ed. Harald Atmanspacher and Robert Bishop (Charlottesville: Imprint Academic, 2002), p. 340.

about; determinism, on the other hand, argues that there is only one causal pathway between events.

Sappington, among many others, notes that there is a logical consequence of upholding a deterministic model of human behavior. Specifically, if our actions are constrained and no other outcomes are genuinely possible, it makes no sense to argue for concepts like morality. Moral decisions are predicated on at least the capacity to choose between alternatives – choice, in the deterministic model proposed, is illusory, and as a consequence, so is the concept of morality.[139] This point is contentious, and will be revisited when challenges to deterministic models are discussed. Sappington notes a distinction between "hard" and "soft" versions of determinism (the latter is frequently referred to as 'compatibilism' or 'libertarianism') – in soft deterministic models, certain elements of choice and volition are determined, but others are freely chosen:

> Libertarians agree with Alexander Pope that human beings are somehow a special case in that they must be understood differently than other natural phenomena. Human choices are not determined by external factors; the cause of a choice is held to be the person who makes it. People must be viewed as active agents. It is true that people are not free to implement all of their choices; they are faced with external constraints that serve as barriers and with personal constraints such as lack of ability. Nevertheless, in any situation, people are always free to make choices and the choices themselves are not determined by any factor outside the individual[140]

This position has been critiqued by many, and at present it is sufficient to note that there is not a clear distinction between soft and hard determinism – all thought is predicated on an underlying substrate or structure, which by definition has rules to follow. We cannot have thoughts without simultaneously having some underlying neural activity, which is a determined action.

[139] Sappington, "Recent Psychological Approaches to the Free Will Versus Determinism Issue," p. 19.
[140] Sappington, p. 20.

Dowe suggests that there are at least four compelling variants of determinism.[141] (all subsequent discussion in the next few paragraphs are drawn from Dowe's work). The first variant is causal theory, which suggests that every event has a prior and sufficient cause (i.e., for every event we can name, there is a corresponding cause that was sufficient to bring the event about). In this model, behavior is explicable in terms of root causation; e.g., the desire to drink is brought about by genetic or social etiologies (upbringing, education, etc.). Dowe notes that there are at least two subsequent challenges this account encounters: first, defining causation is problematic itself, therefore defining determinism in light of causation merely makes the question more opaque; second, causation is asymmetric with respect to time (causes precede effects), while determinism is does not necessarily have this asymmetry (effects can be contemporaneous with their causes). A second variant of determinism stems from Popper's predictability theory; that is, "a system is deterministic if and only if all its states are predicable by the right kind of being with knowledge of the present state and the laws of nature."[142] This being possesses finite knowledge, but it is enough to predict all of the possible and subsequent states of the system. The objection raised by this definition is the concern that it conflates epistemology with ontology (predictability is epistemic while determinism is ontological). Dowe's third variant stems from Bertrand Russell, who suggests that the universe is deterministic if there is a functional relationship between variables at one time with variables at all other times. This, however, raises questions of possibility – Dowe notes that Russell admitted that the model he proposed was problematic. It was equally possible that the world would be deterministic as indeterministic. The fourth variant uses the idea of physical necessity in

[141] Phil Dowe, "What is Determinism?" in *Between Chance and Choice: Interdisciplinary Perspectives on Determinism*, ed. Harald Atmanspacher and Robert Bishop (Charlottesville: Imprint Academic, 2002), 309-19, p. 309-11.
[142] Dowe, "What is Determinism?", p. 309.

infinitely possible worlds, such that "a physically possible world W is deterministic just if for any other physically possible world W', if W and W' agree at any time then they agree at all times."[143] Dowe suggests that this model faces the challenge of the difference between accidental and nomological regularities, but that it is the "front runner for the appropriate theory of determinism."

Unlike Dowe, I suggest that the second model – the Popper model – is a more likely account of deterministic thought, at least as it pertains to human cognition. Underlying cognitive processes are causative, in the sense that they individually or collectively can provide strong impetus to act, if not explicit action itself (these underlying cognitive processes will be explored in greater detail in chapter two). Similarly, Bargh and Ferguson suggest that determinism underlies every element of human choice and volition – the causative elements of human behavior are both conscious and unconscious.[144]

Concepts in the Determinism Debate

In discussing the question of deterministic causation in cognition, what becomes evident is the strenuous nature of the debate and the tenacity with which the advocates hold to their positions. The difficulty, however, is that to date no approach has been able to claim absolute victory – while significant elements of cognition can be assessed objectively (from a third-person perspective), we have not had many inroads into the subjective phenomena of cognition (from a first-person perspective). As Libet argues, both determinism and non-determinism (a catch-all phrase for theories that are not innately deterministic) are "non-proven speculative beliefs."[145] Both Gomes and Spence and Frith note that subjectively we experience our cognition as

[143] Dowe, "What is Determinism?", p. 311.
[144] John A. Bargh and Melissa J. Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," *Psychological Bulletin* 126, no. 6 (2000): p. 926.
[145] Libet, "Do We Have A Free Will?", p. 56.

undetermined – i.e., our phenomena of cognition is one of conscious deliberation, volition, and decision.[146] However, Spence and Frith note that while our subjective experience may be one of volitional control and decision, from an objective perspective, our behavior may be causally explained and predicted. They note that "in this example the subject experiences one reality (their freedom 'to choose'), while another, material reality, coincides with (and indeed pre-empts) their experience."[147] As such, we find ourselves experiencing the original quandary – how can we reconcile an essential element of our subjective experience with objective knowledge of deterministic causation? Dorato argues that there are multiple methodologies by which one can approach the relationship between determinism and subjective indeterminism. These methodologies generally agree as to what constitutes determinism, but differ significantly when exploring and explaining the phenomena of free will.[148] These positions will be elucidated and explored below.

Indeterminism

Several theorists have endeavored to demonstrate the innate indeterministic cognitive and volitional capacities of human moral agents. The justifications for belief include such disparate approaches as discussions of quantum mechanics, voluntary decisional mechanisms, folk psychology, questions of ultimate responsibility, probabilistic causality, etc. The degree to which they can plausibly justify indeterministic human agency varies accordingly, and each leads to questions challenging the stability of their foundation. Indeterministic theories must account for the success of mechanistic accounts of the natural world, as well as the general reliability of prediction by third-person observation of human behavior. The above bases for

[146] Gomes, "Volition and the Readiness Potential,", p. 60; Sean A. Spence and Chris D. Frith, "Towards a Functional Neuroanatomy of Volition," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), p. 12.
[147] Spence and Frith, "Towards a Functional Neuroanatomy of Volition," p. 12.
[148] Dorato, "Determinism, Chance, and Freedom," p. 340.

justification will be discussed through discussion of the arguments of Kane, McCrone, Enç, Libet, Eccles, and Gomes. Each will be addressed in turn.

Kane bases his defense of probabilistic causation on two principles: AP, the principle of alternative possibilities, and UR, the principle of ultimate responsibility.[149] Alternative possibilities has a long history in the indeterminism debate, and generally refers to the necessity that an agent possess alternative choices for a decision to be considered free. Kane argues that ultimate responsibility is fundamentally more important than alternative possibilities – we need the agent to take ownership of her actions in order to genuinely say that her choice was free. Ultimate responsibility is similar to existential notions in Sartre, in that it suggests that past choices are character-forming, and that therefore we take responsibility for who we were, are, and are becoming by the choices we make. Kane calls these 'self-forming actions'.

In circumstances of deliberation and choice, Kane proposes that disparate desires, motivations, and goals conflict at a cognitive level, yielding an 'indeterministic noise' – a barrier to decision-making that must be overcome.[150] The 'noise' stems from one desire's effort to inhibit the other – each is attempting to defeat the opposing impulse so that the desire may be fulfilled. Eventually one desire reaches in activation threshold, which initiates action and choice. Kane suggests that we see the agent as attempting to solve two cognitive problems at the same time – the agent wants both choices, but action is thwarted until one or the other wins out. In this manner, agents can be held responsible for actions that may have gone awry – for instance, the assassin who misses his target is still responsible for his action, because part of him intended to

[149] Robert Kane, "Free Will, Determinism, and Indeterminism," in *Between Chance and Choice: Interdisciplinary Perspectives on Determinism*, ed. Harald Atmanspacher and Robert Bishop (Charlottesville: Imprint Academic, 2002), 371-406, p. 376-7.
[150] Kane, "Free Will, Determinism, and Indeterminism,", p. 383.

succeed.[151] Kane distances his analysis from the influence of chance or luck, which he argues are terms from ordinary language and indicate a lack of control. Instead he argues that the language of indeterminism ought not be construed as implying luck, and instead understand it as necessarily ruling out deterministic causation. In its stead, we ought to adopt a paradigm of probabilistic causation, in which outcomes are not inevitable.[152]

The ownership of a given action stems from its basis in the character of the moral agent. He argues that**:**

> A choice is the agent's when it is produced intentionally by efforts, deliberation and reasons that are part of this self-defining motivational system and when, in addition the agent *endorses* the new intention or purpose created by the choice so that it becomes an additional part of that motivational system and thereafter functions as a further purpose guiding *future* practical reasoning and action."[153]

In essence, each choice the agent makes and endorses becomes integrated into her causal decisional mechanism and motivational scheme. Kane refers to this in literary terms, as he states that "agents exercising free will are both authors of and characters in their own stories all at once."[154] This story is unfinished – the agents have no determined path or character arc; instead they define and refine the story and character as they are written. The fundamental criteria for agentic control in Kane's model is:

> Embodied in the idea of plural voluntary control over a set of options at a time when they have the (i) *ability* or *capacity* to (ii) *bring about* (iii) at that time (iv) *whichever* of the options they will or want, (v) for the reasons they will to do so, (vi) on purpose or intentionally rather than accidentally, by mistake or merely by chance, hence (vii) voluntarily (in accordance with their wills rather than against them), (viii) as a result of their efforts, if effort should be required, (ix) without being coerced or compelled or (x) otherwise controlled or forced to choose one way or the other by some other agent or mechanism. Agents *exercise* such control *directly* when they voluntarily and intentionally *produce* one of the

[151] Kane, p. 384-5
[152] Kane, p. 385.
[153] Kane, p. 388.
[154] Kane, p. 390.

> options (a particular self-forming choice or SFA) *then and there* (at the time in question) under these conditions.[155]

For the moment, I am willing to simply grant these criteria. There are fundamental problems therein, however, that we will return to and discuss shortly. In light of these criteria, we understand indeterminacy to be fundamentally an obstacle to be overcome by the agent, and when this occurs, the agent has succeeded in bringing one of her conflicting desires to fruition.[156] These drives and desires stem from her character and background, ensuring that the resultant choice serves to further define and develop their character.

There are two responses to Kane's proposed model: first, exactly why should this be construed as being something other than a deterministic model; and second, how much of the agent's intentionality counts towards agency? Do we have to be consciously aware of *every* motivational factor? Each of these questions will be addressed in turn.

First, at this point, it is necessary to throw in an observation: The model Kane proposes bears more than a passing resemblance to the model proposed by Paul Henri Thiry, Baron d'Holbach, which is *explicitly and inherently deterministic*. In Thiry's model:

> Man is said to *deliberate* when the action of the will is suspended; this happens when two opposite motives act alternately upon him. To deliberate is to hate and to love in succession; it is to be alternately attracted and repelled; it is to be moved sometimes by one motive, sometimes by another. Man only deliberates when he does not distinctly understand the quality of the objects from which he receives impulse, or when experience has not sufficiently apprised him of the effects, more or less remote, which his actions will produce…He deliberates in consequence; he weighs the various motives that urge his will to go out or to stay at home. He is at length determined by that motive which is most probable. This removes his indecision, which necessarily settles his will, either to remain within or to go out. This motive is always either the immediate or ultimate advantage he finds, or thinks he finds, in the action to which he is persuaded…Choice by no means proves the free agency of man: He only deliberates when he does not yet know which to choose of the many objects that move him…Action always being the effect of his will once determined, and as his will being determined by a

[155] Kane, p. 397.
[156] Kane, p. 401.

> motive which is not in his own power, it follows that he is never the master of the determination of his own peculiar will – that consequently he never acts as a free agent. It has been believed that man was a free agent because he had a will with the power of choosing, but attention has not been paid to the fact that even his will is moved by causes independent of himself…[157]

What is the difference between Kane's argument and saying that our unconscious drives and desires are in conflict until one wins out and causes us to act, as in Thiry's model? Thiry's model seems to meet the criteria for control that Kane suggests, and yet Thiry draws radically different conclusions. If the same line of reasoning can justify both deterministic and indeterministic conclusions, it seems suspect to conclude that it is therefore an argument for probabilistic causality and hence indeterminism.

Second, Kane makes repeated reference to a reflective process in choice and volition, in an effort to have the moral agent take ownership of her actions. In this dissertation, a recurrent critique of indeterministic models stressing character and motives is non-conscious causation – the process that gives rise to character and reflection upon action are essentially deterministic, in that they are influential, but not consciously willed. Hence, we have non-conscious causative factors, which implicitly seem to undercut models of volition predicated on conscious awareness and reflection.

McCrone suggests that a more accurate attempt to understand cognition should shy away from the reductionist methodologies suggested earlier in this chapter. Instead, we ought to 'look to the big picture' of the human mind in all of its contexts and see it as a 'bifold' model of socially constructed software using the resources of the underlying biological hardware.[158] The faculty of free will thus is a social idea in addition to a neurological process. Viewing free will as an interaction of biological processes and cultural input allows us to resist the "bogy of

[157] Paul Henri Thiry, "Of the System of Man's Free Agency," in *Fifty Readings in Philosophy*, ed. Donald C. Abel (New York: McGraw-Hill, 2004), 281.
[158] McCrone, "A Bifold Model of Free Will," 242.

Newtonian determinism" – the social and cultural context in which we operate demands our feelings of autonomy at the same time as it inculcates cultural mores and expectations in our sense of agency.[159] Knowing what we should be doing also necessarily implies what we should not be doing, giving us alternative courses of action, if we were to so choose them. This greater degree of socialization translates into a greater awareness of actively choosing between options.

While McCrone does suggest a unique perspective on indeterminism, it is questionable as to whether the model he proposes actually demonstrates human free will. After all, what would distinguish his model from a deterministic model in which both biology and culture are the determinants? McCrone argues that human action is, in fact, determined in this regard,[160] but he suggests that as part of this cultural determinism we foster our sense of individual autonomy. This seems to be begging the question – if a sense of autonomy is culturally determined, is it actually there (a position we will return to again in the discussion of determinism below)? After all, if part of the inculcation of cultural mores is a developing personal impression of telepathic ability, does it actually mean I can read minds? This suggestion appears dubious, and seems to argue instead that a feeling of autonomy is culturally determined, and therefore not genuine autonomy at all.

Enç argues that there is a fundamental indeterminacy in mental activity,[161] but that this indeterminacy does not itself explain the nature of the feeling that the choice is up to me.[162] He argues that we must distinguish voluntary from involuntary actions, and he suggests that we ought to consider the underlying deliberative mechanism as the basis for this distinction.[163] So long as the underlying mechanism is functioning properly (i.e., absent artificial constraints like

[159] McCrone, p. 254.
[160] McCrone, p. 254.
[161] Enç, *How We Act: Causes, Reasons, and Intentions*, 163.
[162] Enç, p. 168.
[163] Enç, p. 228.

addictions, compulsions, etc.), we have reason to call the action voluntary (so acting on bad or absent information is still a voluntary act, as the underlying decisional mechanism was functioning properly). Our sensation of having willed an action stems from this decisional mechanism: "The reasons for the original intention won over the reasons for satisfying the more immediate desires. But these frustrated desires leave a mark. It is conceivable that the mark is what gives the feel of having exerted one's will."[164] Will, in this sense, is "a power to make rational decisions in varying circumstances."[165] As such, it is possible for the will not to be free – some circumstances may prevent this decisional mechanism from operating properly, and as such, the agent is not free in the choices made. The model Enç ultimately adopts is similar to that of Locke, who discussed conditional restraints on individual agency. Enç argues that:

> What I am suggesting, instead, is a reading of Locke in which freedom is an attribute of *persons* in their capacity as agents in specific contexts. And I propose to add to Locke the proviso that an agent's freedom, in so far as she is contemplating a type of activity in a specific context, comes in degrees. If upon deliberation, I choose a course of action on the merits of the case, and act in accordance with my choice, my act is voluntary. It would be voluntary in spite of the fact that I could not have acted otherwise if I were to choose to act otherwise. But if the alternative courses of action available to me were severely restricted (say by the counterfactual contravener poised to interfere if I made the 'wrong' decision), then the degree of freedom I normally enjoy in that context would accordingly be diminished."[166]

But this necessarily begs the question – if I deliberate upon an action (conscious and non-conscious processes), then I choose how to act based upon the merits of the case (non-consciously modified both by my backstage cognition and cognitive heuristics discussed in the next chapter), then act in accordance with my choice (the ultimate effect of myriad backstage causal elements), my act is voluntary – but the question remains as to whether it is free. While Enç argues for degrees of freedom, it is not entirely clear what the scale is – voluntary and

[164] Enç, p. 229.
[165] Enç, p. 230.
[166] Enç, p. 233.

involuntary actions might not necessary be distinguishable absent a much clearer presentation of what constitutes a properly functioning decisional mechanism. As has been suggested, properly functioning decisional mechanisms do not necessarily mean fully, or even partially, conscious processes.

Libet argues that his experiments suggest some methodology by which free will may operate – a restriction on an urge to act that begins outside of conscious awareness.[167] We can freely choose to follow through on this impulse or to suppress it. However, these experiments do not address the fundamental question as to whether conscious actions are completely determined by natural laws, or whether conscious processes can proceed without them. He notes that if conscious actions are indeed determined, our personal experience of free will is illusory, and exerting one's will is a mere epiphenomenon.[168]

This is a practical concern, and research does seem to indicate that conscious processes are indeed determined by some causal laws. Thought itself tends to fall into patterns of activation, whether one adopts one of the models discussed in the reductionism section or adopts a new model. The phenomenon of being on "auto-pilot" is just as common as the phenomenon of exercising conscious volition. Further, psychological models demonstrate that how we take in and process information tends to be predictable; if cognition necessarily has an underlying substrate, as argued earlier, it would stand to reason that we develop neural activation patterns, which are in fact governed by natural laws (e.g., the all-or-nothing rule of synaptic firing, irreversible and cascading opening and closing of sodium and potassium ion channels as a causing the relay of the signal down the nodes of Ranvier, neurotransmitter release into the synaptic cleft as a result, leading to the inhibition or stimulation of the next neuron, etc.). As we

[167] Libet, "Do We Have A Free Will?" 55.
[168] Libet, p. 55.

will see later, Libet will suggest that we default to an assumption of free will to preserve our beliefs in the everyday phenomena of free will – this seems to be an unjustified claim, as mounting evidence suggests that this is in fact a willful fiction.

Eccles argues quite forcefully that free will is factual – in fact, he argues that to deny it is irrational and illogical, and one ought not to even engage in discussion with someone who claims to be merely responding to complex stimuli with complex conditioning.[169] Discussion with such a "Skinnerian", he argues, devolves into a game of "conditioning and counter-conditioning." Like Libet, Eccles suggests that if "in willing an action one does not *effectively* influence the patterns of neuronal activity in the cerebral cortex and so bring about the desired discharge of motor pyramidal cells, then free will is an illusion, however subtle the philosophical arguments."[170] Eccles is arguing for a series of individual worlds of neural phenomena in which we understand consciousness to be the product of the individual's dominant hemisphere. This dominant hemisphere gives rise to a non-physical phenomenon that can causally interact with the physical material of the brain, affecting behavior and cognition. Despite a spirited rejection of Savage's response that the model is glorified dualism, Eccles does fundamentally face the same problem as Descartes – explaining how the non-physical interacts with the physical.

Even granting that consciousness is the product of the dominant hemisphere – experiments with split-brain patients demonstrate that non-conscious physical actions can originate with the non-dominant hemisphere[171] – there is still a fundamental problem: we have no proof that this phenomena of consciousness is causal. If the non-dominant hemisphere can move the individual's arm or leg without the agent being aware of it or despite consciously

[169] John C. Eccles, "Brain and Free Will," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), 101.
[170] Eccles, "Brain and Free Will," 103.
[171] V.S. Ramachandran and Sandra Blakeslee, *Phantoms in the Brain: Probing the Mysteries of the Human Mind* (New York: Quill, 1998).

willing it to stop, it suggests that epiphenomenal volition is insufficient to counter material causation (similar to the Penfield experiments). If we can have this kind of causation without volition, why should we assume that the sense of will and volition is anything more than a secondary by-product of nonconscious processes – the very thing that Libet's experiments demonstrate? The model of consciousness emerging from the dominant hemisphere does not seem to account for apparently purposeful and willed non-conscious action – a split-brain patient will consciously pick up a pencil with the hand controlled by the dominant hemisphere, and the hand from the non-dominant hemisphere will attempt to put it back down. Appeals to a non-dominant consciousness are question-begging; if it exists, there is no reason to assume it is any more causative than the dominant consciousness.

Like Enç, Gomes notes that modern conceptions of physics have introduced a fundamental indeterminacy at the particle level, which can result in causative chains and effects at higher levels of organization.[172] He argues that while this might allow for indeterminacy in actions and events, it does not bolster the claim of free will – as has been repeatedly argued, part of the conscious experience of free will is the sense of having consciously willed something to occur. If the ultimate cause of a behavior is quantum fluctuation, then I am no more the cause of the action than I am in a deterministic system. As such, Gomes concludes that "Reasons and choice seem as different from chance as from causes. It is difficult, then, to reconcile our naturalistic view of the physical world with the idea that we ourselves, as voluntary agents, are part of this physical world."[173]

Free actions in his model must fundamentally take into account that the mind is subject to causality – a common perception of which we normally are not aware. We often do not perceive

[172] Gomes, "Volition and the Readiness Potential," 60.
[173] Gomes, p. 60.

in first-person terms (agent as agent) the causal factors that can be evident in third-person terms (agent as observer).[174] A necessary part of this perception, however, is that I do not perceive my actions to be caused by factors external to me – I still take ownership of the action and claim responsibility for bringing it about. A further necessary condition for a free action is the genuine possibility of "doing otherwise in the same circumstances.'[175] In this list of circumstances Gomes includes all of the external factors germane to the choice, potentially all of the 'somatic states' of the agent (body conditions external to the nervous system), as well as potentially all of the mental states, such as desires, beliefs and emotions. One would not, however, include the actual deciding system within these causal constraints – that would inherently lead to a contradictory claim that "even if the subject had decided to do the same thing, he could have decided to do otherwise, which is self-contradictory."[176] As such, we should understand free and voluntary acts to implicitly mean that if every other causal factor except those internal to the self were the same, the agent could choose differently. A final key element of free will is choice, not simple agentic determination. The agent must perceive a genuine choice between alternatives, and must willfully choose to pursue one of the alternatives. As such, the action must be deliberate:

> Another important concept for the characterization of free will is the concept of choice. Free actions are actions that are not only determined but *chosen* by the subject. For an action to be considered free, the subject must have chosen to do it, and this implies having chosen not to do otherwise. This means that the possibility of doing otherwise should not be just an abstract possibility, it should not be just a possibility that exists from the point of view of someone who considers the case from the outside. Rather, it should be a possibility for the subject himself or herself. The subject must be able to consider this possibility before the final decision to act. This means that the subject must be conscious of the intention to act now before acting. We conclude that only what we have called *deliberate* acts should be considered as really free. Acts that are voluntary

[174] Gomes, p. 73.
[175] Gomes, p. 74.
[176] Gomes, p. 74.

> but non-deliberate would then manifest an intermediate degree of free will. They are determined by the subject, we consider that the subject could have done otherwise in the same circumstances, they derive from a conscious experience of the situation in which the subject is, but they were not consciously chosen, in the sense that they and the possibility of doing otherwise were not consciously considered by the subject before starting their performance.[177]

This model seems to have an implicit assumption that there is *actually* something fundamentally different between experiencing choice and volition as causative factors instead of effects of non-conscious processes. The model Gomes proposes implicitly assumes a deterministic model – hence his caveat that everything could be the same except for one essential causal factor. If indeterminism were genuinely possible, we should not require such caveats in its defense. Further, as Libet's experiments demonstrate, the *experience* of volitional and deliberation are not necessarily proof that they sprang, *sui generis*, from the agent's conscious processes – the experience of volition occurs *after* the non-conscious brain has 'decided' to act.

While not explicitly aimed at Gomes' argument, a second question can be raised concerning quantum indeterminacy, as some theorists have suggested that fundamental indeterminacy leads to higher indeterminacy.[178] While physics demonstrates the essential unpredictability of atomic and subatomic particles (in our current understanding of particle physics), there is a necessary question as to whether this is necessarily applicable at higher levels of organization. As some have suggested, deterministic philosophies make no sense at the quantum level;[179] the converse question is whether quantum indeterminacy makes any sense at the cellular or social level.

Challenges to Indeterminism

[177] Gomes, "Volition and the Readiness Potential." p. 74-5
[178] Enç, *How We Act: Causes, Reasons, and Intentions*.
[179] Jim Al-Khalili, *Quantum: A Guide for the Perplexed* (London: Weidenfeld & Nicolson, 2003).

Significant challenges have been raised to indeterminist theories. Recurring themes in the challenges include the questionability of whether the agent authored the action, the role of character and values in the decision-making process, the consequences of divorcing cause from effect, the question of false causation, and questions about the construction of subjective phenomena. These challenges will be explored in the arguments of Dorato, Kane, Claxton, and Bargh and Ferguson.

Dorato's challenge to indeterminism involves two essential issues. First, it is an implicit assumption on the part of indeterminism that strict 1:1 causality is rejected in favor of probabilistic arguments – a given cause could have multiple effects, though the probabilities of each may vary.[180] Second, this probability refers not only to causes external to the agent, but to *internal* causes as well, including the agent's beliefs and values. These, Dorato argues, are influential *prior* to the decision-making process, and as such, only influence the decision *probabilistically*.[181] This leads the indeterminist into a bit of a dilemma – we can imagine a context in which the effects of a given choice are completely unrelated to the agent's background and values:

> By analogy, now consider a great number of identical replicas of the person deciding whether to lie or not in front of the jury. Recall that probabilities are ontic, so that the past of each individual is identical to any other replica up to the moment of decision, and the rest of the world is identical, too. Only in a certain percentage of the worlds we are considering does the person lie, while in the others she tells the truth according to the probability we may have. It then follows that the set of *single individual* events and circumstances preceding the decision not to lie – in which we include all the desires, the beliefs, and the moral values of the person in question, and therefore, anything that matters for her – *literally cannot fully explain the decision not to lie, given that they are compatible also with the opposite choice*."[182]

[180] Causality must be probabilistic in the indeterminist model unless one wishes to allow for a complete separation of cause from effect, which is itself problematic. In essence, the indeterminist would agree that certain events are *likely*, but not *necessary*.
[181] Dorato, "Determinism, Chance, and Freedom." p. 358-9
[182] Dorato, p. 360-1

This example does stretch plausibility – it is extremely unlikely that we will ever face a situation of having to account for such choice – but it does have a legitimate basis. Given probabilistic causality, we cannot definitively argue that the actions the individual initiates will have any connection to that individual's character, identity, morals, values, etc. By requiring probabilistic reasoning, the indeterminist opens decision-making to genuinely inauthentic choices, anathema to many conceptions of autonomy (discussed in chapter four).

Kane attacks indeterminism on the grounds of the logical consequences of divorcing cause from effect. He argues that indeterminism does not, in fact, help the cause of free will. Indeterministic systems translate essentially to chance – some events occur only because of change, and *not because I necessarily willed them*. As such, "Indeterminism in nature, if it did play a role in human affairs, would not enhance our freedom and control over events, but diminish freedom and control; and so it is considered to be irrelevant to free will at best, and an obstacle at worst."[183] It is reasonable to question whether an indeterminist understands the debate in terms of causation or in terms of some other concept (e.g., Stace and Holmstrom understand the issue in terms of degrees of control and coercion).[184] However, if one does adopt a definition of indeterminism explicitly separating cause from effect, then there appears to be no way around this criticism – we can agree that an event occurred, but we have no reason to assume that we authored or controlled it.

Claxton suggests that in light of the dominant folk psychology model of consciousness and volition, we ought to approach the question of agency and indeterminism with a skeptical view:

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[183] Kane, "Free Will, Determinism, and Indeterminism," p. 372.
[184] W.T. Stace, "The Problem of Morals (Selection)," in *Fifty Readings in Philosophy*, ed. Donald C. Abel (New York: McGraw-Hill, 2004), 312-19; Nancy Holmstrom, "Firming Up Soft Determinism (Selection)," in *Fifty Readings in Philosophy*, ed. Donald C. Abel (New York: McGraw-Hill, 2004), 319-32.

> *A fortiori*, we have to approach the phenomena of 'free will' distrustful of observations that seem immediate and unequivocal, and construals that appear self-evident. It certainly *seems* as if each of us is a center of volition, and that conscious deliberation plays a causal role in determining our plans and actions – albeit an intermittent one. And sophisticated theoretical superstructures can be built which seem to buttress, but which actually presuppose, this 'common sense'.[185]

Working within the folk psychology model, we only distinguish between sources of action that are either from 'me' or from 'not me' – actions stemming from 'me' are evidence of free will, while actions stemming from 'not me' are determined. If we find the idea of 'not me' causation repellent (but not necessarily untrue), then we then have a motive for arguing for the idea of conscious intention as the source of action.[186] This motive and assumption can give rise to models that appear to make fundamental errors – intentional states lead to conclusions that the conscious self is the cause of action.[187]

Claxton's rejection of intentional states includes a challenge of false causation – intentional states and actions may not necessarily be causally related. The intentional state and the action may both be consequences of a prior cause, and the intentional state may simply occur before the action – which seems to be exactly what Libet's experiments demonstrate. He notes:

> If A and B covary, and A usually preceded B, that doesn't necessarily mean that A causes B. it could equally be that both A and B are manifestations of a third set of processes, C, the time characteristics of which just happen, every so often, to make A pop up shortly before B. On this picture, the facts that intentions are sometimes followed by the intended action and sometimes not, that voluntary actions sometimes occur without concomitant intentions, and that intentions sometimes impede the execution of actions, invite speculation about the relationship between and nature of A, B and C. If we admit that 'C', whatever it is, comprises preconscious processes, then the loose-coupling of A and B no longer has to be construed as aberrant or anathema. Revising the sense of self to

[185] Claxton, "Whodunnit? Unpicking the 'Seems' of Free Will," p. 101.
[186] Claxton, p. 101-2
[187] See, for instance E.J. Lowe, "Self, Agency and Mental Causation," in *The Volitional Brain: Towards a Neuroscience of Free Will*, ed. Benjamin Libet, Anthony Freeman and Keith Sutherland (Exeter: Imprint Academic, 1999), 225-40, in which Lowe defines physical in *spatial* terms, not *physically-constructed* terms (i.e., thought is not 'physical' in his model). This model results ostensibly in a proof of nonphysical causation but which fundamentally involves false dilemmas.

> include such pre- or unconscious processes would then render the perceptual squint unnecessary.[188]

The extent of pre- and unconscious processes in cognition comprises the bulk of chapter two, and as such will not be addressed in detail here. It will suffice to say that indeterminist theories may be quite mistaken in arguing that intentionality preceding action is demonstrative proof of free will.

Bargh and Ferguson argue that the phenomena of will and subjective experience are misleading. Citing the Penfield experiments in neuroanatomy, they note that subjects were unable to prevent movements of their hand even when told of them in advance (Penfield would stimulate areas of the patients motor cortex to induce the movement, and the patient would try to resist). Their 'will' apparently, was insufficient to prevent external control.[189] They further note that belief in free will may be a cultural construct, rather than a universal phenomenon:

> There is also recent evidence that belief in a substantial role for free will or conscious choice in one's life varies by culture. Iyengar and Lepper reported a study comparing the beliefs of Japanese versus American students regarding how many choices they had made during the course of a given day – the American students reported themselves as having made 50% more such choices than did the Japanese students and also reported these choices as being significantly more important to them…Approximately 30% of the Americans, but none of the Japanese, reported wanting to have choices all of the time, and more than half of the American students said they could not imagine a time when they would prefer not to have a choice. Belief in the role and extent of free will as a causal factor in one's life is therefore not a universal and may be at least somewhat a function of the values (e.g., for individualism) of one's culture – the implication being that the feeling of volition is not necessarily based on its true causal status. From all of this evidence, as well as research on the 'illusion of control', one can conclude that people normally and naturally experience their own behavior as intentional and volitional even when that is not the case. It is clear from this that one's subjective experience of volition is a poor and inaccurate guide to its true causal status.[190]

[188] Claxton, "Whodunnit? Unpicking the 'Seems' of Free Will," p. 105.
[189] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," p. 940.
[190] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," p. 940.

Two questions immediately stem from these arguments. First, to what extent can we infer the falsity of free will from the Penfield experiments, and second, does a lack of *belief* in free will necessarily translate into an *actual* absence of free will? There is some reason to suspect that the objections raised by Bargh and Ferguson may not necessarily demonstrate their point.

However, at this point, it is necessary to include a necessary caveat: Bargh and Ferguson have provided a wealth of literature on the automaticity of many cognitive structures – they have demonstrated that specific heuristics are ubiquitous and causal in our everyday experience. As such, there is reason to believe that their challenges to the concept of free will are legitimate. These will be explored more fully in chapter two. For the moment, one can raise a legitimate concern that they are accurate in their assessment of free will – perhaps it is a cultural construct, an automatic heuristic we employ to account for our experience of 'willing' an action.

Determinism

Determinism as an ontological assumption does not require significant detail to explain – it is essentially a system in which probabilities are replaced by necessity; chance is an illusion, as there is only one possible effect from any given cause. Dorato defines determinism as a singular relationship in causality, "in which each event is necessarily caused by a preceding event and in which, as James put it, 'the future has no ambiguous possibilities hidden in its womb: the part that we call present is compatible with just one fixed totality.'"[191] For any given cause, there is only one corresponding event possible – there is no indeterminacy or accidental effects. Causation extends backward and forward, ostensibly *ad infinitum* or to some initial uncaused cause. As a consequence, any experience we have of choice is essentially illusory – we are simply unaware of the causes which determine how we will act.[192]

[191] Dorato, "Determinism, Chance, and Freedom," p. 341.
[192] Dorato, p. 342.

What might constitute causative elements in a deterministic model? What are the necessary assumptions? Determinism, Dowe argues, necessarily requires the contingency of the present and future upon past causes – while it is conceptually possible for effects to be contemporaneous with or precede their causes.[193] A second necessary feature is the link between cause and effect – in deterministic models, this link is fundamental and necessary, in indeterministic models, on the other hand, this link is *possible*, but not *necessary*. Dowe notes that there are several ways in which the link between cause and effect can be made – i.e., there are several ways in which we can understand deterministic causality.[194] First, it is possible that causation is strict in determined events (every event has a cause) and loose in indeterminate events (some events have no prior cause). A second possibility deals with sufficient versus insufficient causation. Determined actions are those whose prior causes are necessary and sufficient to bring about the effect, while indeterminate events have no or insufficient causation. The task of science would then become to discern whether the events in the world have sufficient causes. The third possibility is that where the distinction between determinism and indeterminism is expressed as threshold point (e.g., 70%) that may or may not be context dependent, an alternative labelled 'underdeterminism'. Underdetermined actions are those that have prior causes which collectively sum to or past the threshold point, while the causes in indeterminate actions fail to reach this threshold. Dowe argues that the first two options of deterministic thought ultimately must be rejected, leaving the third option as a workable explanation of determinism:

> Now the first option – that indeterminism is where there is no cause at all – if it allows causation by degrees, will understand this as a case of causation but not determinism. Or if it does not allow causation by degrees, it will understand this as a case neither of causation nor determinism. Either way it gets the wrong result

[193] Dowe, "What is Determinism?", p. 313.
[194] Dowe, p. 314.

> – since the case does raise the appropriate folk worry in connection to free will. The second option – the most common amongst contemporary philosophers – also gets the wrong result, since it takes the 95% case to be a case of indeterministic causation. I conclude that the third option is the appropriate way to secure the contingency of determinism. This means that to get a satisfactory causal theory of determinism, we need a notion of causation which admits causation by degree. Further, we would require the relation between deterministic and indeterministic causation to be transparent.[195]

It is this third relation of causation to determinism that is adopted in this dissertation – this underdetermination is quite compatible with a model of causal influence that is both reductive and additive. It is reductive in the sense that it is explicable in terms of causation at lower levels, much akin to a readiness potential for neural activation. It is additive in the sense that it is a concatenation of causal forces; as has been argued earlier, we can see causation occurring at multiple interactive levels of organization. Causation can be experienced at the level of intracellular communication, intercellular communication, neural pathways, overarching structures and cortices, epiphenomena, past experience, and sociocultural interaction – all of which can interact causally resulting in an action we experience as our own. When left on their own, i.e., when they are not consciously challenged, these strongly causal factors are sufficient to generate action, resulting in purely deterministic agency. When they are consciously challenged and explored, indeterminism is added to the cognitive mix, allowing the agent to exercise conscious control over their actions. This model will be explored more fully in the next chapter.

This ought not to be understood as a behaviorist argument, as it is explicitly delving into the un-/sub-/preconscious processes behind the outward manifestation of behavior. Bargh and Ferguson note that many have suggested that the rejection of behaviorist models constitutes a subsequent rejection of mechanistic accounts; this, they suggest, is fundamentally mistaken, and is only exacerbated by claims that controlled processes (those cognitive faculties that we

[195] Dowe, "What is Determinism?" p. 315.

experience as being controlled by us, like cognition and volition) are undetermined.[196] Rather, they suggest that we make a distinction between automatic processes and controlled processes only because researchers have been able to find the causal factors for one but not the other – they expect the causal factors for controlled processes to be discovered in time. They argue that it is false to suggest that automatic processes are determined and controlled processes are undetermined simply for the reason that *both* processes will ultimately be shown to be determined. I disagree with Bargh and Fergusson on this, as it seems *overly* determined. Conscious processes certainly are contingent upon deterministic elements, but when conscious challenges are raised, the agent has the ability to choose between two options – the original predisposition stemming from avolitional non-conscious elements or the one or more new options generated by challenging the assumptions of the original predisposition. Following this challenge, we have removed the emphasis of our cognition from the backstage processes to the foreground, and are much more likely to spot errors in cognition when we are consciously searching for them, or have others assisting us in discerning them. This would seem to undercut claims of pure determinism in cognition and volition.

Determinism ought not be understood as applying solely to behavior of which the agent is unaware or unwilled – instead, they argue, we ought to concern ourselves with understanding complex behaviors as well:

> Although some may conflate the notion of automaticity with determinism, it is our position that a deterministic interpretation of human behavior should not be confined to behavior that proceeds without consciously aware choice and guidance. Whereas automatic behavior can be easily understood as exemplifying a deterministic account of behavior (it is commonly defined as unwilled, unintentional, aware), the existence of a role played by consciousness or controlled processes in a phenomenon does not preclude a deterministic account of it. After all, the very point of research and theory on judgment and decision

[196] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," p. 938.

> making is the discovery of the causal mechanisms (i.e., determinants) of conscious choice and reasoning processes themselves.[197]

As has been noted, social and cognitive psychologists have found models that can account for upwards of 60% of complex behavior like health decision making, suggesting that the field is moving towards the fruition of Bargh and Ferguson's suggestion. Further, Bargh and Ferguson's own research demonstrates that our perception of volition may not even necessarily require us actually possessing volitional capacity.[198]

Challenges to Determinism

Many challenges have been raised to deterministic philosophies. These range from questions of empirical validation and underlying physical theory to questions of whether it is essentially meaningful to our everyday experience of the world (i.e., does it pass the "So What?" test). These questions are necessary and relevant – after all, why assume that the universe is inherently deterministic? What difference will it make to me, not as a moral agent, but as an everyday person who buys food, goes to movies, works, and attempts to have a social life? Even if behavior ends up being deterministic, why should it matter? I still have the subjective experience of being in control. In the course of addressing these issues, we will explore challenges raised by Dupre, Libet, and Kane.

Dupre argues that determinism fundamentally lacks support for two reasons: first, it has not been empirically validated; and second, the most successful scientific theories propose probabilistic instead of deterministic models.[199] He further argues that to date – in this case, 1993 – there are many fields of scientific study that are essentially probabilistic, and reject pure deterministic claims, e.g. "population and behavioral ecology, economics, meteorology,

[197] Bargh and Ferguson, p. 938.
[198] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," p. 940.
[199] John Dupre, *The Disorder of Things* (Cambridge: Harvard University Press, 1993), p. 184.

evolutionary biology, geology, and almost all of the diverse aspects of human psychology."[200] He notes that it is possible that deterministic models may eventually be found, but the available evidence does not suggest that it should be expected. Dupre ties his rejection of determinism to his prior rejection of reductionism. Specifically, he doubts the ability for science to account for complex phenomena in terms of strict causation at the structural level:

> To provide an adequate grounding for a deterministic universe we need to be sure that *every* causal connection is underwritten by a completely sufficient condition. That this can be done in terms of the concepts at the structural level in question is incredible, so that the retreat to reductionistic accounts of determinism is easy to understand. But reductionism is false. So, therefore, is determinism.[201]

This is problematic, however. As was indicated in the first half of this chapter, reductionistic models are not beholden to an explanation solely in terms of microproperties or cellular phenomena – models can be reductionistic simply by noting that cognition does not occur in entities absent an underlying physical structure, and that changes in that structure at a variety of levels can produce changes at higher levels of organization. Second, it is not clear that psychology should be considered in a list of 'non-deterministic' or 'probabilistic' sciences. Social psychology has been able to offer increasingly accurate predictive models of human behavior. While it is true that as of this writing there are no models that account for 100% of human behavior, the models that are available have accounted for significant portions thereof (e.g., some models report being able to account for 60% of health decision behavior), and are constantly being refined and modified. As this percentage climbs higher, the argument for probability and indeterminism dwindles. This ought not be construed as a complete rejection of indeterminist schools of thought, but rather should be understood as strengthening the basis for believing that human action is more deterministic than is frequently admitted. As I argued

[200] Dupre, *The Disorder of Things*, p. 186.
[201] Dupre, p. 192.

earlier – I *do not* rule out indeterministic elements of volition and choice; however, I *do* argue that unless the cognitive processing we adopt is challenged, the likelihood of indeterministic, conscious mediation occurring is unlikely.

Libet takes a different line of approach regarding natural laws and determinism than Dupre. While Dupre suggests that probabilistic theories have had more success than deterministic theories, Libet embraces determinism in the natural sciences, but suggests that there is a mistake in kind to make the leap from natural laws to subjective experiences. He notes that:

> Determinism has on the whole, worked well for the physical observable world. That has led many scientists and philosophers to regard any deviation from determinism as absurd and witless, and unworthy of consideration. But there has been no evidence, or even a proposed experimental test design, that definitively or convincingly demonstrates the validity of natural law determinism as the mediator or instrument of free will. There is an unexplained gap between the category of physical phenomena and the category of subjective phenomena. As far back as Leibniz it was pointed out that if one looked into the brain with a full knowledge of its physical makeup and nerve cell activities, one would see nothing that describes subjective experience.[202]

This is a quite compelling concern, and the danger of making a category mistake is legitimate. The question for Libet, however, is whether a rejection of determinism in the physical sense necessarily translates into rejection of philosophical determinism (e.g., ontological questions of causality). Further, must we automatically reject the idea that subjective experience itself can be causative, and causative in a manner that is not conscious? Must I be consciously aware of all of my past memories and experiences that are germane to the present situation for them to be causative? How can we reconcile this with cognitive and social psychology research on priming effects and conditioning? How can we reconcile this with data from biological psychology suggesting environmental influences affect biochemistry, which then produce distinct thought

[202] Libet, "Do We Have A Free Will?" p. 55.

patterns and facilitate specific behavioral responses (e.g., situations unconsciously perceived as hostile which activate the sympathetic nervous system, making a fight or flight reflex easier to initiate)? While Libet is correct to note the dangers of misattributing deterministic causality from one field of study into another, there seems to be a significant body of evidence arguing for philosophical and psychological determinism (or underdeterminism).

Kane suggests that even if determinism were true, we would still note a difference between coercive elements and non-coercive elements, and would prefer to be free from the former (e.g., threats of force, addictions, etc.). As such, "esoteric questions about whether determinism is true or not – in the physical or psychological sciences – are considered to be irrelevant to the freedoms we really care about in everyday life."[203] The only freedom that fundamentally matters in these instances are those that we would still feel would have undue influence on our moral agency. This analysis might be true from the perspective of everyday living, but I would suggest that this is not the case in medical decision-making. The choice to forgo medical treatment, for instance, is potentially the last meaningful choice a person might make. Would we as clinicians be acting responsibly if we did not make sure that this choice was as free from unconscious compulsion or coercion as possible? This topic will be addressed in chapter four in discussions of autonomy.

Libertarianism, Compatibilism, and Challenges to Both

There are two models that can be discussed jointly – compatibilism and libertarianism. Dorato notes that compatibilists argue for determined action insofar as we are physical objects and must obey the same rules as other natural events when they are caused. However, we also fundamentally possess the ability to do what we want to do.[204] In essence, we can be the authors

[203] Kane, "Free Will, Determinism, and Indeterminism," p. 372.
[204] Dorato, "Determinism, Chance, and Freedom," p. 343.

and originators of action, but we are necessarily constrained by the same causal laws as other natural events and objects. He sets up the distinction between compatibilists and libertarians by noting that they are defending very different conceptions of freedom:

> It is very important to stress that there are *at least three different intuitive notions of freedom discussed in the literature*, and deciding which among these really captures the essential character of 'what we mean or should mean by freedom' (what we could term freedom with a capital f) is very difficult if not impossible, considering the vagueness of our conceptual and linguistic intuitions. The three notions in question are (1) freedom meant as an absence of obstacles to the realization of our desire; (2) freedom meant as a power or capacity to bring about a new causal chain; and (3) freedom meant as the power to doing otherwise in the very same circumstances. The philosophical debate between hard determinists and libertarians on one side and compatibilists on the other, can, therefore, be explained with the following hypothesis. While the latter claim that freedom essentially coincides with the unconstrained power to do what we want – let us call it, with a somewhat inelegant but useful notation, $freedom_1$ – and that such a notion is *sufficient* to guarantee whatever is important to us from a moral point of view, the former deny this very thesis. Libertarians, in particular, defend the idea that it is only the possibility of originating a new causal chain in an unconditioned way ($freedom_2$), or the power to do otherwise in the same circumstance ($freedom_3$), that would guarantee our *moral responsibility*. Consequently, libertarians are bound to believe that human beings are effectively and actually endowed with one or both of these capacities, a belief attacked by compatibilists.[205]

The question remains, however, in how much control we can genuinely possess – is it honest to say that we can originate new causal chains? Are $freedom_2$ or $freedom_3$ actually possible? For the moment, let us confine the discussion to the sense of $freedom_2$ (the libertarian model), and see whether it holds up to critique; $freedom_3$ will be discussed below.

The fundamental assumption of the libertarian model is that we can originate new causal chains – we are the authors of action. There is an intuitive truth to this claim – by virtue of the fact that I am sitting in a café writing this, I ensure certain causal chains are possible and others are impossible by the simple fact that I cannot be in two places at once. However, in light of my subjective consciousness, I cannot say definitively that there are not other germane elements to

[205] Dorato, p. 347.

any future chain of causation. While I may influence what happens, I cannot be aware of all of the elements that have gone into *my* choice to be one place and not another. I am aware of many important causal factors – the desire to finish this chapter, the desire to stretch my legs, cabin fever, etc. – but I cannot be sure that this list is anywhere near complete, or even indicative of a majority of my underlying causal conditions. My being in this café may have been influenced by the amount of gas in my car, by the availability of distractions in other milieus, by its proximity to a movie theater if I choose to reward myself for a job well done, etc. There are other apparently 'accidental' elements in the overall causal chain (i.e., elements that do not *appear* to be initially related to any subsequent decisions I may make) that are in fact germane and necessary antecedents to the causal chain that 'I' originate. In short, while the causal chain may be dependent upon me for origination in some sense, it is unclear whether I am necessary or sufficient for this chain to come about, and it is unclear whether this sense of autonomy and 'self-direction' is meaningful. I may simply be acting as a result of prior causes in bringing about other ends, not determined by me. Freedom$_2$ – the libertarian model – seems to be excessively optimistic about the agent's *actual* role in causation.

Challenges to freedom$_3$ are found in Searle's critique of the compatibilist argument. Ignoring questions of psychological determinism, the compatibilist must address the question of whether a human agent could genuinely have acted differently if all of the contextual variables were the same. The compatibilist school of thought considers internal and external variables in causation – that is, it argues that outcomes are determined partially by factors external to the agent and determined partially by internal variables (psychological state, etc.). The challenge thus becomes whether an agent could act differently if the same causal inputs were given – if the agent could genuinely act otherwise, free will is not an illusory concept, if the agent could not,

then free will is illusory, despite the compatibilists supposed arguments to the contrary.[206] Searle argues that there is no reason consistent with compatibilist philosophy that would allow the agent to have done anything other than what was done before – in essence, because of the causative model suggested, compatibilists essentially argue for deterministic causation, the antithesis of free will.

As has been noted previously, there are some concerns about the dismissive stance taken by Searle to psychological determinism – these causal factors are significant and can be very determinative of behavior. However, he is correct to note that there are concerns about the ability to do otherwise in a compatibilist system. If the agent could not genuinely have chosen otherwise, then there is no reason to call the compatibilist argument supportive of free will; as Searle suggests, it becomes a shell of the concept – an intrinsically hollow idea. A very similar objection is raised by Savage, who notes that we can directly manipulate internal causes:

> How does the compatibilist distinguish between free and unfree actions where both are internally caused? The actions of a normal person are caused by neuronal discharges in his cerebral cortex; but so are the actions of an epileptic during a seizure. What justifies our calling the one type of action free, and the other type unfree? The compatibilists have never answered this objection. And their failure to do so is the most important argument in favor of indeterminism. But the indeterminist view is no less objectionable. In contrast to compatibilism, it provides a clear definition of a free action; namely, a free action is one caused by uncaused volition. But it forces us to hold that if any action is free, then some events are uncaused. And it is an article of scientific faith that every event has a cause. To believe otherwise is to open the door to superstition and magic.[207]

Some philosophers have argued that causation ought not to be identified as a necessary condition for free will,[208] but rather that free will be viewed in terms of coercion. So long as an action is not coerced, either from external sources (e.g., threats of physical violence or economic

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[206] Searle, *Minds, Brains, and Science*, p. 89.
[207] C. Wade Savage, "An Old Ghost in a New Body," in *Consciousness and the Brain: A Scientific and Philosophical Inquiry*, ed. Gordon G. Globus, Grover Maxwell and Irwin Savodnik (New York: Plenum Press, 1976), p. 145.
[208] Stace, "The Problem of Morals (Selection)."

gain/loss) or from internal sources (e.g., genetic predispositions or education), then the action is free (i.e., it stems from the psychological state of the individual). But this is problematic – psychological states can be deterministic. Identical situations and information can be perceived quite differently dependent on the person's psychological state, and this interpretive bias is not necessarily self-evident. Others have suggested that what is important in deciding between free will and determinism is the degree of control that the moral agent exercises over the situation – the more control the individual has, the greater their free will.[209] Subjective control consists of examining orders of desire (i.e., first order: "I want this"; second order: "I want to want this"; third order: "I want to want to want this"; etc.). Some concern can be raised concerning these definitions as well – how much control is enough? Are there any orders of desire that cannot be manipulated, leading to a loss of control?

In light of these concerns, it should be evident that there are significant concerns about both libertarianism and compatibilism. If there are fundamental questions about how much control or independent agency we genuinely possess, then there are also legitimate questions about the acceptability of models arguing for independent agentic control (libertarianism) or internally causative conditions (compatibilism).

Alternative Theories in the Determinism Debate

There are two alternative theories that will be considered: dissolutionism and hermeneutics. Both seek to do away with the determinism debate by changing the frame of reference. Dissolutionism changes the frame of reference used in considering the action, while the hermeneutic argument suggests changing the nature of how we view the individual. Each of these will be explored briefly.

Dissolutionism

[209] Holmstrom, "Firming Up Soft Determinism (Selection)."

Dissolutionism argues that while deterministic causation necessarily is applicable and unavoidable in the larger natural world, but there are other issues to be considered at the level of individual autonomy and volition. Dorato suggests that the dissolutionist argues that a distinction must be made between the *evaluative* attitude towards the agent and the *descriptive* attitude towards the action performed. By shifting the type of language used, it is possible to remove talk of causation from the discussion:

> Once the talk of causes is eliminated, the threat of determinism to our freedom is thereby eliminated, too, since it would be meaningless to claim that our actions are determined by antecedent causes…To put it simply, the dissolution of the problem of the relationship between determinism and free will calls into play a linguistic analysis of the respective conceptual domains, from which the conclusion emerges that they are *incommensurable*.[210]

The problem is thus dissolved – we no longer have to speak of determinism because the concept makes no sense in the evaluative framework being used. Dorato offers a quick refutation of this linguistic approach – discussion of causes of actions is not meaningless. Rather, it is a necessary foundational element in discussions of the psychological development of the moral agent whom we are evaluating, and hence is germane to the discussion at hand.[211] A second problem alluded to in Dorato's argument is the extent to which this is a linguistic dodge. It is troublesome that language would be seen as a barrier to a discussion of a fundamental problem, and this has more than a passing resemblance to the linguistic modeling of *1984*, wherein agents attempt to refine and sculpt the language so as to prevent meaningful evaluation of Big Brother. After all, if we can't express it in the language we have, then by definition it cannot be a problem. The fallacious nature of this argumentation is evident – the problem *does*, in fact, exist; the fault lies with the language adopted.

Hermeneutics

[210] Dorato, "Determinism, Chance, and Freedom.", p. 341-2
[211] Dorato, p. 345.

The second alternative model stems from hermeneutic phenomenology – it seeks to understand the world not in the terms science has dictated, but rather in an undistorted vision consistent with everyday, pretheoretical thinking. Guignon suggests that returning to a phenomenological perspective will allow us to better appreciate the fundamental experience of free will, and thereby transform the entire debate.[212] He suggests that a full understanding of free will necessarily implies two things: ultimate responsibility for one's actions, in the sense that the action originates with the agent, as well as the presence of genuine alternatives to the action chosen.[213] In short, an action lacking either in ultimate responsibility or a lack of alternatives implies a lack of free will. If one analyzes our everyday experience, he continues, one realizes how artificial and implausible the determinism dilemma seems to be. Guignon argues that the worldview that leads to the dilemma of determinism stemmed from revolutions in scientific thought:

> The new Galilean and Newtonian conception of science brought with it a set of ontological assumptions that have dominated our thought ever since. Reality is no longer regarded as a meaningful cosmic order; rather, it is seen as a 'universe', that is, as a vast aggregate of causally interacting material substances in a space-time coordinate system. The result is an objectifying ontology that treats as real only the objectively specifiable properties of things, where 'objectively specifiable' means 'in a way not subject to interpretive dispute,' ideally, those properties that are quantifiable. Humans occupy an oddly ambiguous position in this conception of the natural order. On the one hand, they are regarded as organisms in a natural environment, products of evolutionary forces, constantly affected by causal factors in the surrounding world. On the other hand, they are seen as knowing subjects who are capable of grasping the external world and transforming it for their own purposes. Given either account, a human is regarded as an object that is distinct from, yet in constant causal interchange with, the array of non-human and human objects surrounding it in the world.[214]

[212] Charles Guignon, "Ontological Presuppositions of the Determinism-Free Will Debate," in *Between Chance and Choice: Interdisciplinary Perspectives on Determinism*, ed. Harald Atmanspacher and Robert Bishop (Charlottesville: Imprint Academic, 2002), 322.
[213] Guignon, "Ontological Presuppositions of the Determinism-Free Will Debate," p. 323.
[214] Guignon, p. 324-5

As a consequence of this scientific viewpoint, we assume that all elements of nature, including ourselves, must be understood as adhering to the same causal laws, and that the same rules of explanation must necessarily apply to all events. Phenomenologists, Guignon argues, suggest that we not be so hasty in assuming that the rules which apply to some aspects of our lives (physical interactions, in which such assumptions can prove to be quite useful) apply equally to others (cognition and volition). The aim of phenomenology, he argues, is an effort to dissolve the problem by "challenging the very framework in terms of which the problem is formulated."[215] He notes that our everyday experiences do not have a static character – they shift and are redefined as the situation changes. Our initial fear and apprehension may dissolve into amusement and relaxation, with corresponding shifts in attention and attitudes.[216]

How, then, should we see the world and our experience in it? Guignon argues that we should understand it not in the mechanistic terms of the scientific view, but as a complex interweaving that is holistic and dynamic. In fact, phenomenology would argue that the scientific viewpoint prevalent in modernity is itself derived from and contingent upon a far richer tapestry of experience than can be expressed in the language of science.[217] The phenomenological viewpoint argues that we understand actions in terms of the wider context in which they are embedded: the unfolding life-story of the agent. This shift in viewpoint and

[215] Guignon, p. 325.
[216] This is the point at which the first fundamental challenge can be raised – the context-dependent reactions are explicable, and testable, in terms of neurochemistry and sympathetic activation. We are fearful, and therefore we are primed for fight or flight; when we see the threat abate, we relax, and are no longer primed for such behavior. This doesn't require a linguistic shift or phenomenological analysis. Such approaches seem to make the issue unnecessarily complex as simpler, empirical explanations exist. Ironically, Guignon expects this response, and suggests that science is simply one world-view among many. I would respond that while this is true, it also has the added benefit of being repeatable, objectively accessible, and dynamic – but in the Kuhnian sense of the term, rather than the Heideggerian (i.e., adopting and refining a paradigm that answers more questions than it generates and offers a better explanation than the current paradigm). I would suggest that Guignon's assessment of an irreducibly complex and dynamic nature of everyday events is an attempt to render the transparent opaque.
[217] Guignon, p. 328.

holistic understanding cannot be expressed in terms of causal connections.[218] The difficulty, and the challenge I would raise to Guignon's argument, is whether the process of engaging in a life-story is conscious, subconscious, unconscious, or a combination thereof. Any sub- or unconscious elements would seemingly take away 'control' of an action from the agent and relegate it to something outside of conscious control, and hence, opens the door to non-agentic causality. This seems to lead to determinism or underdeterminism. Further, we are not perceptually aware of every germane element in the decision-making process, but priming effects and other studies in cognitive psychology have demonstrated that they do change how we behave. If Guignon requires conscious interaction for his phenomenological model, his argument gets derailed by studies of non-conscious elements of cognition.

Guignon refers to the Heideggerian concept of *Dasein* and its requisite 'facticity' by which is meant the "meaningful prior commitments and involvements in terms of which an agent grasps what is at stake in a situation."[219] This is an open-ended concept, constantly informed, changed, and redefined as events unfold. In essence, what happens to us in the past takes on new meanings in light of present experiences. While this certainly has intuitive appeal and is both easy to account for in and is commensurate with our everyday cognition, again, there is the problem of non-conscious cognition. On Guignon's analysis of Heidegger, the agent constantly engages and redefines the influential elements of his life-story. This cannot be done at a non-conscious level; meanings change upon reflection, but we cannot reflect upon all causative elements at once, nor can we even know every specific element of our life-story that is directly or indirectly germane. This model seems to be impossible to reconcile with what we know of human cognition. When Guignon insists that seeing our actions as part of an overarching life

[218] Guignon, p. 329.
[219] Guignon, p. 331.

story makes explanation in terms of causative neural phenomena less plausible,[220] there is an immediate question begged. As was noted earlier in this chapter, we cannot have a mental process without a corresponding physical process; further, understanding our 'life-story' requires reflection or intuition – both of which require conscious/subconscious/unconscious *causal* processes. Our memory is realized in a physical structure, which can break down or be physically influenced, altering our perception of self and others. The question then becomes how a phenomenological model can account for this level of reductive interaction.

Guignon argues that Heidegger's conception of agency makes determinism untenable for two reasons:

> First, humans have the ability to reflect on what has come before, redefining the past by endowing it with a different meaning. Since there are no facts about the past independent of these meanings, there is no way to specify the causal antecedent of an action in a way that satisfies the requirement of generality of causal statements. And, second, humans are beings who can envision a range of possibilities as defined by the cultural context in which they act, and so always make choices against a backdrop of alternative ways of acting. This 'standing out into a range of possibilities' is not something that can be grasped by physicalist causal statements.[221]

The difficulty with this suggestion is two-fold. The first objection again concerns the assumption of a fully conscious process. The second concern is that there is an unreasonable assumption being made – Guignon suggests humans can envision "a range of possibilities as defined by the cultural context in which they act"; but this seems counterintuitive. Can I simply choose to see the world as another human being in an identical cultural context, and then simply ignore variables like race, gender, intelligence, education, etc.? Can I simply imagine how a neurosurgeon would address a given case, not having had the requisite training? As of this writing, I am a young man; is it within my power to simply choose to see the world or choice

[220] Guignon, p. 332.
[221] Guignon, p. 333.

from the perspective of a middle-aged menopausal housewife, and experience I by definition cannot have? There is the possibility that I am interpreting this argument too literally; perhaps Guignon is not arguing for such a radical perspective shift. Perhaps, instead, he is suggesting that given my cultural context, I can imagine a variety of different possibilities or alternatives. This, too, seems to be a rather contingent function, dependent as it is upon background, education, neurological structure, intelligence, race, gender, etc. Each of these can contribute to the possibilities I see available, as well as how I approach the problem. While Guignon may see this in terms of an unfolding life-world, it is equally plausible and less complicated to view the situation as a culmination of underdetermining causal factors.

The Resulting Philosophical Model and the Consequences for Cognition

In concluding this chapter, we return to Libet, who suggests that we ought to adopt a model of cognition that does not describe free will as illusory. He appeals to the ubiquitous phenomenon of free and independent choice as *prima facie* evidence of free will. These intuitive feelings ought not be dismissed, and should be represented in any model we ultimately choose to adopt.[222] He concludes that a model of free-will is:

> [A]t least as good, if not a better, scientific option than is its denial by determinist theory. Given the speculative nature of both determinist and non-determinist theories, why not adopt the view that we do have free will (until some real contradictory evidence may appear, if it ever does). Such a view would at least allow us to proceed in a way that accepts and accommodates our own deep feeling that we do have free will. We would not need to view ourselves as machines that act in a manner completely controlled by the known physical laws.[223]

As such, by adopting this model, we can account for both the scientific data and the perceptions from folk psychology, the best of both worlds.

[222] Libet, "Do We Have A Free Will?" p. 56.
[223] Libet, p. 56-7

This does not, however, seem to be supported by the available evidence. While one can see why Libet's suggestion is attractive – we would be able to continue to believe what we believe – there do seem to be some significant cracks in the foundation of non-deterministic philosophies. Libet's own experiments remove the basis of volition from conscious control, and whether we see the results in terms of determinism or "free won'ts" there is reason to believe that at a deeper cognitive level we will find genuine causal determinants. In the next chapter, we will explore the common cognitive structures germane to decision-making from cognitive and social psychology. These structures allow for response prediction, bias in information processing, and patterns of thought and evaluation that occur without our conscious control. On the whole, the evidence supports a model in which we are perceptually aware of only the tip of the cognitive and volitional iceberg. What emerges is not a model of pure determinism, but a model in which backstage elements exert significant causal force – an underdeterministic model. We are motivated, strongly, by elements of which we might not be aware, and only through exploration of the thought processes involved can we become aware of these motivation factors. It might not be possible to ever be fully aware of such causative elements, but one ought, as a bare minimum, attempt to unearth and identify those which may distort the individuals 'normal' thought pattern – i.e., those cognitive patterns which may result in inauthentic decision-making. In the context of forgoing medical treatment, a decision that can and frequently does have lethal consequences, it seems negligent not to ensure that the decision made is genuinely the patient's, and not merely that of a particular cognitive distortion (a theme we will return to in chapter four). Patient autonomy, if it is to be meaningful, must necessarily understand what we can and what we cannot control.

CHAPTER 2 – "RATIONALITY" ISN'T SO RATIONAL – AUTOMATICITY, BACKSTAGE COGNITION, AND COGNITIVE HEURISTICS IN "RATIONAL" THOUGHT

The focus of the dissertation now turns to cognition and influences upon it at various stages. Cognition is not a single-stage process, as noted in the last chapter – there are many levels of organization in the brain, and they interact with each other in many ways which are open to influence. Conscious thought – the result of these myriad physical and social interactions, is also a construct; a concatenation of many different types of cognition, operating in conscious and backstage capacities. The focus of this chapter will be on the backstage elements of cognition – conscious choice models (i.e., autonomy models) will be addressed in chapter four. Backstage cognition involves a variety of related concepts, e.g., reflex thought patterns with affective and behavior components, generation of novel meanings for situations and objects from the mental assembly of other situations and objects, distinctions between algorithmic and heuristic thought, etc. A full treatment of cognition is well beyond the purview of this work – what follows is an overview of a much larger body of literature, and it is certain that some elements and arguments have been omitted. A complete accounting of cognition from a psychological perspective would occupy several volumes, and complementing it with philosophical analysis would require several more. As a consequence, the treatments given to certain concepts are by necessity quite brief, but I believe that the most salient elements are covered.

A related concept, a meta-question perhaps, that results from the current discussion concerns the definition of 'rational thought'. That 'rational thought' exists is generally accepted as a given, but, as should be evident at the end of this chapter, what it necessarily is isn't clear. Much like the problem facing Socrates' interlocutors, defining 'rational' becomes exceptionally difficult. How do we understand 'rational thought'? Do we consider thought processes that

result in cognitive errors as rational? What if the error does not reflect a processing problem, but rather a perceptual one? If the same cognitive process can lead us to accurate decision-making in one context but inaccurate in another, is the process 'rational'? A recurring theme in the literature notes that there are distinct qualities of cognition that are purely individual interpretation, but are generally perceived as 'rational'. We draw on the information available to us, consciously and unconsciously, but tend to do so in a self-serving fashion:

> A general thread that runs through much of the decision-making and reasoning research is that we are often overly influenced by the general world knowledge that is stored in our memories. The influence of stored information is quite pervasive; it affects how we perform in the classic forms of reasoning as well as in less well-defined judgment and decision-making situations. A second thread is just as pervasive, and just as important in decision making; far more than is logical, we tend to search for evidence that confirms our decisions, beliefs, and hypotheses, and as such are considerably less skeptical that we ought to be.[1]

Is this process rational? As we will see in discussion of cognitive heuristics, our typical mix of algorithmic and heuristic cognition has been evolutionarily advantageous (after all, we *are* still around to discuss the matter), but can also be a source of systematic error. The question remains, "What does 'rational' even mean in light of heuristic thinking processes?" *By definition, they can be inaccurate*.

The overall conclusion of this chapter will parallel that of the previous chapter – there are elements of our cognition that are strongly deterministic, but they can be overridden by indeterministic conscious elements. The challenge, however, will be in recognizing the automatic and backstage elements of our cognition – errors cannot be addressed or corrected if we are not aware of them. The discussion of automaticity and backstage cognition will cover reflex processes that can result in automatic cognitive, affective, and behavioral responses. In the process of this, we will explore conceptual blending and mental spaces as means of

[1] Mark H. Ashcraft, *Human Memory and Cognition* (New York: HarperCollins College Publishers, 1994), 520.

information processing at a nonconscious level – complex processes can occur outside of our perceptual awareness. We will then discuss heuristic thought processes – mental 'rules of thumb' regulating cognition at a nonconscious level. We will explore commonly occurring heuristics, challenges to them, as well as complementary and supplementary cognitive models. We will close with a discussion of affect – emotional responses to and valences of situations and events that can affect cognition and behavior. Our discussion of affect will draw this chapter to a close and segue to our discussion of the family of depressive disorders and their rate of comorbidity in five common medical illnesses. All of this will demonstrate the potential complexity of the cognitive processes of the patient contemplating forgoing medical treatment – faced with a difficult decision, our patients can be influenced by a variety of causal determinants for action of which they may not be aware. In light of the severity of the choice they face, we would be remiss were we not to ensure that the decision-making process to forgo medical treatment is as unaffected by sources of error as possible. This may be a chance our patients can make only once – it should not be made lightly, nor should it be made for the wrong reasons.

BACKSTAGE COGNITION, MENTAL SPACES, AND AUTOMATICITY

As the last chapter indicated, there are a variety of deterministic elements that necessarily must be accounted for in cognition. Much research has indicated that contrary to folk models of psychology, we are both perceptually aware of only a fraction of our brain's activity and naïve in our assumptions that the full extent of cognition in decision-making is accounted for by those few conscious elements. Rather, a more accurate presentation of cognition is a quite apt culinary comparison:

> In an analogous way [to a bubbling pot of soup], as information is activated in memory (by whatever influences produce activation, including spreading activation from other active nodes, residual activation from primes that directly activated the information earlier), that information drifts higher in the soup pot of

> the mind, like a noodle or a vegetable. Whatever bits are at the top of the soup correspond to the bits of information in the person's current conscious experience. The metaphor suggests at least one further implication. It derives from the fact that the pieces in the heating soup are all continuously active to some degree, even if they are nowhere near the top of the pot. They are still absorbing activation, still bumping against each other; parts underneath are still supporting the parts of the soup that are at the surface. The analogy suggests that the parts of the mind that are out of awareness similarly remain engaged in work, spreading activation amongst themselves, and in some cases serving to support the edifice that's made it to consciousness at the top of the pot. In such a model, many different areas of partial activation compete continuously for access to consciousness, but of necessity only some small fragment of these competing elements can be in consciousness at any given moment.[2]

As such, we are aware of only a fraction of our cognitive activity, and these various layers interact and inform each other. This chapter will concern itself with both processes, conscious and backstage, and how influences can creep in at a variety of levels. Our first concern, however, is not with the conscious elements of cognition – conscious phenomena are predicated on deeper phenomena. We cannot have surface cognitive phenomena without deeper structures, much as we cannot build a castle before constructing its foundation. All of the myriad sense data we take in initiate complex activation pathways, associating current stimuli with previous experiences, affective data, and other valence structures. These deeper cognitive phenomena are not simplistic processes – they are layered, quite complex, exceptionally fast, and quite independent of our volition:

> We've encountered two important lessons of cognitive psychology already. First, mental processes can occur with hardly any conscious awareness at all. This is especially (or maybe only) true of processes that have received a great deal of practice, as in reading skills. Second, even though these processes can operate very quickly, they are nonetheless quite complex, involving difficult motor, perceptual, and mental acts. Their complexity makes it even more amazing how efficient, rapid, and seemingly automatic they are.[3]

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[2] Charles S. Carver, "Associations to Automaticity," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 97.
[3] Ashcraft, *Human Memory and Cognition*, 7.

Their automaticity belies their complexity – just as complex physical responses can be initiated without volition, so too we should recognize that our cognitive processes can be induced to action. An environmental trigger can give rise to the activation of many complex systems – a particular memento can trigger complex memory and affective components with corresponding behavioral components.[4] For instance, I may pass a photograph of my grandfather, which triggers a series of memories (living with my grandparents, visits, holidays, advice given to me, etc.), eliciting specific affective responses (sorrow at his passing and resolution to fulfill promises made to him), and culminating in behavioral changes (renewed vigor in completing this chapter). None of these responses were necessarily *chosen* by me – they are all direct results of the environmental stimulus; further, this same stimulus can affect me well after I actually encounter it – my *memory* of the stimulus can provoke identical psychological and behavioral responses.

What is more, these backstage processes are also able to introduce errors into cognition – the way we perceive the world is dependent upon a variety of factors, some within our control, some well outside control. We will see that a requisite part of accurate cognition is appreciating and understanding when we are making choices based upon the indeterministic elements within our control and the deterministic elements lying outside our volition or awareness.

None of this is meant to deny that consciousness has no causal role in choice. Though authors will disagree about the deterministic versus indeterministic nature of consciousness, they do agree that consciousness acts as a mediator of backstage and automatic elements – after all, there has to be some manner of restoring systematic functioning to and making sense of the myriad processes active beneath our conscious awareness. Bargh suggests that by shifting away

[4] Eliot R. Smith, "Preconscious Automaticity in a Modular Connectionist System," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 199.

from the folk model of consciousness being the sole causal determinant of action we may actually understand its purpose more clearly:

> In removing consciousness from its privileged place at the mediational center of everything, by moving from a serial stage to a parallel process metatheory, one is not claiming that there is no role or function for conscious processing…Consciousness still exists as we move from a serial to a parallel model of mind. In fact, by getting rid of its overstated position in the middle of serial models, we may end with a clearer sense of its role and purpose.[5]

We will explore the meanings of parallel processing models of the mind shortly in our discussion of automaticity. For the moment, let it suffice to say that consciousness can help us to make sense of the backstage, reflex processes that characterize human cognition. As was stated above, however, our present concern is not with our conscious experience of choice and autonomy – we will return to this in greater detail in later chapters. Let us return our attention to the processes of which we are unaware, for as Turner notes, "Reason and choice depend upon 'backstage cognition.'"[6] The discussion of backstage cognition necessarily concerns two key concepts: automaticity and conceptual blending. Both will be addressed in turn.

Automaticity

Automaticity is a significant element of cognition – a variety of processes simply occur without volitional cueing.[7] Bargh understands automatic cognitive processes to occur "reflexively whenever certain triggering conditions are in place; when those conditions are present, the process runs autonomously, independently of conscious guidance."[8] This can refer

[5] John A. Bargh, "The Automaticity of Everyday Life," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 52.
[6] Mark Turner, "Backstage Cognition in Reason and Choice," in *Elements of Reason: Cognition, Choice, and the Bounds of Rationality*, ed. Arthur Lupia, Mathew D. McCubbins and Samuel L. Popkin (New York: Cambridge University Press, 2000), 271.
[7] The simplest means of demonstrating this is by asking the question "What is the first thing you think of when I say the words 'white bear'?" The normal reaction is to call to mind immediately an image of a polar bear – this was not a voluntary process, however, in that had the words pointed to some other cognitive target, you would be free to think of myriad other things instead of white bears.
[8] Bargh, "The Automaticity of Everyday Life," 3.

both to physical processes – such as navigating an automobile while thinking of something entirely different – as well as cognitive processes – such as references to white bears cueing the imagination of polar bears. Bargh argues that these are deterministic processes – they satisfy 'if…then' conditionals, in that, if a particular stimulus is present we cannot but help fulfill the 'then' criterion, but offers a few key distinctions:

> The nature of these necessary preconditions (the *if* side of the equation) can vary. Some require only the presence of the triggering environmental event; it does not matter where the current focus of conscious attention is, what the individual was recently thinking, or what the individual's current intentions or goals are. In other words, this form of automaticity is completely unconditional in terms of a prepared or receptively tuned cognitive state. These are *preconscious* automatic processes…They can be contrasted with *postconscious* and *goal-dependent* forms of automaticity, which depend on more than the mere presence of environmental objects or events. Postconscious automaticity is commonly studied through the experimental technique of *priming*. Priming prepares a mental process so that it then occurs given the triggering environmental information – thus, in addition to the presence of those relevant environmental features, postconsciously automatic processes do require recent use or activation and do not occur without it. Goal-dependent automaticity has the precondition of the individual intending to perform the mental function, but given this intention, the processing occurs immediately and autonomously, without any further conscious guidance or deliberation (e.g., as in a well-practiced cognitive procedure or perceptual-motor skill). What it means for a psychological process to be automatic, therefore, is that it happens when its set of preconditions are in place without needing any conscious choice to occur, or guidance from that point on.[9]

Once the eliciting stimulus occurs, the agent has no recourse but to manifest the targeted behavior – a psychological knee-jerk response with cognitive, affective, and motivational elements. Bargh notes that this response can also influence later cognition; as such, preconscious automaticity can affect postconscious function. He understands preconscious processing to refer to the "initial state of cognition in which the world makes contact with our minds" – in essence, we cannot have a conscious experience of the world without having a preconscious experience of

[9] Bargh, "The Automaticity of Everyday Life," 3.

it first.[10] Isen and Diamond clarify Bargh's model, noting that automatic processes are best understood as 'parallel process' – they do not take up cognitive processing resources (attention or effort), so they can occur parallel to other cognitive processes which do require these resources.[11] Because it does not tax cognitive resources, automatic processing can be performed much more rapidly and earlier than other types of processing. This may explain our 'gut instincts' in certain situations – our full processing has not yet finished, leaving us with only a general impression of necessary action. Berkowitz notes that the deterministic model suggested by automaticity is frequently undervalued by many people – there is a frequent visceral objection to the idea that our cognitive processes are heavily influenced by environmental determinants. These can be manifested as objections to experimental results or methodologies or as appeals to the indeterministic claims of folk psychology. Berkowitz suggests that, if nothing else, "Persons interested in gaining a truly adequate understanding of the complexities of human conduct should at least adopt a healthy skepticism toward the assumption that conscious processes are necessarily involved in all human behavior.[12]

Preconscious processes develop as the result of conditioning – we develop patterns of psychological responses to stimuli. As is claimed by behaviorist thought, we make associations between stimuli and psychological responses, facilitating future responses along those same psychobehavioral lines. It becomes easier for stimuli to elicit behavioral, emotional, and motivational responses in us, producing automatic cognitive processing. Initially these responses

[10] Bargh, "The Automaticity of Everyday Life," 8.
[11] Alice M. Isen and Gregory Andrade Diamond, "Affect and Automaticity," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh (New York: Guilford Press, 1989), 126.
[12] Leonard Berkowitz, "Some Thoughts Extending Bargh's Argument," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 85.

require work, but like other recurring responses, the amount of conscious effort they require consistently decreases to the point where they require no conscious processing at all.[13]

This type of processing can extend to the goals we set for ourselves as well as the means of attaining them. Once we encounter a particular cognitive trigger, we can manifest complex behaviors and plans automatically, which Bargh refers to as the 'Auto-Motive Model'. These goals operate on all available information that is applicable to the situation, outside of our volitional control and regardless of whether that is the information upon which we want to fixate.[14] Bargh notes that there are significant implications for automatically activated goals:

> First, behavioral and cognitive goals can be directly activated by the environment without conscious choice or awareness of the activation. Second, the goals, once activated, direct information-processing and social behavior. Third, the states activated by the priming manipulations in these studies have motivational qualities. Fourth, these states also exist in chronic form and there are individual differences in these chronic motivations. Finally, the activated goals operate autonomously, bypassing the need for any conscious selection or choice, but producing outcomes different from those that would occur if the individual would choose if the goal were not primed. In short, every postulate of the auto-motivation model was supported by these studies, demonstrating that the entire sequence from environmental information to goal and motivation to judgment and action can and does occur automatically and unconsciously.[15]

This has serious ramifications – it means that if we encounter a particular cognitive trigger, we can initiate goals, motivations, and resultant behaviors automatically. Absent volitional control, we may not necessarily be able to control the kinds of thoughts and actions that result. In a clinical setting, for instance, a particular diagnosis may be an emotional trigger for a variety of subsequent thought processes and associations. The mere word 'cancer' may elicit a slew of memories and experiences involuntarily and instigate thought processes culminating in a comorbid depression, which may radically affect how our patient perceives his or her current

[13] Bargh, "The Automaticity of Everyday Life," 10.
[14] Interested readers should also see Carver, "Associations to Automaticity." for treatments of automaticity in goal activation.
[15] Bargh, "The Automaticity of Everyday Life," 47-8.

health and prognosis. When asked about treatment preferences, and whether the patient desires a particular course of treatment, we may have unknowingly set into action an automatic process that results in an outcome our patient might not otherwise desire. We will return to these kinds of concerns throughout the chapter.

Reaffirming the deterministic model Bargh noted, Bargh and Ferguson note that the phenomena of consciousness and willfulness are not necessarily objections to deterministic systems – they feel that it is inappropriate to believe that a deterministic model necessarily equates to a lack of choice or influence on the process.[16] They explore a key element of Bargh's previous research into automaticity – the role of awareness on control of automatic processes:

> Is this to say that one is usually not in control of one's own judgments and behavior? If by control over responses is meant the *ability* to override preconsciously suggested choices, then the answer is that one *can* exert such control in most cases. The occurrence of preconscious influences on interpretation of input and generation of evaluations is probably not controllable in the immediate, on-line sense (but is perhaps alterable through extensive and controlled rechanneling of unwanted interpretative biases, as through cognitive therapy). However, one can reduce or perhaps eliminate such preconscious influences on judgments by an intentional search for and examination of relevant evidence… But if by 'control' is meant the actual *exercise* of that ability, then the question remains open. The assertion of control over preconscious, postconscious, and context-dependent automatic influences…can only occur if one is aware of those influences. My own hunch is that control over automatic processes is not usually exercised, not so much because of a lack of motivation as because people tend not to accept the idea that there are many ways in which awareness, judgment, and behavior may be influenced without one's knowledge. As long as most people believe that they are aware of all such influences, that subjective awareness is an objective reflection of reality, and that their introspective ability is fully capable of sorting out the true causes of one's emotions and evaluations, then they will not take care to counteract the hidden preconscious biases and other unintended influences upon thought and behavior that are discussed in this chapter.[17]

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[16] John A. Bargh and Melissa J. Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," *Psychological Bulletin* 126, no. 6 (2000): 925-45, p. 925-6; see also Gordon D. Logan, "Automaticity and Cognitive Control," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh (New York: Guilford Press, 1989), 52-74.

[17] John Bargh, "Conditional Automaticity: Varities of Automatic Influence in Social Perception and Cognition," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh (New York: Guilford Press, 1989), 3-51, p. 39-40.

Bargh and Ferguson reiterate Bargh's assessment (which we will also return to in chapter four). They suggest that awareness of one's automatic reactions can assist in guiding and informing one's resulting behavior – there is a direct parallel to this in both rational-emotive and cognitive-behavioral therapies. In both of these interventions, the patient is asked to think about the automatic thoughts that resulted from a particular stimulus or situation, explore its meaning and source, and examine whether it is an appropriate or inappropriate response. By willfully breaking the stimulus → thought → emotion → behavior chain, a patient is empowered and able to exercise more control over subsequent emotional responses and his or her subsequent behaviors.[18] Bargh and Ferguson place a necessary caveat on this process, however – they feel that the higher processes which become aware of this automaticity are themselves subject to automatic and deterministic control mechanisms.[19]

In contrast to the highly deterministic model proposed, Baumeister and Sommer suggest that consciousness introduces explicitly indeterministic elements.[20] As Bargh noted, consciousness allows us to recognize when automatic processes are occurring, and to exercise control in the behavioral process. In contrast to Bargh's suggestion that conscious processes may be determined, I tend to follow Baumeister and Sommer. Introducing some indeterminacy into decisional models does not contradict underdetermined decisional models, and it allows for ownership of action with accompanying ethical valence (moral praiseworthiness/blameworthiness). It does, however, reinforce the necessity of exploring the decisions we make to ensure that they are, in fact, the result of conscious mediation, and not

[18] Some individuals have suggested that there is a significant automaticity in certain psychopathologies (see, for instance E. Tory Higgins, "Knowledge Accessibility and Activation: Subjectivity and Suffering from Unconscious Sources," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh [New York: Guilford Press, 1989], 75-123.).
[19] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," 928.
[20] Roy E. Baumeister and Kristin L. Sommer, "Consciousness, Free Choice, and Automaticity," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 75.

simply the result of underlying automatic processing. I wish to stress that there are *strongly* deterministic causal factors in cognition, and that we must be aware of the myriad influences upon our choices, especially in critical situations such as forgoing treatment.

As noted above, automatic processing can have significant 'downstream' effects (i.e., conscious effects). The clearest example of this occurs in priming studies – Bargh and Ferguson cite prior research demonstrating how affect and affective responses can be influenced by priming objects. Participants reported their moods following exposure to priming objects. Participants exposed to positively valenced priming objects reported significantly better moods than individuals exposed to negatively valenced priming objects. In short, they demonstrated that downstream mood can be determined by automatic emotional processing of earlier situations and experiences.[21] Clore and Ketelaar concur with Bargh's conclusion of (deterministic) conscious mediation of automatic processes. They note that it would be false to model behavior solely on controlled versus automatic processes. Rather, they suggest that our normal cognition is a mixture of automatic and controlled processes, and that the other phenomena of our cognitive processes (e.g., affect) do not fall into neat categories like 'controlled' or 'automatic'. They argue that affect and cognition are involved in both the conscious/unconscious and automatic/controlled domains.[22]

Automaticity, therefore, can be a powerful motivator for action, resulting in affective changes, goal activation, and deterministic mediators of conscious processes. These resultant changes are necessarily interactive and modifying causal elements of further cognition:

> It seems undeniable that conscious processes are themselves causal agents within the same deterministic framework as nonconscious processes. Conscious and nonconscious processes presumably act in concert with one another, and with

[21] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," 932.
[22] Gerald Clore and Timothy Ketelaar, "Minding Our Emotions: On the Role of Automatic, Unconscious Affect," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 118.

> stimuli outside of our bodies, according to physical laws. Any mental circuit or system that guides behavior extended over time (as opposed to single, one-off reflex responses) must have access to information in the environment to do so. That an individual is currently consciously aware of this information at the same time does not mean that the process is any less determined. If one takes any of the perception-behavior studies described above, one finds that the effect required information of which the person was consciously aware and could report on – such as walking down the hallway more slowly after priming with elderly-related stimuli or helping to pick up pens in the elevator. Yet the automatic goal operation experiments provide more telling and, in hindsight, rather obvious evidence that even controlled mental processes are themselves controlled and determined. Goals – such as to form an impression of someone, or memorize information, or achieve the best score possible on a task, or treat others fairly – are executive processes that operate on information held in working memory and 'do things with it'. This is the functional essence of a goal structure. Therefore, if these goals are nonconsciously activated and operating without the person's knowledge but still producing the same outcomes and using the same brain structures as when the goal is being consciously pursued, this means that the executive processes and working memory operations are themselves being controlled by the automatically operating goal.[23]

As a result, we see that cognition has strongly deterministic elements at all levels of pre- and post-conscious processing. These elements necessarily conflict with our folk model of cognition, in which our cognition is essentially free. As we will see, automatic processes add a further layer of complexity into cognition – there is nothing that guarantees that these automatic processes will be accurate or unbiased, a theme we will return to in our discussion of heuristics.

Cohen extends the model, noting that there are strong sociocultural determinants of perception and behavior. Our automatic perceptions, cognitions, and behaviors occur within a particular cultural context – we cannot simply ignore the specific cultural bases of cognition, or the behavior cues it initiates. He suggests that "The 'self-evident' truth of what we must do in a situation can be the product of a preconscious that is highly accultured. Things that seem like a natural stimulus-response connection differ markedly across cultures."[24] This does not seem to

[23] Bargh and Ferguson, "Beyond Behaviorism: On the Automaticity of Higher Mental Processes," 939.
[24] Dov Cohen, "Ifs and Thens in Cultural Psychology," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 123.

require significant leaps of logic – as we will see elsewhere (e.g., in conceptual blending), the particular culture in which we learn how to react to and process stimuli has instilled in us particular values and normative standards to which our actions conform. This is not to suggest that cultures produce homogeneous agents, but it is certainly much more likely that members of the same culture will react more akin to each other than to members of a radically different culture. Within this cultural context, Mischel adds an additional layer of complexity by noting that there are significant and unique causal and modifying conditions in every reasoning agent which affect how we react to external stimuli:

> But in a comprehensive analysis of social cognition, feeling, and action, the *if-then* relationships that have to be considered include internal events and conditions – the situations inside the head – such as the person's chronic affective states, styles of encoding information, self-representations and expectations (e.g., about one's own efficacy), goals, values, self-regulatory strategies, and action scripts, all of which are likely to interact with and change the impact of the external stimulus.[25]

Mischel chafes, however, at suggestions that cognition ought to be understood as a necessarily deterministic model – he notes that human goals often mediate automatic reactions, preventing us from acting on our impulses or reactions. He suggests that the conclusion Bargh reaches – i.e., that these mediating processes themselves are determined – may restrict fields of inquiry (e.g., social psychology) to the discovery of stimuli.[26] He does stress that environmental stimuli can have a strong influence over behavior, however, and does not fully divorce stimulus from response. In light of Mischel's concerns, Bargh clarifies his remarks, noting that it is not simply environmental triggers that can produce automatic responses – his analysis applies to psychological situations:

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[25] Walter Mischel, "Was the Cognitive Revolution Just a Detour on the Road to Behaviorism? On the Need to Reconcile Situational Control and Personal Control," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 181-86, p. 182-3.

[26] Mischel, "Was the Cognitive Revolution Just a Detour on the Road to Behaviorism? On the Need to Reconcile Situational Control and Personal Control," 185-6.

> In hindsight, my use of the terms *situation* and *environment*, combined with my invocation of precognitive (Skinner) and preinteractionist (e.g., Milgram) ghosts, was unintentionally (consciously, at least) misleading. By situation, I meant the *psychological* situation, and this certainly includes the immediate internal reactions of the individual…the objective external situation is not a cause as much as the taken meaning and internal experience of that situation – and this can certainly vary from individual to individual. Environmental events can directly trigger these internal reactions without the intervention or need of conscious choice…and as these internal reactions can vary from individual to individual, these automatic effects, can and do, vary as well. In fact, when these individual differences in the meanings of external situations are taken into account, substantial cross-situational consistency is shown in emotional and behavioral reactions, as Mischel's own research shows.[27]

As such, the model that emerges from this discussion is that of a consciously mediated but often deterministic, reflex processing in response to both external and internal stimuli which can have long term effects on affect, perception, and cognition. In short, the choices that we make can be heavily influenced, *but not necessarily determined*, by factors outside of our control. As clinicians, we should be very aware of the role that context and psychological stimuli have upon the decision-making process. If a patient chooses to forgo medical treatment, we would be remiss if we were not to ensure that it is done for the right reasons, and not as an automatically processed reaction to the situation in which the patient finds him or herself.

Conceptual Blending and Mental Spaces

The discussion of cognition must contain a discussion of 'mental spaces' – a theory of cognition positing the assemblage of novel ideas and constructs from earlier ideas and constructs, occurring outside of our conscious awareness.[28] Fauconnier argues that language cues give rise to cognition outside of our awareness, building complex cognitive structures that can exceed the extent of the information presented:

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[27] John A. Bargh, "Reply to the Commentaries," in *The Automaticity of Everyday Life*, ed. Robert S. Wyer (Mahwah: Lawrence Erlbaum Associates, 1997), 232.
[28] Turner, "Backstage Cognition in Reason and Choice," 266.

> In order for thinking and communicating to take place, elaborate constructions must occur that draw on conceptual knowledge, schema-induction, and mapping capabilities. Expressions of language do not in themselves represent or code such constructions – the complexity of the constructions is such that the coding, even if it were at all possible, would take very large amounts of time and be extremely inefficient. Instead, languages are designed, very elegantly it would seem, to prompt us into making the constructions appropriate for a given context with a minimum of grammatical structure. Language does not itself do the cognitive building – it 'just' gives us minimal, but sufficient, clues for finding the domains and principles appropriate for building in a given situation. Once these clues are combined with already existing configurations, available cognitive principles, and background framing, the appropriate construction can take place, and the results far exceeds any overt explicit information…We notice only the tip of the iceberg – the words – and we attribute all the rest to common sense.[29]

This seems to be a principal of spoken language, but there seems to be grounds to extend these principles. Thought must occur in some form of language of self-presentation[30] – after all, there must be some manner in which we can consciously and unconsciously consider information. In light of this, it would follow that there would be a mental space construction (which may be unique to each individual). Fauconnier suggests that *any* form of thought or cognition produces such mental spaces.[31] He stresses that these ought not to be considered simulations of reality of 'possible worlds' – as such, we ought not to envision them as such, or compare them to types of heuristics setting up simulations of possible outcomes (e.g., as in some heuristics later in this chapter).[32] These elements, however, are not necessarily accessible to us consciously – we are engaging in a phenomenon called 'backstage cognition'.

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[29] Gilles Fauconnier, *Mental Spaces: Aspects of Meaning Construction in Natural Language* (New York: Cambridge University Press, 1994), xviii.

[30] This may or may not necessarily parallel our representations of spoken language. My argument does not require that our language of self-presentation necessarily have an ordered grammar or syntax; all that is being posited is some manner in which information is brought up for cognition, and by which it is compared to, adapted by and fused with other information in the same manner in which it occurs when we create mental spaces. In light of what cognitive psychology has learned (a small sampling of which is contained in this chapter), it seems unlikely that we do not engage in some similar process for the processing of internally elicited cognitive stimuli as we do for externally elicited stimuli.

[31] Fauconnier, *Mental Spaces: Aspects of Meaning Construction in Natural Language*, xxxvii.

[32] Fauconnier, *Mental Spaces: Aspects of Meaning Construction in Natural Language*, 152.

In developing the presentation of backstage cognition, Turner notes that it potentially conflicts with the 'truths' of common experience – just as we witnessed folk theories in the last chapter, we encounter them again here regarding cognition, and they are just as mistaken. Folk theory would suggest that we are perceptually aware of the elements of our own cognition – we know both what and how we think. This idea, however, is undercut by the available evidence of cognitive science:

> The central insight cognitive science offers to social science is that reason and choice operate, indispensably and inescapably, through what Gilles Fauconnier calls 'backstage cognition.' Backstage cognition is the integrated activity of intricate, systematic, powerful, and complex mental operations of interpretation and inference. Crucially, the backstage cognition that constitutes most of reason and choice takes place outside of consciousness, and so we do not even recognize that it is happening.[33]

Any appreciation of reason and cognition, therefore, must be in light of what we now know – the cognitive processes of which we are aware are surface phenomena, and merely a subset of all the phenomena occurring when we consider choices and options. Thought and judgment are much more complex processes than our everyday folk accounting would suggest, and any model of 'rational autonomy' must account for a profound empirical criticism – 'rationality' isn't so rational after all. The implications of this for several popular ethical theories will be addressed in chapter four.

As indicated above, cognition in mental spaces involves a series of interactions between inputs (internal and external stimuli). Some inputs are projected into the mental space, where they can combine to produce new meaning ('emergent structure') that is not directly available to either of the inputs (i.e., the meaning is something novel, rather than an intrinsic property of the input). This interaction and production of novel meanings and interactions is a process Turner

[33] Turner, "Backstage Cognition in Reason and Choice," 265.

and Fauconnier refer to as 'conceptual blending'.[34] This novel meaning can then be projected back to the input in question, giving them new properties and meanings.[35] These three basic interactions lead to greater claims about the nature of conceptual blends:

> Fleshing out the claim that reason and choice depend on backstage cognition yields the following more specific claims: basic cognitive operations like conceptual blending are the basis of reasoning and choice; basic cognitive operations like conceptual blending are systematic and highly intricate; we rarely notice these basic cognitive operations or the details of their operation. Operations of backstage cognition, like conceptual blending, typically operate below the horizon of observation, too intricately for consciousness to handle, interactively with each other, on-line, quickly, [and] with powers of access and recognition not otherwise available. This combination of features is partly responsible for the power of backstage cognition. It is also partly responsible for the difficulty – notorious in cognitive science – of recognizing the existence of these cognitive operations, or the greater difficulty of noticing them as they operate, or the yet greater difficulty of analyzing what it is they do when they operate.[36]

This is a very different model than what we encounter in classical models of cognition, which posit an agent as rationally mapping out the consequences of particular actions and assigning objective probabilities to each (this will be addressed further later in the chapter). Cognition appears generally to be more *ad hoc* – judgments and meaning seem to be constructed by conceptual blending in mental spaces, rather than the results of conscious deliberation. Turner notes that evolution favors rapid processing and blending of the sort discussed above – slower processes like the classical model do not appear to have evolutionary consequences. Instead "What does have consequence for evolution is whether, in total, over decades, you manage, sometime, to make certain things happen. On that requirement, human reasoning needs to be

[34] Turner, "Backstage Cognition in Reason and Choice," 266.
[35] Turner, 268.
[36] Turner, 272.

inventive, creative, reliable, strategic, opportunistic, social, natural, effective, and nonfatal, but it does not need to be especially good at cascading implication."[37]

What, then, would characterize cognition in this model? We have already seen that it is a backstage process – it is a fast process by which we can rapidly respond to environmental and discursive stimuli. These blends can accommodate information from a wide variety of sources, and we associate them with frames of meaning (e.g., contextualizing meaning).[38] Fauconnier and Turner offer pattern completion as an example of conceptual blends drawing information from a variety of sources to give meaning to an incomplete series or pattern: "We see some parts of a familiar frame of meaning, and much more of the frame is recruited silently but effectively to the blend."[39] Further, the blends that result from mental spaces are not necessarily deterministic – they stress that it is nonsensical and counter to actual human thought to require that a specific blend *must* result from any particular two inputs. How we interpret inputs can vary, and therefore the effect it has on us can vary as a result – we will return to the means by which involuntary or nonconscious processing can affect cognition in our discussion of heuristics later in the chapter.

Continuing the description of what characterizes the proposed cognitive model, we are aware of the end products of this blending, but not the processing that brings it about:

> The perception available to consciousness is the *effect* of complicated interactions between the brain and its environment. But we integrate that effect with its causes to create emergent meaning: the existence of a *cause* – namely, the cup – that directly presents its *effects* – namely, its unity, color, shape, weight, and so on. As a consequence, the effect is now in its cause: the color, shape, and weight are now intrinsically, primitively, and objectively in 'the cup.' In perception, at

[37] Turner, "Backstage Cognition in Reason and Choice," 285.
[38] By way of clarification, this discussion of framing ought not to be understood in terms of how questions are framed, or other equivocable meanings. Frame of meaning here refers to a general frame like "sitting at a table" or to specific frames like "sitting at a table in the Barnes and Noble café at 6:47 PM on May 14th." These frames can affect how we recall information and how we associate it with other meanings.
[39] Gilles Fauconnier and Mark Turner, *The Way We Think: Conceptual Blending and the Mind's Hidden Complexities* (New York: Basic Books, 2002), 48.

> the level of consciousness, we usually apprehend only the blend of cause and effect. We cannot fail to perform this blend, and we cannot see beyond it…Brain damage, psychoactive drugs, and certain neurobiological syndromes can cause breakdown of these integrations and consequent bizarre perceptions. But for the most part, when we are functioning normally, consciousness cannot see the rest of the blending network.[40]

The material that is drawn into the blend does not have to be part of the current stimulus – it is entirely possible for one to draw upon old experiences and memories as inputs into a conceptual blend. This will be an important part of the heuristic model as well – experience and memory provide the information accessed most readily, in addition to emotional valences. We are not necessarily aware of all of the blends that our minds produce – as it is a backstage process, it is entirely possible for meanings and associations to be blended, but to be preconsciously rejected in favor of other interpretations.[41] They may be rejected for a variety of preconscious reasons; while we do not presently have a full accounting of preconscious processes or reasoning (and, in light of our complexity, one might reasonably ask whether we will *ever* have such an account), we have several candidate theories in heuristics-and-biases, ecological rationality, bounded rationality, and 'fast and frugal' heuristics. These will all be addressed later in this chapter.

A further characteristic of the blends we form is their dependence on our cultural setting; it does not seem to be contentious to suggest that how we interpret environmental and cognitive stimuli will be informed by our cultural context. Ignoring temporal differences between cultures and societies, we can see that individuals from contemporaneous and sympatric communities can form vastly different associations based on biological and sociocultural cues.[42] They provide us with different experiences with concomitant differences in frames of reference; as such, we blend

[40] Fauconnier and Turner, *The Way We Think: Conceptual Blending and the Mind's Hidden Complexities*, 78-9.
[41] Fauconnier and Turner, 321.
[42] I test this perception every semester in my Introduction to Philosophy class when we discuss determinism. It is interesting to see how many ardent supporters of indeterminism readily admit that our perspective, method of cognitive processing, and interpretations of external and internal stimuli are affected by such determinants as genes, age, gender, race, social class, upbringing, degree of education, and exposure to other cultures.

different elements with common stimuli. Fauconnier and Turner suggest that there is "no other way for us to apprehend the world" – forming conceptual blends is an automatic and essential part of our cognitive lives. It is a requisite part of our consciousness and memory.[43]

COGNITIVE HEURISTICS

How do we approach the world cognitively? How do we make sense of the myriad sense phenomena that greet us every waking moment of every day of our lives? How do we make associations between past, present, and future? Are we homuncular theorists, picking and choosing the information we will use in formulating a judgment? Are we purely rational, coolly and calmly weighing evidence and choosing a utility-maximizing solution? Are we intuitive, relying on elements of cognition and impressions that we cannot fully explain, but seem compelling? Any of these? None? A mixture thereof? These questions are both fascinating and fecund, but regrettably outside the purview of this work. What will have to suffice is a treatment of several fundamental ideas, building to a model that notes individual differences in cognition, the inapplicability of a purely rational utility calculus, and the necessity of a conception of bounded rationality – we cannot exceed our innate limitations as human cognizers.

Nisbett et al. note that the dominant theory "poses a view of man as lay scientist, attempting to infer causes for the effects he observes. The causes he attributes determine his view of his social world, and this view may determine his behavior."[44] Making generalizations about the world as he understands it leads to patterns in interpretation and perception. These patterns then influence and modify future events and experiences, and are in turn modified by these experiences. These schema are selective, however, in what information they take in and

[43] Fauconnier and Turner, 390-1.
[44] Richard E. Nisbett, et al., "Popular Induction: Information is not Necessarily Informative," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 101.

how they process it. Specifically, the data that are subjectively most influential are not necessarily the strongest information:

> We believe that the present research and examples drawn from everyday life show that some kinds of information that the scientist regards as highly pertinent and logically compelling are habitually ignored by people. Other kinds of information, logically much weaker, trigger strong inferences and action tendencies. We can think of no more useful activity for psychologists who study information processing than to discover what their subjects regard as information worthy of processing.[45]

In essence, the way we think about many things is not necessarily based on the strongest information or the most accurate understanding of what information we do choose to focus on. Further, we are often called upon to evaluate novel situations, or at least situations that are novel to us. In this context, we find that there are several typical constraints upon what we view as likely versus unlikely, based upon any germane or potentially relevant information we possess. We construct scenarios to evaluate how we can reach the targeted outcome; the more plausible the scenarios we discern, the more likely the target event. We have innate limitations, however, on how much information we can manage in constructing these scenarios; as a consequence, we tend to only alter simple elements or factors, which may not conform to reality or may be counterintuitive.[46] Further, once we construct a particular scenario, we tend to find it difficult to imagine other possibilities – we become tied or 'anchored' to one given possible explanation or course of action (see below), which limits our ability to generate further scenarios or to see other potential outcomes.[47] Kahneman and Tversky further note that in judging probabilities and unknowns, our decisions are only adequate if the judgment is in accord with the entire collection of beliefs held by the thinking agent. This poses a problem in assessing rationality: there is no

[45] Nisbett, et al., "Popular Induction: Information is not Necessarily Informative," 116.
[46] Amos Tversky and Daniel Kahneman, "Availability: A Heuristic for Judging Frequency and Probability," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 177.
[47] Tversky and Kahneman, "Availability: A Heuristic for Judging Frequency and Probability," 178.

simple way to check whether any particular set of probability judgments are compatible with the individual's collective whole. Instead, the individual simply strives for compatibility with his knowledge, assessments of probability, and his own heuristics and biases.[48] In other terms, the individual strives to make his decision as authentic as possible.

We also respond differently when we begin to add information into our cognitive schema. Our mind occasionally has difficulty filtering useful information from worthless information – studies demonstrate that "people respond differently when given no evidence and when given worthless evidence. When no specific evidence is given, prior probabilities are properly utilized; when worthless evidence is given, prior probabilities are ignored."[49] When information is present, we assign it decisional weight and importance, but may potentially give it undue weight, leading us to become either overly reliant upon that particular piece of information (anchoring), or overly confident in our assessment of its worth, a failure rampant across lay and professional decision makers:

> The weighing of evidence and the formation of belief are basic elements of human thought. The question of how to evaluate evidence and assess confidence has been addressed from a normative perspective by philosophers and statisticians; it has also been investigated experimentally by psychologists and decision researchers. One of the major findings that has emerged from this research is that people are often more confident in their judgments than is warranted by the facts. Overconfidence is not limited to lay judgment or laboratory experiments. The well-publicized observation that more than two-thirds of small businesses fail within four years suggests that many entrepreneurs overestimate their probability of success. With some notable exceptions, such as weather forecasters, who receive immediate frequentistic feedback and produce realistic forecasts of precipitation, overconfidence has been observed in judgments of physicians, clinical psychologists, lawyers, negotiators, engineers,

[48] Amos Tversky and Daniel Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 20.
[49] Tversky and Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," 5.

> and security analysts. As one critic described expert prediction, 'often wrong, but rarely in doubt.'[50]

But this seems to conflict with our traditional understanding of humans as rational agents – surely a rational agent employs a more systematic means of assessing probabilities and novel situations. Simon notes that traditional cognition has been modeled around subjective utility theory (SEU) – a model in which the cognizer follows certain procedures and rules in constructing probability estimates and judgments based on a utility-maximizing calculus. There are four key assumptions of such models:

> First, the theory assumes that a decision maker has a well-defined *utility function*, and hence that he can assign a cardinal number as a measure of his liking of any particular scenario of events over the future. Second, it assumes that the decision maker is confronted with a well-defined *set of alternatives* to choose from. These alternatives need not be one-time choices, but may involve sequences of choices or strategies in which each subchoice will be made only at a specified time using the information available at that time. Third, it assumes that the decision maker can assign a consistent *joint probability distribution* to all future sets of events. Finally, it assumes that the decision maker will (or should) choose the alternative, or the strategy, that will *maximize the expected value*, in terms of his utility function, of the set of event consequent on the strategy.[51]

After all, these would all seem to be logical and necessary assumptions in any decisional algorithm – a common standard to decide between distinct alternatives, appreciation of the consequences of these choices, and the process culminating in the choice that maximizes the return the agent receives as measured by the common standard. In a clinical setting, this is a description of our idealized patient and our ideal of informed consent – authentic choices predicated on an understanding of the procedures and risks involved and knowledge of the reasonably predictable outcomes. There is a problem, however – this standard is impossible.

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[50] Dale Griffin and Amos Tversky, "The Weighing of Evidence and the Determinants of Confidence," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 230.

[51] Herbert A. Simon, "Alternative Visions of Rationality," in *Rationality in Action: Contemporary Approaches*, ed. Paul K. Moser (New York: Cambridge University Press, 1990), 194.

Human cognition does not follow this pattern of utility maximization; our cognition is characterized by values, emotions, prior knowledge, raw intelligence, and many other factors that do not fit nicely into this idealized model. Simon's first objection to the SEU model is that it assumes that we are capable of viewing everything at once, and seeing the influences between these disparate elements. This is an unrealistically optimistic assessment of what we can do – we are far more likely to hone in on a few key instances and base our cognition upon them (e.g., availability and anchoring). Second, the SEU model assumes that we have enough prescience to appreciate the full range of alternatives and consequences of each for our lives; again, this is an unrealistic expectation. While we can certainly conceive of a handful of alternatives and some of their consequences, we cannot do this in a lump-sum manner or easily decide between competing factors. Third, he notes that the SEU assumes that we have somehow managed to reconcile all of our fully and partially-formed values and have been able to synthesize them into a single scale. This is extraordinarily unrealistic – conceptually one would have to find common denominators for issues of morality, philosophy, theology, health, lifestyle, and psychosocial function. Each of these factors presents unique challenges and definitional debates and controversy – it seems to be a stretch of logic that they can be contained on a single scale. For all of these reasons, Simon proposes that the SEU model has never actually been applied, and in fact can never be applied, especially in questions of human cognition.[52]

As alternatives to this Olympian model (so named due to the superhuman requirements it necessitates), Simon proposes two models that interact and together characterize human cognition. The first model, which he characterizes as 'bounded rationality', is essentially behavioral, has several characteristic elements. Posing a challenge to his reader, Simon suggests that the following are characteristic of most of the judgments we make:

[52] Simon, "Alternative Visions of Rationality," 195.

> First, your decisions are not comprehensive choices over large areas of your life, but are generally concerned with rather specific matters, assumed, whether correctly or not, to be relatively independent of other, perhaps equally important, dimensions of life…Second, when you make any particular decision, even an important one, you probably do not work out detailed scenarios of the future, complete with probability distributions, conditional on the alternative you choose…Third, the very fact that you are thinking about buying a car, and not a hose, will probably focus you attention on some aspects of your life and some of your values to the relative neglect of others…Hence, it is unlikely that a single comprehensive utility function will watch over the whole range of decisions you make. On the contrary, particular decision domains will evoke particular values, and great inconsistencies in choice may result from fluctuating attention.[53]

These characteristics are defining elements of bounded rationality (Simon includes a fourth, noting that we also routinely seek out information for some of our judgments). As a philosophical/psychological approach, it implicitly denies that humans generally are capable of the same kinds of algorithmic processes we program into computers – our cognition is dynamic, predicated on fast and efficient thought. From an evolutionary perspective, we have neither the time nor the energy to commit to an algorithmic thought process for all of our decisions.[54] We will return to bounded rationality later in this chapter.

Fast and efficient processing does not mean that we will necessarily always make the same choices – the same backstage elements can produce different motives depending on the context and what we (preconsciously) judge to be relevant, a point first mentioned in chapter one. Simon notes that:

> Rationality of the sort described by the behavioral model doesn't optimize, of course. Nor does it even guarantee that our decisions will be consistent. As a matter of fact, it is very easy to show that choices made by an organism having these characteristics will often depend on the order in which alternatives are presented. If A is presented before B, A may seem desirable or at least satisfactory; but if B is presented before A, B will seem desirable and will be chosen before A is even considered.[55]

[53] Simon, "Alternative Visions of Rationality," 197.
[54] Simon, 198.
[55] Simon, 200.

This may come as a surprise, but this concept should become less controversial as the present argument progresses – a recurring theme in this chapter is that how we perceive information, its relevance, and its application are dependent upon a variety of issues that may not necessarily be important to, or even connected with, the information in question (e.g., the order in which information is presented should not make a difference to us, in that it's objective truth value is not contingent upon the order in which we see it). Just like judging the probability of an outcome based upon what we expect to see represents a source of bias and error, so too could the order of information exert undue influence on our perception of its weight and merit.

The second model Simon proposes stems from intuitive knowledge – some individuals have demonstrably greater knowledge than others (i.e., specializations, whether it be medicine, botany, or chess), and can come to conclusions based upon that greater knowledge intuitively, while others with less experience require much more conscious effort (e.g., a physical chemist who has memorized a reactivity series intuitively will be able to predict the probability and spontaneity of a given reaction much more readily than a general chemistry student, who will have to examine charts to determine which materials are more reactive than others, perform the relevant stoichiometry, and calculate the relevant enthalpy, entropy, and free energy.).[56]

These two models, intuitive and behavioral, do not contradict each other, nor are they specialized to specific hemispheres. Simon notes that "All serious thinking calls on both modes, both search-like processes and the sudden recognition of familiar patterns. Without recognition based on previous experience, search through complex spaces would proceed in a snail-like fashion. Intuition exploits the knowledge we have gained through our past searches.[57]

[56] Simon, "Alternative Visions of Rationality," 203.
[57] Simon, 203.

Stanovich and West note that throughout the literature, the models of cognition that emerge indicate two discrete processes – the first is generally an automatic, heuristic based system (referred to as System 1), while the second is a much more controlled process that decontextualizes and depersonalizes information (two-process models will be explored more thoroughly in the discussion of alternative theories below). These two processes operate in parallel, allowing them to potentially override some System 1 cognition.[58] As part of this parallel processing model, we find that there are unique issues that arise as a result of contextualizing information – what Stanovich and West refer to as a 'computational bias':

> The fundamental computational bias is meant to be a global term that captures the pervasive bias towards the contextualization of all informational encounters. It conjoins the following processing tendencies: (a) the tendency to adhere to Gricean conversational principles even in situations that lack many conversational features; (b) the tendency to contextualize a problem with as much prior knowledge as is easily accessible, even then the problem is formal and the only solution is a content-free rule; (c) the tendency to see *design* and pattern in situations that are either undersigned, unpatterned, or random; (d) the tendency to reason enthymematically – to make assumptions not stated in a problem and then reason from those assumptions; (e) the tendency toward a narrative mode of thought. All of these properties conjoined together represent a cognitive tendency toward radical contextualization. The bias is termed *fundamental* because it is thought to stem largely from System 1 and that system is assumed to be primary in that it permeates virtually all of our thinking. If the properties of this system are not to be the dominant factors in our thinking, then they must be overridden by System 2 processes so that the particulars of a given problem are abstracted into canonical representations that are stripped of context.[59]

This type of error does not occur in isolation – we have a tendency to find patterns in the information around us, regardless of whether there is actually a pattern to be found, due to the information we include as germane to the current situation and how we choose to perceive it.

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[58] Keith E. Stanovich and Richard West, "Individual Differences in Reasoning: Implications for the Rationality Debate," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 436.
[59] Stanovich and West, "Individual Differences in Reasoning: Implications for the Rationality Debate," 438-9.

In light of all of this, the image that emerges demonstrates that we have a tendency to process information in a manner which may seem rational, but which produces apparently irrational results (i.e., basing decisions on irrelevant or worthless data is irrational [e.g., basing a choice to forgo medical treatment based on the price of coffee in Barbados]). As Ashcraft notes, this is an interesting characteristic of much of our 'everyday reasoning'.[60] This apparently paradoxical cognitive processing and the many subtypes found within are collectively are referred to as 'heuristics' – a term we turn to now.

Definitions

In general, heuristics refer to the rules we employ during cognition that are optimized for quick interpretations of data and situations, based on probabilities or frequencies. Schwartz traces the etymology of the term back to ancient Greek (*heuriskein* – to discover), in that these heuristics are discovered 'rules' we apply to specific situations and contexts to make sense of them. However, Schwartz notes, experientially-based heuristics are not necessarily good guides – we can have experiences that skew our perceptions in unrealistic or biased ways.[61] The probabilistic interpretation has been linked to Kahneman and Tversky's research (see below), and will be the default assumptions for the purposes of this work. Alternatives to the heuristics proposed and discussed will be covered below.

Ashcraft cogently summarizes the traditional contrast between algorithmic cognitive processes and heuristic processes, noting that they employ very different methodologies:

> In many reasoning and problem-solving settings there are two general approaches that can be taken in order to achieve a problem solution or reason out an appropriate answer. One approach is termed an algorithmic approach, and the other is a heuristic approach. An algorithm is a *specific rule or solution procedure, often quite detailed and complex, that is guaranteed to furnish a*

[60] Ashcraft, *Human Memory and Cognition*, 561.
[61] Steven Schwartz, "Heuristics and Biases in Medical Judgment and Decision Making," in *Applications of Heuristics and Biases to Social Issues*, ed. Linda Heath, et al. (New York: Plenum Press, 1994), 46.

> *correct answer if it is followed correctly…*A heuristic is a '*rule of thumb,*' as opposed to a formal specified rule. It's an informal, 'seat of the pants' strategy or approach that works under some circumstances, for some of the time, but *is not guaranteed to yield the correct answer*.[62]

This is an area of potential concern: by relying on heuristics – a characteristic of much of our everyday cognitive operation – we open ourselves to systematic errors in information processing and decision-making. We rarely have the time or opportunity to engage in algorithmic processing, and as a consequence, we have adapted and adopted a method of handling information in a manner that is generally fast, helpful and useful, if potentially mistaken. Gilovich and Griffin raise three concerns that are necessary caveats to the discussion that follow.[63] First, although heuristics have reasoning patterns distinct from algorithmic processes and open the door to potential error, they should not be understood as irrational. Second, despite the characterization of heuristics as 'quick and dirty' tools, they are based on highly sophisticated cognitive processes. Third, these heuristics are normal responses to all types of questions asked – they are not simply invoked in atypical or unlikely situations. In fact, these cognitive processes are very similar to the concepts involved in automaticity, as discussed earlier in this chapter. They further note that research in heuristics "stresses the fact that much of mental life is not the product of deliberate processing, but of quicker, more reflexive processes that are less available to conscious intervention."[64]

What emerges from these observations is a cognitive model that is constantly invoked across instances that allows for fast interpretation of data and circumstances, that generally is adaptive and beneficial, and is potentially open to error. In a clinical setting, this translates into

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[62] Ashcraft, *Human Memory and Cognition*, 541.
[63] Thomas Gilovich and Dale Griffin, "Introduction - Heuristics and Biases: Then and Now," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 3.
[64] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 16.

issues applicable to both the patient and to the members of his treatment team.[65] As will be addressed later, these cognitive issues are not limited to particular people; they are characteristic of the way all of us think. We will further refine this definition and our cognitive model when we address dual system theories of cognition below. Finally, there is some controversy about these heuristics – in some paradigms they are seen as principally sources of bias and error, while others see them as evolutionary adaptations designed to benefit the species.

Specific Heuristics

There are many heuristics that have been proposed and researched, but in this work we will focus on only a handful that seem particularly germane. Specifically, we will focus on availability, representativeness, anchoring, simulation, durability, automated choice, and proposed metaheuristics. Each of these presents salient aspects of cognition that generally assist individuals in making decisions, but may also introduce sources of methodological error, and each will be addressed in turn.

Availability

The availability heuristic refers to the ease with which information is recalled. In essence, events or situations that are very vivid or that frequently occur are called to mind more readily than other events or situations.[66] Kahneman and Tversky offer the following example:

> For example, one may assess the divorce rate in a given community by recalling divorces among one's acquaintances; one may evaluate the probability that a politician will lose an election by considering various way in which he may lose support; and one may estimate the probability that a violent person will 'see' beasts of prey in a Rorschach card by assessing the strength of association between violence and beasts of prey. In all these cases, the estimation of the

[65] David M. Eddy, "Probabilistic Reasoning in Clinical Medicine: Problems and Opportunities," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 249-67; Tversky and Kahneman, "Judgment Under Uncertainty: Heuristics and Biases."; Neal V. Dawson and Hal R. Arkes, "Systematic Errors in Medical Decision Making: Judgment Limitations," *Journal of General Internal Medicine* 2 (1987): 183-87.

[66] Tversky and Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," 13.

> frequency of a class or the probability of an event is mediated by an assessment of availability. A person is said to employ the availability heuristic whenever he estimates frequency or probability by the ease with which instances or associations could be brought to mind.[67]

As a consequence, we tend to base our judgments and perceptions based upon what scenarios are called to mind, regardless of whether these may be the most accurate source of information. While it is almost a truism to note that, in general, the more frequent an event is the more probable it is that it will occur, we can unintentionally introduce a source of error into our cognition when we introduce outside factors unrelated to frequency into our probability estimates.[68] A common phenomenon, for instance, is when a celebrity is diagnosed with a particular disease or disorder. Public health centers note a spike in individuals seeking testing following the publication of the diagnosis, indicating that people are more likely to believe that they may have the disease or disorder, regardless of the actual base rate of the condition – an example of bias or error in cognition. Further, the more someone is preoccupied with a given outcome, the more available that outcome is to future cognition, and as a consequence, the more likely it is perceived to be.[69] Ashcraft extends Kahneman and Tversky, noting that *any* salient factor which causes us to remember a piece of information may potentially be a source of cognitive error:

> Basically, any factor that leads to storage of information or events in memory can influence our reasoning here, since our judgments are based on what can be remembered easily. If reasonably accurate and undistorted information is in memory, then the availability heuristic probably does a reasonable job. But to the extent that our memory contains information that is inaccurate, incomplete, or influenced by factors other than objective frequency, there may be biases and distortions in our reasoning. In particular, any factor besides frequency that calls attention to the event may make the event more memorable, make it stand out more in memory…such events may be more *accessible* for retrieval. This will

[67] Tversky and Kahneman, "Availability: A Heuristic for Judging Frequency and Probability," 164.
[68] Tversky and Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," 11.
[69] Tversky and Kahneman, "Availability: A Heuristic for Judging Frequency and Probability," 178.

> bias our estimates, since the ease of retrieval would be influenced not simply by frequency but also by those other memorability factors.[70]

This is a key concern, as it speaks to a variety of potential reasons we may choose to remember an event – emotional valence, salience, impact on then contemporaneous activity or judgment, etc. This feature of availability will become especially relevant in chapter four, when we discuss the debate between depressive realism and depressive bias.

There are a few issues to be addressed with availability, however. Dawson and Arkes, for instance, caution that employing availability does not necessarily lead to error – there is no inherent bias in recalling frequent events more readily than infrequent events.[71] There is a reason why frequent events come to mind so readily – in general they do occur more frequently, and therefore we are justified in believing them to be more probable. In fact, rather than viewing such frequency-based recall as a source of cognitive error, some have suggested that this is an evolutionary advantageous cognitive schema (e.g., Gerd Gigerenzer [see below]). Schwartz notes that there is reason to avoid embracing availability too readily – there have been relatively few experiments which demonstrate explicitly that subjective probability estimates are based on ease of information recall.[72] He argues that most of the data have demonstrated correlation, not necessarily causation. As such, it may simply be happenstance that when we make probability judgments we are recalling germane events contemporaneously.

Schwarz and Vaughn extend this concern, noting that there is an implicit confound in experiments designed to test availability – efforts to facilitate the ease of recall may also affect the amount of information recalled. They note that "In most real-world situations, these two factors are naturally confounded. Unfortunately, this confound renders it difficult to determine if

[70] Ashcraft, *Human Memory and Cognition*, 553.
[71] Dawson and Arkes, "Systematic Errors in Medical Decision Making: Judgment Limitations," 184.
[72] Schwartz, "Heuristics and Biases in Medical Judgment and Decision Making," 51-2.

the obtained estimates of frequency, likelihood, or typicality are based on participants' phenomenal experiences or on a biased sample of recalled information."[73] This is a real concern, they note, as agents tend not to recall every piece of information that is salient, but rather tend to cut the process short. It is possible, however, to disentangle the two factors and thereby eliminate the confound, restoring confidence to the original data on availability. They note that:

> [T]he reviewed research highlights that recall tasks render two distinct sources of information available: the recalled content and the ease or difficulty with which it could be brought to mind. In most situations, these two sources of information are naturally confounded and the experience of ease of recall goes along with a greater amount of recall. This confound rendered many of the classic tests of the availability heuristic nondiagnostic. When this confound is disentangled, however, the available evidence supports the original formulation of the availability heuristic: Individuals estimate the frequency of an event, the likelihood of its occurrence, and its typicality 'by the ease with which instances or associations come to mind.'[74]

As such, we can have confidence in the cognitive model that influences our judgments based on the ease of recall of specific pieces of information. We must, however, remain cognizant that this necessarily introduces a source of error into the process – the information that we recall may not be the most accurate or directly germane to the judgment at hand.

Representativeness

Representativeness refers to our predisposition to evaluate questions of probability based on whether or not we feel the outcome proposed resembles the outcome we expect to see, i.e., is the outcome or probability representative of our impression of the situation. Tversky and Kahneman propose this as an explanation of the phenomenon observed when individuals are asked to select the likely profession of a person based upon their characteristics; if someone is

[73] Norbert Schwarz and Leigh Ann Vaughn, "The Availability Heuristic Revisited: Ease of Recall and Content of Recall as Distinct Sources of Information," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 104.
[74] Schwarz and Vaughn, "The Availability Heuristic Revisited: Ease of Recall and Content of Recall as Distinct Sources of Information," 117-8.

educated and professionally dressed, we are more likely to view this person as a lawyer or doctor instead of a farmer, even if base-rate probabilities of professions are provided and run contrary to our expectations.[75] We look for patterns in data and situations, in defiance of actually probabilities; for instance, when asked which result of six coin tosses is more probable – HHHHHH or HTHTTH – most people will pick the latter, because it looks more like a random distribution, which is what we would expect. Many people feel that HTHTTH is more *representative* of six coin tosses, despite the fact that both options are *equally* probable. In light of this, Ashcraft characterizes representativeness as "a judgment rule in which your estimate of the probability of an event is determined by one of two features: (1) how similar the event is to the population of events it came from, or (2) whether the event seems similar to the process that produced it."[76] This can have clinical impacts as well, in the sense that we may feel that certain states are more indicative or representative of a given diagnosis or intervention. For instance, it is possible that a patient may associate limited medical intervention (e.g., mechanical ventilation) with full intervention (mechanical ventilation, artificial nutrition and hydration, dialysis, etc.). When asked to visualize their life with mechanical ventilation or artificial nutrition and hydration, a patient may pick a more extreme vision than warranted, simply because he feels it is more representative of medical interventions, a potential source of bias and error.

Anchoring

Anchoring refers to a variety of phenomena. Chapman and Johnson note at least three different understandings of the term:

> Because it has been used in many different areas, the term *anchoring* has been used to mean somewhat different things. We group these definitions into three types: One refers to an *anchoring procedure* in which a salient but uninformative number is presented to subjects. A second meaning is an *experimental result*, in

[75] Tversky and Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," 4.
[76] Ashcraft, *Human Memory and Cognition*, 545.

> which the uninformative number influences the judgments. Finally anchoring and adjustment is sometimes used to refer to the *psychological process* by which the uninformative number has its effect.[77]

In the context of this work, I will use anchoring to refer to a tendency to attach one's cognition to one or a small group of characteristics subjectively viewed to be salient to the decision in question. The deciding agent assigns significant weight, consciously or unconsciously, to this factor or these factors, which may potentially result in skewed interpretation or perception. Parallels abound in a variety of contexts[78] – conscious anchoring in politics, when a voter hinges her vote on a single issue, diagnosis, when a physician places undue weight on a particular lab result (e.g., insisting a tendon tear is impossible because he/she didn't see it on an MRI) or diagnosis (e.g., insisting on the presence of bipolar disorder or schizophrenia without the patient having met sufficient diagnostic criteria or with the patient having met the criteria for a less common disorder). We have a tendency to subjectively weigh information and thereby assign it precedence in cognition. The potential for error occurs when the weight assigned is inappropriate, or leads to pathological states (e.g., rumination). Certain terms have this effect when they are included as part of a differential; for instance, if a patient were presented with a list of possible causes for hemo-positive stool – hemorrhoids, gastric ulcer, colorectal cancer, tear in the intestinal vasculature, etc. – the likely result is that they will anchor on 'cancer', despite an absence of other risk factors and the higher probability of another cause. As a consequence, it is likely that cognition about his current medical condition will revolve around this one element of the differential (cognitive weight greater than is warranted by the presentation) – the patient has 'anchored' on the diagnosis of cancer.

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[77] Gretchen B. Chapman and Eric J. Johnson, "Incorporating the Irrelevant: Anchors in Judgments of Belief and Virtue," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 121.
[78] Chapman and Johnson, "Incorporating the Irrelevant: Anchors in Judgments of Belief and Virtue," 137-8.

The reason for concern in the context of this work is the weight patients may assign to elements in their lives that may or may not be affected by the treatment being offered – if patients anchor on a particular element of their life, they may potentially skew their cognition, resulting in a choice that, in retrospect, they may regret. Our clinical concern should be to ensure that the choices made by our patients are as accurate as they can be, and to challenge undue weights and other sources of error – after all, it is quite possible that the patient will be making a terminal choice. We ought to make sure that that choice is made for the right reasons.

Simulation

The simulation heuristic refers to situations in which we are asked to imagine either a future outcome or what things would be like were an event to turn out differently. Ashcraft notes that the heuristic derives its name from its parallel to computer simulation, in that we are asked to simulate a future, alternate reality or hypothetical situation.[79] This simulation is aided or hindered by the availability of information (in the sense of the availability heuristic) which allows us to construct plausible scenarios – this 'ease of construction' is the key factor in the function of the simulation heuristic. Citing Kahneman and Tversky, Ashcraft notes that individuals tend to make 'downhill changes' – that is, people change the circumstances in simulations to make them seem more 'normal'. This is to say that we have a tendency to replace those elements which seem unusual with seemingly more usual events (replacing usual with unusual events is making an 'uphill change'). This represents a cognitive error in that it fiats probabilities on events which may be unwarranted – sometimes the unusual is usual. We discount the unusual in our simulations simply because they may be "difficult to imagine or construct."[80] This leads to a potential problem – we can create a positive bias based on the ease

[79] Ashcraft, *Human Memory and Cognition*, 555-6
[80] Ashcraft, 557-9.

of the construction of our simulation, leading us to expect positive outcomes more than is realistically warranted.[81] We will return to unrealistic optimism in chapter four, in the discussion of depressive realism versus depressive bias. For the moment, it will suffice to note that errors in simulation heuristics are related to the phenomenon of bias in affective forecasting and the durability bias.

Durability Bias

Durability bias and affective forecasting refer to common sources of error in predicting how we will feel about a particular events in the future. Research has indicated that people frequently make mistakes in how they will feel about a particular life event or quality of life in the future. This has immediate clinical impact – patients frequently state that there are states of living that they would not want to experience or persist in. Paralyzation, amputation, and persistent or terminal illness frequently are referenced as states to which many people are averse. The literature, however, notes that when these individuals find themselves in such states, many have found their lives still have personal value and meaning.

Gilbert et al. explored this phenomenon, and found several potential triggers for the common (mis)perception that people have regarding how they will feel about future events, and how long those feelings endure. Citing eleven previous studies, they note a demonstrable overestimation of the extent to which people anticipate negative life events will affect them.[82] They note that most people are reasonably happy and emotionally stable most of the time, despite experiencing profoundly positive or negative life events. Why, then, would people continue to overestimate the duration of affective responses? Gilbert et al. suggest that there a

[81] Ashcraft, *Human Memory and Cognition*, 560.
[82] Daniel T. Gilbert, et al., "Durability Bias in Affective Forecasting," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 293.

variety of potential causes: misconstrual, inaccurate theories, motivated distortions, focalism, and immune neglect. Each of these will be addressed in turn.

Misconstrual refers to a tendency to ignore other possible interpretations and conceptualizations of the future event or emotional state. People have a tendency to imagine events as being more powerful than they actually are, and as a consequence, they imagine that the emotional response it provokes will also be more powerful and of longer duration than warranted.[83] In a clinical context, individuals may overestimate how profoundly an illness will impact their lives, and as a consequence, may overestimate how long they will feel burdened or depressed about the illness.

Discussion of inaccurate theories refers to misunderstanding a situation, whether due to understanding too little, too much, or by having the situation modified by culture or experience.[84] Some of the knowledge that results from our perception and understanding is likely to be wrong – aside from potentially misremembering our experiences and/or their context-specific details, we tend to give our experiences emotional valences, but we also tend not to properly remember these valences or why we assigned them as we did. When we attempt to make rules based on these early experiences and emotional valences, we tend to make errors when predicting how we will respond emotionally to future situations. We will return to the topic of emotional valence at the end of this chapter.

Motivated distortions stem from the fact that affective forecasts inspire emotion at the time the forecast is made. For instance, if someone feels good about getting married, he may extend that emotional reaction to cover how he will feel throughout the marriage.[85] In a like manner, people may engage in 'defensive pessimism' – they will overestimate their negative

[83] Gilbert, et al., "Durability Bias in Affective Forecasting," 294.
[84] Gilbert, et al., 294.
[85] Gilbert, et al., 294.

experience so that when things turn out better than expected, they are pleasantly surprised. This has significant clinical correlates – people brace for bad diagnoses, and can overestimate how profoundly the diagnosis will affect them as a result. Many people feel very averse to contracting cancer, and this negative valence can set up an expectation of misery and suffering, which can then lead to an overestimation of how they will feel throughout the course of their illness, should they ever be diagnosed.

Focalism refers to the tendency to focus solely on the event in question, and in the process, ignore the larger social context of the future event.[86] Personal events do not occur in isolation – our lives are not static, and we can easily ignore mitigating factors or events about which we will feel good. In a clinical context, of someone focuses solely on her diagnosis of cancer, she may focus on that to the extent that she ignores the possibility of other personal and familial triumphs and opportunities. She may ignore the very real chance that other events will assist in her emotional coping and recovery.

The prime focus of Gilbert et al.'s article is on the phenomenon of immune neglect – drawing a parallel to physical health, they note the presence of a psychological immune system that enables people to maintain a relatively stable state of mental health:

> In science, literature, and folklore, people are famous for making the best of bad situations, remembering their successes and overlooking their excesses, trumpeting their triumphs and excusing their mistakes, milking their glories and rationalizing their failures – all of which allow them to remain relatively pleased with themselves despite all good evidence to the contrary. Psychologists from Freud to Festinger have described the artful methods by which the human mind ignores, augments, transforms, and rearranges information in its unending battle against the affective consequences of negative events…Some of these methods are quite simple (e.g., dismissing as a rule all remarks that begin with 'You drooling imbecile') and some are more complicated (e.g., finding four good reasons why we did not really want to win the lottery in the first place), but taken

[86] Gilbert, et al., "Durability Bias in Affective Forecasting," 295.

> in sum, they seem to constitute a *psychological immune system* whose job it is to protect the individual from an overdose of gloom.[87]

Immune neglect, therefore, is when we fail to take into consideration our existent reserves and innate ability to manage bad situations. When asked to predict how we will feel about a particular future situation, we don't factor in our inherent tools for managing bad situations. The direct clinical correlate is the statement that "I won't be able to live this way," – because of our inability to predict how we will actually feel and respond, statements like this become suspect.[88]

Automated Choice

Taking a different tack (i.e., switching from heuristics governing information processing and perception to how we make choices), Frederick raises the question of whether choice can actually be predicted via the theoretical cognitive models based on rationalistic optimal choice – i.e. models suggesting that human choice is predicated on some utility calculus divorced from affective content. He suggests that one must attend to the actual phenomena of choice in all its cognitive and emotional complexity to address the issue adequately.[89] This is, however, not as easy as it seems. When we face novel situations (Frederick considers the example of someone given a list of statistical information and asked to make a choice), we don't simply have an epiphany and make a choice. Instead, the decision maker must consciously choose what methodology to employ to process the information and to reach a decision. The decision maker is aware of the strategy employed, and can make adjustments to it should the need arise, as any

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[87] Gilbert, et al., "Durability Bias in Affective Forecasting," 296.

[88] This is not meant simply to undercut choices to forgo medical treatment or to doubt a particular patient's preferences. It is, however, an important consideration as to how accurate and comprehensive the patient's predictive abilities are. Is the decision an honest and accurate assessment of future quality of life, or is it an emotionally valenced and focalist reaction which ought to be challenged? This is a very problematic issue in clinical ethics, especially in light of the current avowed avoidance of paternalistic attitudes and desire to incorporate the patient's values in the course of treatment. In a similar vein, there are significant concerns with and reasons to doubt the veracity of advanced directives and living wills – see chapter four.

[89] Shane Frederick, "Automated Choice Heuristics," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 548.

deficiency in the outcome of the methodology chosen is likely to become apparent to the decision-maker. Frederick suggests that this type of cognition employs 'deliberate choice heuristics' – which he characterizes as fundamentally of a different kind than the processes typically considered in the heuristics-and-biases program of Kahneman and Tversky.[90]

Frederick suggests that the fundamental difference between 'deliberate choice heuristics' and 'automatic judgmental heuristics' is a function of the different types of stimuli employed in choice versus judgment tasks. He argues that the choice heuristics are the products of analytic and abstract situations which do not evoke any intuitive elements of cognition, while the judgment heuristics implicitly appeal to intuition: "traditional judgment heuristics are *System 1 heuristics* – they result from cognitive processes that are rapid and not entirely controllable – whereas traditional choice heuristics are *System 2 heuristics* – they result from slower and more deliberate mental processes."[91] Frederick then proposes two types of choice heuristics to demonstrate his model – *choosing by liking* (in which a choice is made based on the option that generates the most favorable response) and *choosing by default* (in which choice is made based on the option that first comes to mind). Initially it is not entirely clear why Frederick refers to his model choice methodologies as 'heuristics'; in light of how we are defining heuristic (fast and frugal, rules of thumb), it seems more likely that these are algorithmic approaches (slow and deliberate). However, he does stress that while these model methodologies may require more processing time, "they are governed by rapid and intuitive processes, they are relatively immune to introspection, and their associated biases may not be recognized by the people who use them."[92]

[90] Frederick, "Automated Choice Heuristics," 548-9.
[91] Frederick, 549.
[92] Frederick, 550.

The first of the choice heuristics concerns decisions informed by affective responses – he argues that ignoring germane emotional responses and attempting an overly cognitive analysis can undercut the validity of the evaluation. In spite of these benefits, affect is not a perfect tool for evaluation, and at times may be severely detrimental in choice, because "(1) it is insufficiently sensitive to quantitative detail; (2) it is unduly influenced by transient contextual cues; (3) it is excessively affected by familiarity."[93] We will return to the issue of affect in judgment at the end of this chapter.

The second of the choice heuristics concerns default settings that may influence our choices – we frequently have general preferences to which we defer in choices, and research has also demonstrated that absent new information, humans in general have a tendency to select defaults (i.e., we tend not to consciously veer from the default unless we have a compelling reason to do so). These defaults tend to have some personal salience, whether through precedent, conspicuous differences with the other options, or some other feature that makes it stand out.[94]

As such, it appears that in addition to the judgment heuristics already discussed, there may also be automatic processes by which we make choices – elements outside our conscious awareness that shape our selection between options. In line with the theme of this chapter, both how we process information and how we make decisions are less homuncular and purely volitional than we initially assumed. Both cognitive functions appear to be heavily influenced by backstage cognition and 'fast and frugal' heuristic processes.

Metaheuristics

Einhorn has proposed that a further distinction can be made between heuristics, the 'rules of thumb' or 'fast and frugal' cognitive structures, and metaheuristics, the rules which create the

[93] Frederick, "Automated Choice Heuristics," 551.
[94] Frederick, 555.

heuristics in the first place. He argues that when we are faced with novel situations, we are initially unsure how to proceed and what actions to undertake – we have no rules of thumb already generated, so we must rely on more fundamental structures – metaheuristics – in order to generate the rules to govern the novel situation. Learning, he argues, is inductive, in the sense that "one experiences specific instances or cases and heuristics are developed to provide some general way to deal with them."[95] We initially make blind guesses and observe the consequences, and formulate explanations based upon our observations. This is almost Humean in its empirical basis – the rules governing novel situations, and the associations that we make based upon them, are all self-generated. He suggests that heuristics like availability, anchoring, representativeness, etc. may in fact be metaheuristics, in that they generate the rules which govern other heuristical approaches to cognition.[96] This allows us to maintain plasticity in cognition and perception – we can adapt to each new piece of information and novel situation.

There is, however, an implicit difficulty in this – because experience both informs us and gives rise to our understanding of the world around us (judgments of the probability of future events based on past experience), we can receive positive feedback for bad heuristics.[97] We can create rules that are irrelevant or fundamentally wrong; the easiest parallel is spontaneous generation – the theory fits the evidence and is reinforced by repeated validation, despite being fundamentally flawed and ultimately false. In essence, the reinforcement we receive through repeated experience is not just a force that works for accurate heuristics and rules; more often than not, our inaccurate judgments of probability get reinforced and correction of our mistakes is infrequent:

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[95] Hillel J. Einhorn, "Learning from Experience and Suboptimal Rules in Decision Making," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 270.
[96] Einhorn, "Learning from Experience and Suboptimal Rules in Decision Making," 271.
[97] Einhorn, 273.

> I do not mean to imply that it is impossible to learn to make well-calibrated probability judgments. If one makes *many* probability judgments in the *same situation*, such as weather forecasters and horse-racing handicappers do, and outcome feedback is quickly received, such conditions may not be outcome irrelevant, and feedback can be self-correcting. However, such conditions would seem to be the exception rather than the rule for most of us.[98]

In short, we frequently are unaware of the inaccuracy of the probabilistic assessments and decisions we make. Much has been said affirming the truth of the heuristics-and-biases model of intuitive judgment, but this is not to say that it is iron-clad or infallible. Significant critiques and alternative theories have been raised, and we will explore the challenges to the heuristics-and-biases approach first.

Challenges

As indicated earlier, there are some critiques of the concept of heuristics – as it represented a new paradigm when it was first introduced, the concept of cognitive heuristics has come under fire from a variety of positions. Several categories of critique have emerged in the literature, and are represented here by the critiques from Schwartz and Gigerenzer. They raise significant conceptual and practical concerns, which are responded to by Gilovich and Griffin.

Schwartz

Schwartz expresses several concerns with the heuristics-and-biases program. He notes questions of the generalizability of the research, questions concerning the actual existence of heuristics, questions about what normative behavior actually entails, and the applicability of probability to individual events. Each of these will be addressed in turn. In addition, Gilovich and Griffin raise responses to each challenge (see below).

The first concern Schwartz raises concerns the widespread criticism in the literature concerning the ability to extend the findings from the laboratory to the real world:

[98] Einhorn, "Learning from Experience and Suboptimal Rules in Decision Making," 276.

> Many well-known writers have questioned the generalizability of the research on judgment heuristics and biases. Some have gone so far as to suggest that the research itself is biased. These critics claim that the problems used by psychologists are specially chosen to produce confusion and that judgment biases are difficult to demonstrate except in contrived laboratory experiments. It is difficult to refute this criticism. Most of the judgment bias research has used highly artificial tasks. Few studies have been conducted in the clinic.[99]

As such, there is a genuine concern about the generalizability of the research – it may be applicable to the artificial environment of the research laboratory, but it may not necessarily be an accurate presentation of how we function in the real world. This is not to say that we don't make mistakes; rather, it is meant to note that the environment that produces the mistakes in research settings is very artificial and confusing for subjects, and as such, we shouldn't be surprised that errors occur as often as they do.

The second concern is whether heuristics and biases actually exist. Schwartz notes that thinkers like Gigerenzer have argued that what are being described as biases are, in actuality, merely "artifacts of an arbitrarily chosen normative theory."[100] We have arbitrarily chosen how people are supposed to act, and therefore when people deviate from this expectation, it is claimed that they have committed some kind of error. If the normative basis is inaccurate, the claim of error or bias is likewise inaccurate.

Questions of normative theory tie into the third objection raised, which addresses the question of what probabilities are and how they ought to be implemented in decision-making. Schwartz notes that even among statisticians there is considerable disagreement on both of these questions.[101] Schwartz notes that there are two competing conceptions of the term probability – pure frequency estimates and Bayesian analysis, which understands probabilities to refer to both degrees of belief as well as frequency. Two distinct concepts of probability leads to the

[99] Schwartz, "Heuristics and Biases in Medical Judgment and Decision Making," 65.
[100] Schwartz, 65.
[101] Schwartz, 65.

possibility of equivocation, logical inconsistency, and consequent confusion when attempting to model human cognition.

Schwartz does note, however, that even if "the theoretical value of the heuristics and biases research program proves to be unclear, its practical value is hard to deny."[102] After all, mistakes in cognition occur – missed or botched diagnoses or surgical procedures, systematic errors in judging the likelihood of outcomes or predicting courses of action, etc. By virtue of the fact that the heuristics-and-biases research program has opened up these systematic errors to exploration and experimentation, the work and effort that has gone into it is justified, so long as we recognize it as a beginning of explanation, and not necessarily its conclusion.[103]

Gigerenzer

Gerd Gigerenzer provides a sustained critique of the heuristics-and-biases program, arguing that they explain both "too little and too much."[104] He argues that they explain too little because we do not know when they work or their mechanism, and too much because one can apply a given heuristic to nearly every experimental result.

The first conceptual critique he offers addresses the normative model Kahneman and Tversky propose, suggesting that their selection of Bayesian models and conjunction, for example, are arbitrary and assumptive. By narrowing attention to a handful of models, they are not looking at cognition as problem-analysis (i.e., how people approach the problem and understand it).[105] In essence, he argues that by asserting that any particular probability model is normative, we are systematically accusing people who conceptualize the problem differently of committing gross cognitive errors.

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[102] Schwartz, "Heuristics and Biases in Medical Judgment and Decision Making," 66.
[103] Schwartz, 66.
[104] Gerd Gigerenzer, "On Narrow Norms and Vague Heuristics: A Reply to Kahneman and Tversky," *Psychological Review* 103, no. 3 (1996): 592.
[105] Gigerenzer, 592.

By choosing a normative model absent context and content analysis, Gigerenzer continues, they are explicitly creating conditions that will *make* people make mistakes, and then claiming that these errors are fundamentally part of cognition. He stresses that people interpret context and problem content in different ways, and therefore, we ought to recognize these differences in cognition as salient and legitimate means of problem solving – for instance, 'probable' means different things to different people.[106] To demonstrate this, Gigerenzer considers a famous example of probability assessment referred to as the 'Linda Problem', in which participants are given a description of a woman which highlights her intellectualism, academic success, and concern for women's issues and social justice. In light of this description, the participant's are asked which is more probable: "Linda is a bank teller" or "Linda is a bank teller and active in the feminist movement". Many participants select the latter option, in light of her intellectual talents and pursuits. However, if one considers conjunction as a normative probability assessment tool, the former option is more likely – to arbitrarily assign numbers and say that the likelihood of either condition individually is 0.5 (from 0 to 1), then the probability of fulfilling the first option (bank teller) is 50%, but the probability of meeting both options (bank teller and feminist) is 0.25 (0.5 x 0.5). Gigerenzer argues that the people selecting option two ought not to be considered as having committed an error – after all, they may have interpreted the context/content differently:

> The phrase T&F can be understood as the conditional 'If Linda is a bank teller, then she is active in the feminist movement.' Note that this interpretation would not concern and therefore could not violate the conjunction rule. Recent studies using paraphrasing and protocols suggest that participants draw a variety of semantic inferences to make sense of the Linda problem: Some 10 to 20% seem to infer that *and* should be read as a conditional, and some 20 to 50% seem to infer that the alternative 'Linda is a bank teller' implies that she is not active in the feminist movement. These semantic inferences can lead to choosing T rather than T&F. Semantic inferences – how one infers the meaning of polysemous terms

[106] Gigerenzer, "On Narrow Norms and Vague Heuristics: A Reply to Kahneman and Tversky," 593.

> such as *probable* from the content of a sentence (or the broader context of communication) in practically no time – are extraordinarily intelligent processes. They are not reasoning fallacies. No computer program, to say nothing of the conjunction rule, has yet mastered this form of intelligence. Significant cognitive processes such as these will be overlooked and even misclassified as 'cognitive illusions' by content-blind norms.[107]

This illustrates the implicit shortcomings of arbitrarily selecting one method of determining the probability of events – there are compelling reasons to suspect that what we understand to be 'errors' may simply be different, but equally rational, methodologies.

The second conceptual concern Gigerenzer raises concerns the vague nature of cognitive heuristics – he compares them to inkblots in which researchers can read into them what they will. He argues that there is an implicit absence of falsifiability in the heuristics-and-biases model – there is no way to demonstrate that they were incorrect or that the research does not support them. Falsifiability is a hallmark of the modern scientific method – the only way to progress in scientific knowledge is to test and retest a hypothesis that is capable of being proven wrong. Gigerenzer argues that neither of these is applicable to the heuristics-and-biases program because "The two major surrogates for modeling cognitive processes have been (a) one-word-labels such as *representativeness* that seem to be traded as explanations and (b) explanation by redescription."[108]

Like Schwartz, Gigerenzer does note the significant effect of the heuristics-and-biases program on stimulating further research, but he suggests that we recognize that what has emerged has been more discordant results than consistent support. As such, he argues that we need clearer models and more consistent results before adopting a new research and modeling paradigm.[109]

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[107] Gigerenzer, "On Narrow Norms and Vague Heuristics: A Reply to Kahneman and Tversky," 593.
[108] Gigerenzer, 593-4.
[109] Gigerenzer, 595.

Response to the critiques: Gilovich and Griffin

Gilovich and Griffin note several recurring objections to heuristics. First and foremost, they note frequent objections falling into a category of responses arguing that 'we're not that dumb':

> The most common critique of the research on heuristics and biases is that it offers and overly pessimistic assessment of the average person's ability to make sound and effective judgments. People by and large manage their lives satisfactorily, something they would be unlikely to accomplish, the argument goes, if their judgments were so prone to bias…This critique owes much of its persuasiveness and appeal to the fanfare that the negative message of heuristics and biases program has generated at the expense of its positive counterpart. There is, of course, some inevitability to this: Negative information typically dominates the positive. Just as there is a 'bad news bias' in media reporting ('if it bleeds, it leads'), it is hardly surprising that the negative message of the heuristics and biases program would capture more attention, inspire more like-minded research, and serve as the focal point of disagreement and controversy.[110]

One ought not to assume that simply because most of the *coverage* of the heuristics and biases research has reflected the negative aspects of our cognition that the *total content* of our cognition is biased or prone to error. The research has drawn attention to this facet of our cognition simply because while we generally are not error prone, we do make mistakes, and we seek the source of those mistakes. By noting these heuristics, we can account for some of the systematic errors we *do* generate.

There are legitimate questions remaining within this train of thought – one can question the ecological validity of the heuristic in question, that is, how well does it translate from abstract concept to actual outcome in the world. Has the research found a genuine cognitive bias, or have we simply found a lab condition in which we make mistakes? Gilovich and Griffin note that research in the heuristics and biases tradition have not necessarily demonstrated ecological

[110] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 8.

validity, but that it is probably high, despite there likely being exceptions.[111] It is clear that we make cognitive mistakes, and if we can demonstrate plausible instances in which cognitive errors occur in the laboratory, it seems likely that we can infer a relationship between the test conditions and manipulations and their ecological correlates. We get along quite well more often than not, but we clearly demonstrate questionable judgment at times, and there must be a source.

Within this category of objections one can find arguments that the mind is innately modular, with specific tools adapted for varying situations. Even within this 'mental toolbox' motif, we still find room for error – we may encounter a novel situation, which requires us to attempt to adapt an existing tool for a new use, which may have varying degrees of success:

> At some times, and in some contexts, tasks are performed by just the right module and sound judgments are made. At other times and in other contexts, however, specific tasks are co-opted by the wrong module and systematically biases judgments are the result. On still other occasions, of course, the mind might not have the right module to handle the problem (no Swiss Army knife does everything an outdoorsman needs done) and so the task is assigned to the 'next best' module, and imperfections in judgment should once again be the result. A modular mind should also produce a pattern of judgments whereby a problem described or structured in one way yields one type of response, whereas the same problem described or structured another way yields a vastly different response – exactly the pattern of results reported countless times in the heuristics and biases literature.[112]

In short, while there is an admitted gap between abstract concept and ecological validity, there is good reason to suspect that heuristics are a good means for accounting for the very real errors we make in cognition.

A second category of objection argues that the results generated by research in heuristics-and-biases are simply curiosities of the laboratory, and indicative of the 'tricky' nature of the testing conditions. These objections assume that cognition outside the laboratory is greatly superior to that within the lab. Gilovich and Griffin note that this type of objection seems to

[111] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 8.
[112] Gilovich and Griffin, 10-11.

ignore that the motivation for the heuristics-and-biases program was the errors typically made outside of the laboratory.[113] There is some validity to the objection, however, in that there is always a concern in experimentation that the research participant will misconstrue what is being tested or the questions asked. Errors can easily result from poor testing conditions, bad questions, etc. – it makes no sense to then interpret the results of experimenter error as a cognitive bias on the part of the research participant. However, this type of critique may be appropriate for a small percentage of the experiments conducted in the heuristics-and-biases program – the great majority have demonstrated the heuristics in question repeatedly in a variety of contexts and with a variety of participants and operating paradigms. While some experiments can be doubted and their results questioned, this objection ought not be raised to the majority of the research conducted.

The third category of objection stems from a belief that those conducting research into heuristics and biases are holding their participants to a standard that is inappropriately high or unreasonable. Gilovich and Griffin note:

> Perhaps people – especially people participating in unfamiliar or otherwise misleading experimental games – make performance mistakes that mask their underlying rational competence, but by definition, human intuition must be rational. This critique usefully points out two aspects of the 'rationality problem.' First, it has a distinct 'Alice in Wonderland' flavor: People can and do define rationality in many contradictory ways. Second, it brings to the fore the crucial role of axioms in justifying a normative theory.[114]

Clearly there is a difference of opinion concerning an appropriate definition of rationality – if our cognition by definition is rational, then clearly there cannot be cognitive distortions or biases. This is problematic, however, in that in addition to doing away with any conception of bias, it potentially does away with our conception of mental illness – after all, the process by which

[113] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 11.
[114] Gilovich and Griffin, 12.

auditory and visual hallucinations are generated are also explicitly 'rational' in this definition, in that they result from distinct cognitive processes, just like the heuristics and biases currently being discussed. It would seem that the resulting definition of rational is therefore unacceptable – unless we define so broadly as to include its own negation (i.e., the irrational becomes rational so long as it is the result of discrete cognitive processes). Further, if we simply reject the axioms of normative theory (those which describe the way individuals ought to behave), we reject notions that have a lot of empirical support and subjective appeal, which is difficult for us to do. As Gilovich and Griffin note, "it is the tension between the general agreement with the abstract rules of probability and the violation of those rules in richer contexts that give the heuristics and biases demonstrations their power.[115]

A fourth category of objection to heuristics and biases suggests that one ought to understand cognition in terms of frequentistic thinking, not probabilistic thinking. That is to say, when we attempt to understand the likelihood of a given event occurring, we examine the frequency of the likely outcomes in order to establish which is more likely to occur. Gilovich and Griffin suggest that this class of objections includes those proposing theories like 'ecological rationality' (a position stemming from evolutionary psychology, see below), in addition to classically frequentist approaches. They note that frequency-based cognitive approaches face numerous concerns, which undercut fundamentally undercut their argument:

> Given the controversy surrounding the normative status of frequencies and subjective probabilities, it is not surprising that those who favor an evolutionary defense of rationality ('ecological rationality') should through in their lot with the frequentists. Evolutionary psychologists maintain that success in our ancestral environment required only a talent for working with frequencies, not probabilities. This argument, precisely because it cannot be tested empirically, remains a matter of faith and ideology. However, the frequentist argument for evolutionary rationality contains a component that can be tested empirically: The evidence for heuristics and biases, it is claimed, 'disappears' when stimuli are presented and

[115] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 13.

> questions are asked in terms of frequencies. This was a bold argument when first introduced and it is even bolder to maintain now when a score of studies have indicated that it simply does not hold up empirically. In fact, presenting frequencies rather than probabilities sometimes makes judgment distinctly worse, sometimes makes judgments distinctly better and quite often leaves the quality of judgment largely unchanged. Even more troublesome for the evolution/frequency argument, Kahneman and Tversky's original explanation of the probability-frequency discrepancy provides a unified account of when frequency formats improve judgments and when they do not.[116]

As such, there is reason to believe that frequency-based assessments of probability are not fundamentally different (superior or inferior) to non-frequency-based assessments of probability.

In light of Gilovich and Griffin's responses, despite the need to connect concept with ecological validity, there is significant conceptual support for a heuristic approach to human cognition. Some controversy remains, and alternatives to this approach have been proposed, to which we will turn shortly. For them moment, it is sufficient to note that a recurring theme in human cognition is a series of avolitional cognitive processes used to take in, analyze, and adapt information quickly and efficiently, which may as a consequence occasionally produce errors in cognition.

Complementary/Supplementary Theories

There are several additional areas of research and theory that are useful for the discussion of heuristics – some speak to the overall structure of cognition (i.e., heuristics as an element of another, larger process) or that offer alternative explanations and valuations of heuristics (i.e., heuristics as evolutionarily advantageous alternatives to algorithmic processes). Each will be addressed in turn.

Dual Processing Models

Dual-process models of cognition suggest the contemporaneous operation of both algorithmic and heuristic modes of cognition. Smith notes that these models are supported by

[116] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 14.

empirical testing, noting specifically "the predictable effects of manipulations that drain cognitive capacity (such as distraction) or increase or diminish motivation to process carefully; these manipulations seem to knock out conscious processing and leave automatic processing relatively unaffected."[117]

There are multiple versions of dual-process theories, positing different interactions and classifications. Gilovich and Griffin, for instance, describe a two system model based on both quick and holistic judgments as well as deliberate and rule-based system:

> An associationist, parallel-processing system ('System 1') that renders quick, holistic judgments is always in operation – not just when motivation is low and judgments are made on the cheap. The assessments made by the associationist system are then supplemented – and sometimes overridden – by the output of a more deliberative, serial, and rule-based system. These models fit the cognitive miser perspective less well because they do not postulate two different 'routes' of information processing that operate in either-or fashion according to the motivation of the information processor (although they too can account for motivational influences through variation in the effort applied to the rule-based system). As we alluded to earlier, the heuristics and biases program has most often been seen through the cognitive miser lens. People are thought to employ various heuristics to save effort. But the idea that heuristics are 'natural assessments' is clearly much more consistent with the two-systems perspective[.][118]

Echoing Gilovich and Griffin's model, Sloman suggests that a two systems model based on associative and rule-based elements accounts for an essential tension we feel when conceptualizing human cognition. It's a general feature of human experience that we encounter an apparent divide between our intuitions and our 'rational' beliefs:

> The tension is revealing because it reflects a gap within our own heads between, on one hand, our intuitions and, on the other hand, those of our beliefs that we consider *rational*. The classic demonstrations often suggest two minds at work: one following the 'natural assessment methods' like representativeness and availability; and the other working to form coherent, justifiable sets of beliefs and plans of action. As Tversky and Kahneman have repeatedly shown, the two minds do not always agree. The distinction between these two minds can be

[117] Smith, "Preconscious Automaticity in a Modular Connectionist System," 197.
[118] Gilovich and Griffin, "Introduction - Heuristics and Biases: Then and Now," 16.

> construed in terms of one of the central puzzles in experimental psychology – whether people are best conceived as parallel processors of information who operate along diffuse associative links, or as analysts who operate by deliberate and sequential manipulation of internal representations.[119]

These two systems interact and can occasionally counter each other. They do not have specific purviews or problem domains – they are both applied to the same situation, and can generate fundamentally different results. Sloman notes that examples of disagreeing responses from the two systems can be found "in every domain of reasoning that has been studied in detail."[120] In short, we always receive feedback from our associative system – after all, it is the first to respond and we cannot simply turn off the process by which we make associations between stimuli – which may be influenced or overruled by our rule-based system.[121]

Why would we need a dual process system of thought? Sloman notes that there are distinct advantages to both:

> Why should human beings need two systems of thought? One answer is that the systems serve complementary functions. The associative system is able to draw on statistical structure, whereas a system that specializes in analysis and abstraction is able to focus on relevant features. A different sort of complementarity is that associative paths that are followed without prejudice can be a source of creativity, whereas more careful and deliberative analyses can provide a logical filter guiding thought to productive ends. Mathematics, law, and (probably) all disciplines demand this combination of creativity and rigorous rule application.[122]

Thus, perhaps having both systems has allowed us to overcome inherent limitations found in only one system cognition.

Dual process theories of cognition have intuitive appeal – we do find ourselves conflicted at times between our hearts and our heads, between our gut instincts and our reasoned logic.

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[119] Steven A. Sloman, "Two Systems of Reasoning," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 379.
[120] Sloman, "Two Systems of Reasoning," 382-3.
[121] Sloman, 391.
[122] Sloman, 395.

Available evidence suggests that we are not simply experiencing disorganized cognition or simply ambivalence – perhaps we are using two different cognitive approaches representing two different yet necessarily interacting and influential systems. At present, two system models of cognition are conjecture, but they certainly have both an intuitive as well as rational appeal. Fundamentally, "The fact that people are pulled in two directions at once suggests two forces pulling."[123]

Bounded Rationality

We initially discussed Simon's work concerning bounded rationality at the outset of the discussion of heuristics. Bounded rationality refers to an approach to cognitive modeling that is predicated on an understanding of the resources of human cognition as finite and limited in scope. Chase et al. note that the classical model of human cognition viewed it as innately rational, a product of probability assessments and logical reasoning – this assumption has been significantly challenged by the heuristics-and-biases program, as noted earlier.[124] Noting that current models still incorporate probability estimates into their assessment of normative human cognition, Chase et al. argue that there are at least three major conceptual problems to be addressed: first, there is no single conception of probability shared by all statisticians and philosophers; second, the normative model adopted by heuristics-and-biases ignores the content and context of the objects of inference; third, the kind of rationality suggested by models paralleling classical rationality place unreasonable expectations upon human cognition.[125] These objections are quite serious, and deserve further explanation.

[123] Sloman, "Two Systems of Reasoning," 396.
[124] Valerie M. Chase, Ralph Hertwig, and Gerd Gigerenzer, "Visions of Rationality," *Trends in Cognitive Science* 2, no. 6 (June 1998): 206.
[125] Chase, Hertwig, and Gigerenzer, "Visions of Rationality," 207.

The first concern – paralleling earlier challenges – concerns whether probability is a principle applicable to individual events or to classes of events. If one adopts a frequentist approach, the probability rules proposed by heuristics-and-biases researchers do not make sense, as they are an arbitrary assignment of probability, instead of a genuine normative standard.[126] The second concern notes the failure of many research experiments to understand that the content and context of the questions they ask can be interpreted in ways that differ from the arbitrarily chosen norms and yet are perfectly reasonable (i.e., content and context can mediate reasonable versus unreasonable approaches). It is entirely possible for an agent to assign different weights to information, and to judge some information to be irrelevant, despite what the rules of Bayesian inference may require (for instance, judging some information as irrelevant may be inappropriately interpreted as base-rate neglect).[127] The third challenge notes that some problems are much more complicated than in experimental conditions, and as a consequence, the kind of modeling proposed (e.g., Bayesian assessments, SEU maximization, etc.) would require more cognitive resources than we actually possess. Quite simply, the kinds of normative models generated by laboratory experiments may not necessarily be tenable in the real world, which clearly parallels earlier discussions of ecological validity. Quite simply, these models require us to be able to do too much – the typical reasoning agent isn't "a supercalculator with unlimited time, knowledge, and computational power."[128] Chase et al. put particular emphasis on this third challenge, and notes that 'rationality' is best understood as a tool for organisms to attain goals, not as conforming to a particular normative model. As a consequence, for a cognitive model to be valid, it must demonstrate ecological validity – it must be attainable by an organism in its

[126] Chase, Hertwig, and Gigerenzer, 207.
[127] Chase, Hertwig, and Gigerenzer, 207.
[128] Chase, Hertwig, and Gigerenzer, 207.

environment within its existing cognitive capacity; hence, rationality must be understood as being bound by the innate capacities of the organism in question.

Chase et al. explore two approaches within bounded rationality. The first is rational analysis, which "entails specifying the goals of the cognitive system, developing a formal model of the environment, and deriving the optimal behavioral function based on the goals, formal model, and minimal cognitive constraints. This function is then compared to human performance, and the model duly refined to bring the two into closer correspondence."[129] While this model has proven to be useful, they note that there are some limitations: it can only be performed when an optimal solution can be discovered, and a tractable model requires significant simplifications of the assumptions of the real-world environment. The second model considered are 'fast and frugal heuristics', which are proposed as specific cognitive tools which have evolved to minimize processing and reaction time to novel stimuli – a feature they refer to as 'ecological rationality', the subject of our next alternative approach to human cognition.[130] We will return to 'fast and frugal' heuristics below.

Ecological Rationality

Organisms develop advantageous cognitive mechanisms in response to environmental stimuli, allowing them to thrive, in contrast to organisms that did not develop these mechanisms:

> These organisms that had a rather difficult time reaching decisions would have been left behind, genetically speaking, by their more fleet-witted conspecifics. As a result, we can expect to see evidence of simple decision-making mechanisms at work in the behavior of modern organisms, including humans. These mechanisms should help individuals make decisions quickly, both by limiting the amount of information they use to select a course of action or choose an available options, and by focusing attention on just that information that will be most useful in making the choice. Thus, these simple evolved inference mechanisms can help organisms overcome one form of the classic *frame problem* plaguing any information-processing decision maker: how to avoid having to consider the

[129] Chase, Hertwig, and Gigerenzer, "Visions of Rationality," 208.
[130] Chase, Hertwig, and Gigerenzer, 209.

> infinite number of possible real-world options and their unlimited future consequences when choosing a course of action.[131]

If we went through each individual possibility and evaluated each and every potential outcome of every environmental stressor or challenge we face, we would quickly find ourselves stuck – disadvantageous in the event of a hostile organism or immediate crisis. As a consequence, we needed faster responses, physical and mental. But this in itself isn't sufficient – what good is an ability to make quick decisions if they are not accurate? If I am asked to quickly perform a problem of arithmetic and immediately declare that the answer is "Belgium", I have provided a response to a stimulus, but not one that is reasonable in light of the information provided. As such, the decision-making processes we would have to evolve would need to be accurate.

This is a classic dilemma: should we adopt an algorithmic process, which is slow but very accurate, or a heuristic process, which is fast but more inaccurate? Todd suggests that we need not compromise – we adapt our fast processes to the demands of the immediate environmental cues:

> Some complexity and structure in decision-making mechanisms can certainly be beneficial in terms of increasing the inferential accuracy of those mechanisms. However, that structure need not lie entirely within the decision maker. Instead, organisms can take advantage of the structure inherent in the decision environment itself. By matching the internal structure of simple decision-making mechanisms to the external structure of information in the environment, inferential accuracy can be achieved without computational complexity. Evolution need not compromise – speed and simplicity can be had at the same time as accuracy. We call this view of adaptive, accurate decisions made with simply, fast, information-frugal and environment-exploiting mechanisms *ecological rationality*.[132]

Fundamentally, ecological rationality explores the cognition of an organism within the specific environmental conditions it faces. The way we approach problems in adverse conditions (e.g.,

[131] Peter M. Todd, "The Ecological Rationality of Mechanisms Evolved to Make Up Minds," *American Behavioral Scientist* 43, no. 6 (2000): 941.
[132] Todd, "The Ecological Rationality of Mechanisms Evolved to Make Up Minds," 941.

scarcity) tends to be different than how we approach problems in beneficial conditions (e.g., abundance). We employ different "tools" depending on the different situations that we face.[133] Todd notes an implicit link between ecological rationality and fast and frugal heuristics, the next approach to cognition to be explored.

Fast and Frugal Heuristics

In contrast to the pessimistic attitude towards cognitive heuristics in the heuristics-and-biases program, some researchers view them as beneficial adaptive tools. As has been demonstrated in this chapter, it is easy to portray heuristics as a means of introducing distortions and errors into cognition. However, if one views these cognitive phenomena from an evolutionary psychology perspective, they demonstrate a significant and beneficial source of knowledge acquisition and interpretation. We have survived to this point because of these heuristics – as such, we should see them as evolutionarily advantageous, rather than as nuisances and sources of error.

The term 'fast and frugal' is easily explained. Gigerenzer et al. note that "We call these heuristics 'fast' because they process information in a relatively simple way, and we call them 'frugal' because they use little information."[134] Demonstrating why 'fast and frugal' heuristics are preferable to the classical rationality model, Todd notes that an agent employing fast and frugal heuristics would be able to make a choice much more readily and with less irrelevant or peripheral information:

> When multiple cues are available for guiding decisions, how can a fast and frugal reasoner proceed? A decision maker following the dictums of traditional rationality would collect all of the available information, weight it appropriately, and combine it optimally before making a choice. A more frugal approach is to

[133] Todd, "The Ecological Rationality of Mechanisms Evolved to Make Up Minds," 942.
[134] Gerd Gigerenzer, Jean Czerlinski, and Laura Martignon, "How Good Are Fast and Frugal Heuristics?" in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 561.

> use a stopping rule that terminates the search for information as soon as enough has been gathered to make a decision.[135]

The fast and frugal model employs a battery of 'one-reason' decision heuristics – decisional patterns that allow an agent to reach a conclusion based on only one particular criterion. He references three of these criteria as indicative of these 'one-reason' heuristics: *Take the Best*, which looks for the decision which most resembles the targeted outcome, *Take the Last*, which uses the most recent successful decisional cue, and *Minimalist*, which selects cues in random order. Each of these one-reason decision heuristics stops the cue search as soon as one is found allowing a choice to be made.[136] These three are not a complete list of the heuristics available to the typical agent – Todd suggests that many more are likely, as we encounter a wide variety of problem domains with corresponding heuristics.

Gigerenzer et al. laud the simplifying nature of heuristics and reject more classical models of rationality (including algorithmic processing and regression models) as overly complex and possessing little ecological validity. Further, they describe several advantages of fast and frugal heuristics over multiple regression analysis (a higher-order decision tool):

> In summary, our fast and frugal heuristics learn with less information, perform fewer computations while learning, look up less information in the test phase, and perform fewer computations when predicting. Nevertheless, fast and frugal heuristics can be almost as accurate as multiple regression when fitting data. Even more counterintuitively, one of these fast and frugal heuristics, Take the Best, was, on average, more accurate than Regression in the more realistic situation in which the training set and test set were not the same (cross-validation).[137]

This finding is quite compelling – we have a quick method of discerning between environmental cues which may prove to be *more* accurate than an algorithmic process. Gigerenzer et al. find this to be significant insofar as it could allow for two completely different approaches to

[135] Todd, "The Ecological Rationality of Mechanisms Evolved to Make Up Minds," 945.
[136] Todd, 946.
[137] Gigerenzer, Czerlinski, and Martignon, "How Good Are Fast and Frugal Heuristics?" 572.

cognition – someone who must make quick decisions may be better off using a heuristic approach, while someone with significant more time and resources may be able to employ a more Bayesian (classical probability) methodology (which Gigerenzer does suggest may be more accurate in these situations).[138]

The debate between Tversky and Kahneman's heuristics-and-biases program and Gigerenzer's fast and frugal heuristics is much more complex than is alluded to in this chapter, and were one to review the literature in greater depth, the great acrimony between the authors would be more evident. It is not the purpose of this chapter to argue which one of these approaches is correct; the 'take home' message is instead that our cognition does not resemble the classical model previously championed. Rather, our cognition is principally accounted for by a variety of spur of the moment decisions, predicated on elements of backstage cognition and automatic processes. Both the heuristics-and-biases research program and the 'fast and frugal' program note that errors can creep into reasoning using these methodologies. For our purposes, the main difference between the two is how we choose to valence these errors – are they systematic or aberrations of an otherwise rational process. Regardless of whether one takes an optimistic or pessimistic view of heuristics, it is important to note that they do not generate 100% accurate decisions, and when used in conjunction with each other, the possibility of error in a particular judgment can be compounded. In a clinical setting, this can have profound consequences, especially in choices of life and death. What we have addressed to this point has been essentially a purely cognitive account of judgment and decision-making. The final section of this chapter addressing the affective component of cognition, noting how emotional valence affects recall, categorization of information, and cognition.

[138] Gigerenzer, Czerlinski, and Martignon, "How Good Are Fast and Frugal Heuristics?" 580.

AFFECT AND RATIONALITY

The last necessary element of discussion is the effect of affect on cognition. In the chapter that follows, we will be exploring the family of depressive disorders, as well as their biological and psychosocial causes. Depression can affect individuals at each of the levels of reductionism discussed in the last chapter. There are physiological correlates to depressive disorders that affect biochemistry (cell-cell interactions, chemical pathway reactions, and other lower-order levels of cognition) as well as psychosocial function (interpersonal relationships, self-image, cognitive processing, and other higher-order levels of cognition). Our focus for the moment, however, is to explore the degree to which affect is comparable to automaticity and heuristic thinking. We will see that affective responses can be developed into automatic reactions, these affective valences can affect current and future cognition, and that when a disease process produces a subjective sense of loss, these affective/cognitive effects can persist for significant periods of time.

Isen & Diamond

We begin with Isen and Diamond, who suggest a bridge between automaticity and affect by noting how affective valences can arise without conscious volition. They suggest that affective responses may function 'automatically' in the sense that they become reflex and avolitional responses:

> One point we wish to explore is the idea that affect, in most instances in which it operates seemingly automatically, like cognitions found to operate automatically, may also involve overlearning and very familiar material…Thus, the same kind of processes that makes cognitive material appear to operate automatically, or without effort and irresistibly, may be responsible for the sense that we sometimes have that feelings take no effort to be felt and are irresistible: Perhaps this occurs when they involve very common, well-learned basic complexes of stimuli, anticipated effects, responses, and outcomes…This analysis would suggest that more frequent, familiar feelings, especially in familiar situations or contexts, would tend to seem more spontaneous and in fact might show characteristics of

> automaticity, but that less familiar feelings, or feelings in unfamiliar situations or contexts, might take more effort or require conscious attention in order to occur and exert influence.[139]

These reflex feelings result in automatic emotional valences for specific situations; as such, we may assign emotional value to situations without necessarily being aware that we have done so. As we will see shortly, these emotional valences are complex, both in their retrieval and how they can influence cognition. Much like cognitive processes, reflexive affect valence processing is the result of overlearning – we associate the same stimuli with the same emotion repeatedly, to the point where we no longer have to consciously make the link between the stimulus and our emotional response. Much in the way that we can drive to work without consciously thinking about every turn, braking or acceleration, so too can we assign emotional valence without consciously thinking about how we feel. As such, Isen and Diamond note that "automaticization may be seen as a gradual process, and automaticity itself as continuous with other kinds of processing, rather than discrete".[140]

There is one final argument Isen and Diamond propose which is necessary to note – automatic affective valence processing is not an inflexible or uncorrectable phenomenon. They suggest that affective processing can be modified and subject to intervention.[141] As such, if the emotional valence assigned to the current situation were to be challenged or corrected, any resulting cognition may be affectively debiased – the agent will be able to overcome implicit distortions of cognition. We will return to this in discussing Schwarz below, and in chapter four with the work of Aaron Beck's Cognitive-Behavioral Therapy.

[139] Isen and Diamond, "Affect and Automaticity," 139.
[140] Isen and Diamond, 147.
[141] Isen and Diamond, 147.

Slovic et al. and the Affect Heuristic

Slovic et al. define affect as "the specific quality of 'goodness' or 'badness' (1) experienced as a feeling state (with or without consciousness) and (2) demarcating a positive or negative quality of a stimulus."[142] This term will take on greater and more explicit meaning in the next chapter, but for the present discussion it will suffice. In essence, affect is the emotional feeling and valence we give to a particular event or situation. Affect encompasses universally recognized emotions like anger, sadness, disgust, anxiety, etc., and, as will be seen, can fundamentally change the way we approach cognition. Slovic continues to note that affect is an automatic feeling – we cannot simply turn on and off the elements of backstage cognition which give rise to an event's or situation's perception and valence, as Isen and Diamond noted above. Because of this automaticity of perception and evaluation, Slovic argues that the experience is indicative of an affect heuristic.[143] This is in direct contrast with a great majority of the heuristics presented and studied, as these are essentially purely cognitive, and ignore emotive elements. Emotion, he notes, is relatively rarely studied as a causal factor in choice and decision-making models. The ironic aspect of this, however, is that emotion is a necessary part of cognition – studies have demonstrated that individuals lacking the ability to give situations emotional valence make worse decisions than individuals combining rational and emotive elements. Citing Antonio Damasio, Slovic notes that individuals with damage to the ventromedial frontal cortices – damage which would not affect their intelligence, memory or ability to think logically but would their ability to valence these cognitive processes with emotional content – exhibit "a form of sociopathy that destroys the individual's ability to make

[142] Paul Slovic, et al., "The Affect Heuristic," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 397.
[143] Slovic, et al., "The Affect Heuristic," 397.

rational decisions; that is, decisions that are in his or her best interests."[144] In short, the kind of abstract and absolute rationality championed by some philosophers ends up being detrimental when divorced from emotive elements. Using this conceptual basis, Slovic suggests that affect may offer the very same type of judgment cue as the cognitively-based heuristics previously discussed. In fact, these emotional cues may be much more readily accessible than the resource-utilization-heavy cognitive heuristics.[145] Because of their comparative ease of access, recalled emotional valence can affect future cognition and evaluation.

Previous work in the development of affective heuristics recognize two precursors – Pratkanis's argument that an 'attitude heuristic' causes the agent to favor or disfavor classes of objects, resulting in approach or avoidance strategies, and Loewenstein et al.'s review of research supporting a 'risks-as-feelings' hypothesis, which suggests that agents' emotional valences differ from their cognitive valences, and that these emotional valences can lead to very different risk-taking behaviors.[146] Both of these models note the significant role emotion can play in decision-making; at times, they can produce behaviors completely at odds with purely cognitive models. This parallels earlier discussions about dual-process systems, which also produce complementary/oppositional behavioral impulses.

Just as with cognitive heuristics, however, there is the potentiality for this recalled valence leading one to error.[147] No cognitive model to date is without critique or potential of compromise; emotional models are subject to the same concerns, and cannot be considered to be the sole basis of cognition or experience:

> However, like other heuristics that provide efficient and generally adaptive responses but occasionally leads us astray, reliance on affect can also deceive us.

[144] Slovic, et al., "The Affect Heuristic," 399.
[145] Slovic, et al., 400.
[146] Slovic, et al., 415.
[147] Slovic, et al., 416.

> Indeed, if it was always optimal to follow our affective and experiential instincts, there would have been no need for the rational/analytic system of thinking to have evolved and become so prominent in human affairs. There are two important ways that experiential thinking misguides us. One results from the deliberate manipulation of our affective reactions by those who wish to control our behaviors. The other results from the natural limitations of the experiential system and the existence of stimuli in our environment that are simply not amenable to valid affective representation.[148]

The possibility of decisional error is compounded when heuristics are compounded. For instance, it is plausible that an agent will face a choice that will trigger cognitive heuristics like availability and/or anchoring, relate that choice to earlier situations which gave rise to emotional valences, and believe that the choice will have emotional effects extending into the future, which requires affective forecasting. Each of these influences is potentially advantageous or deleterious (i.e., potentially accurate or inaccurate). If we are optimistic in estimating advantageous instances and argue that our heuristics are arbitrarily right 90% of the time, the likelihood of all four of the heuristics referenced being error-free is only 65% (0.9 x 0.9 x 0.9 x 0.9). If we were to add more heuristical elements, or we decrease their accuracy, we open up an even greater possibility of judging a situation incorrectly (e.g., a 5% reduction (85%) in the accuracy of the referenced heuristics reduces the likelihood of error-free judgment to 52%; a 10% decrease (80%) reduces the probability of error-free judgment to 41%, and so on). If the decision in question is to forgo medical treatment, we are opening up the decision-making process to potentially lethal errors.

Schwarz

Schwarz argues that the type of cognition we engage in may be heavily influenced by our current affective state. He notes that "a growing body of research indicates that happy moods foster reliance on a top-down, heuristic processing strategy, whereas sad moods foster reliance

[148] Slovic, et al., "The Affect Heuristic," 416.

on a detail-oriented, bottom-up processing strategy."[149] The algorithmic nature of cognition when sad is an important and controversial concept which we will return to in chapter four, when we discuss the question of depressive bias versus depressive realism. For the moment, it will suffice to note that emotion does not simply result in the potential application of new heuristics, but that it can lead to an entirely different cognitive model. Schwarz notes that this shift has been linked to mood-congruent recall of valenced material and feelings as a source of information.[150]

Mood-congruence implicitly suggests two things. First, our memory is enhanced when our affective states at the time of memory-encoding and memory recall match (state-dependent learning) – we form happy memories more readily when we are happy and sad memories more readily when sad. Second, any particular piece of information will be more easily recalled when in a mood-congruent state (mood-congruent memory).[151] Schwarz notes that these two phenomena are more likely to occur for the self-referencing memories; we recall our own states much more readily than those of others. As such, mood-congruent recall affects us selectively; there are times when it does not influence us at all. Instead, we derive emotional valences from our feelings themselves. Schwarz's discussion of mood-congruence differs significantly from Isen and Diamond, who found asynchronous mood congruence (positive affect served as a much more effective recall device than negative affect).[152]

Feelings-as-information is the result of our limited appreciation of our immediate experience – we lack the ability to simultaneously reflect on our current affective state and our

[149] Norbert Schwarz, "Feelings as Information: Moods Influence Judgments and Processing Strategies," in *Heuristics and Biases: The Psychology of Intuitive Judgment*, ed. Thomas Gilovich, Dale Griffin and Daniel Kahneman (New York: Cambridge University Press, 2002), 534.
[150] Schwarz, "Feelings as Information: Moods Influence Judgments and Processing Strategies," 535.
[151] Schwarz, 535.
[152] Isen and Diamond, "Affect and Automaticity," 136.

previously valenced experiences. In short, we lack an ability to distinguish affective responses brought on by earlier experiences from affective responses brought on by the current situation:

> Because we have only one window on our immediate experiences, however, we may mistake feelings due to a preexisting mood state as a reaction to the target, resulting in more positive evaluations under happy than under sad moods. This feelings-as-information assumption generates a number of predictions that cannot be derived from the assumption that mood effects on evaluative judgments are mediated by mood-congruent recall or encoding, of which I address only two.[153]

As such, individuals may simply rely on the feelings they have at that moment as a source of information about their overall attitudes toward the present situation. Feelings-as-information, however, is a correctable phenomenon – by challenging the impressions an agent has about his immediate circumstances, it is possible to discount his reliance upon his emotions as a source of information. This type of discounting, however, does not occur with historically valenced material – we cannot argue that previously negative valenced situations are simply the result of current triggers.[154] In essence, if we feel bad now, we can feel better by blaming our mood on the weather – but we can't do this for depressing memories.

The theoretical model Schwarz proposes suggests that "reliance on one's feelings is particularly likely (1) under conditions in which one's feelings are a highly relevant source of information, and (2) under conditions in which one's feelings allow for the simplification of an otherwise demanding task."[155] It does not seem that much of a stretch to imagine instances when one is considering refusal of life-sustaining treatment to be heavily emotionally valenced (e.g., patients frequently fear dying attached to a machine) and/or potentially simplified by resorting to 'gut reactions' to simplify the decision (e.g., "I don't want this"). Schwarz also notes that more algorithmic processes may incorporate mood effects in judgment. When individuals have

[153] Schwarz, "Feelings as Information: Moods Influence Judgments and Processing Strategies," 536.
[154] Schwarz, 537.
[155] Schwarz, 538-9.

sufficient time and energy to engage in more thorough decision-making processes, their present mood state will aid them in recalling mood-congruent memories, which can then influence or alter their perception of their current situation.[156] Further, he argues that emotions do not necessarily exert influence only on the immediate situation – strong emotions may result in residual mood states, which may generalize to other events, related or unrelated. In short, strongly valenced emotions can exert short-term and long-term effects, both of which can affect cognition and perception. If the reasons for these valences are challenged, they may abate, which may prevent some skew or bias from affecting the agent's perception and cognition. In a clinical context, this would necessitate exposing underlying disorders potentially affecting cognition (e.g., depression), its source (e.g., reaction to the diagnosis), and challenging the thought process leading to the emotional valence (e.g., addressing possible heuristical thought or bias):

> As this selective review indicates, our feelings can have a pronounced impact on judgment and decision making. Depending on conditions, they may influence which information comes to mind and is considered in forming a judgment, or serve as a source of information in their own right. The use of one's feeling as a source of information is particularly likely when the feelings are relevant to the judgment at hand or allow the judge to simplify the task by relying on a "How-do-I-feel-about-it?" heuristic. When our feelings do, in fact, reflect our actual affective reaction to the target, this heuristic does not result in undue biases. Yet, it is often difficult to distinguish between one's reactions to the target and one's preexisting mood state. In this case, reliance on the "How-do-I-feel-about-it?" heuristic results in systematic biases, as reviewed previously. However, individuals do not rely on this heuristic when the informational value of their feelings is called into question.[157]

Obviously, our goal as clinicians is to make sure that our patients make decisions that are as accurate in both their cognitive content and personal authenticity as possible; the prevention of

[156] Schwarz, "Feelings as Information: Moods Influence Judgments and Processing Strategies," 539.
[157] Schwarz, 546.

heuristic biases or errors in cognition is especially critical in the kinds of life and death decision-making considered in this dissertation.

A final area of concern for Schwarz is how the effect of understanding emotional valence affects conceptions of rationality. Like Slovic et al., he notes that there are serious questions raised if one attempts to divorce cognition from emotion – a growing body of research demonstrates what Damasio noted: ignoring affective information undermines judgment.[158] However, the solution is not simply to attend to the emotional valences – the issue becomes complex precisely because we have difficulty disentangling the emotions we feel from a particular environment or stressor from the long-term effects of past strongly emotionally-valenced experiences. As such, we must be aware of our emotions, we must understand how they can both help and hurt our decision-making capacity, and we must challenge emotional content when it seems to exert undue influence. In chapter four, we will return to this concept, as there is literature suggesting that not all forms of depression result in unrealistic or skewed perception and interpretation.

Tait and Silver

What kinds of events can provide the kind of emotional keystones referenced in affect heuristics and emotional influences on cognition? Tait and Silver have argued that major life events can have significant repercussive influence; this almost appears to be a truism, but their research further argues that how the event is experienced shapes its emotional valence[159] – a key element when discussing emotional influences on cognition. In fact, negative life events can frequently intrude on normal cognitive experience – we are forced to think about them repeatedly and involuntarily, a pathological form of memory referred to as rumination. They note that this

[158] Schwarz, "Feelings as Information: Moods Influence Judgments and Processing Strategies," 547.
[159] Rosemay Tait and Rozane Cohen Silver, "Coming to Terms with Major Negative Life Events," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh (New York: Guilford Press, 1989), 352.

rumination is a stress response, and "the experience of these ruminations tends to be correlated positively with the degree of reported stress and with levels of negative affect, and inversely related to indices of positive emotion."[160]

Coping with these emotional responses and ruminations is a critical part of recovery from the event or stressor. Negative life events can impact our lives in a variety of ways, short-term and long-term. The means by which we cope with them initially and work through them as part of the healing process have profound effects on our ability to function later in life. Tait and Silver note that so long as the agent has not found a way to work through negative life events, these events will remain salient to his experience and result in counterfactual rumination. Until we can work through the negative life event, we run the risk of being mired in "What if…?" thinking, instead of being able to move on with our lives. They further suggest that finding meaning in the negative event plays a key, and potentially pathological, role in recovery from the event – if the agent has on-going difficulty finding meaning for a given event, "the search may persist for extended time periods, contributing to ongoing cognitive and emotional involvement in the event. A persistent search for meaning has been found to be inversely related to psychological recovery and positively related to the occurrence of involuntary, intrusive, and distressing event-related ruminations."[161] In short, we seek meaning, even at the expense of our emotional and cognitive well-being. In the context of medical decision-making, it is quite likely that an pessimistic diagnosis and prognosis represent instances of these kind of rumination-producing events, in the sense as they can be experienced as a form of loss:

> The experience of loss may represent a significant common denominator among the major negative events described by respondents in our research. This loss may be literal (e.g., the loss of a particular person, environment, role, or relationship) and/or symbolic (e.g., the loss of future possibilities, cherished

[160] Tait and Silver, "Coming to Terms with Major Negative Life Events," 352.
[161] Tait and Silver, 355.

> hopes, goals, or plans). Our basic emphasis is on the meaning of events, situations, or responses, and represents a symbolic interactionist approach. From this perspective, any event that threatens or violates important models or theories of self, others, or the world may represent a loss. Moreover, dissonance between the meaning of experience and central models or theories of reality may pose a significant threat or loss, insofar as they undermine the predictability of one's situational and/or social experience.[162]

If the patient experiences this subjective experience of the loss of one's hopes or abilities, it is quite likely that they will engage in the kind of rumination currently discussed, with all of its concomitant influence on cognition and perception.

As a final note on Tait and Silver, it is necessary to contrast their findings with our earlier discussion of affective forecasting and the durability bias. It is clear that elements of Tait and Silver conflict with what we have already established concerning our ability to predict how we will feel in the future, in light of our present predicament. We may address this by noting that affective forecasting requires us to predict our emotional state in the *future*, which may be inappropriately negatively biased by present circumstances and ignorant of many types of common coping skills we naturally employ as part of our psychological immune system. Tait and Silver's argument, on the other hand, opens up the possibility that *present* circumstances may influence our *present* choices, and *possibly* may extend into future cognition.

What emerges from the literature is a sense that rationality is not a purely cognitive process – affect is a critical part of our thought process, and our rationality may be limited or undercut by removing affective valence. However, this emotional valence heuristic is subject to the same kinds of error that cognitive heuristics encounter – our emotions can aide or hamper the choices we make. As such, we must be cognizant of the impact emotional responses and coping skills have on our ability to weigh information and choose to undergo or forgo medical interventions. In the next chapter, we will explore the family of depressive disorders and five

[162] Tait and Silver, "Coming to Terms with Major Negative Life Events," 373.

common medical conditions that can give rise to comorbid depression. This comorbidity is frequently undiagnosed, meaning that patients have made choices that may have been unduly influenced by a depressive disorder.

CHAPTER THREE: HIDDEN COMORBIDITIES: DIAGNOSTIC CRITERIA OF DEPRESSIVE DISORDERS AND THEIR PREVALENCE IN FIVE COMMON MEDICAL CONDITIONS

The previous chapters have laid out arguments that cognition is a deterministic process mediated by conscious challenges. They have concerned themselves with compelling philosophical and psychological theories and experimentation, and have set the stage for discussions of clinical influences on cognition, the basis of this chapter. What will be explored now are a family of disorders that are characterized by their affective components, as well as potential influences on cognition and behavior. They influence our cognition at all levels of organization from biochemical to psychosocial. They will provide the basis for the chapter to follow, which explores the proposed effects on judgment that result from the disorders contained herein. In framing this discussion, we will see that depressive disorders are quite prevalent and frequently underdiagnosed. In light of their potential to influence cognition, especially in emotionally taxing situations like diagnosis with a profoundly life-changing condition, we will see the danger they pose to patient's making accurate and authentic treatment decisions.

There are a variety of causes of depressive disorders; factors causing depression are not limited to matters of psychiatric illness, but can be the result of sociological, medical, hormonal, or pharmacological factors.[1] This chapter will offer a summary of seven types of depressive disorders, and their etiologies. The descriptions of the disorders will follow the models listed in the *Diagnostic and Statistical Manual, Fourth Edition, Text Revision* (DSM-IV-TR)[2] and the *International Statistical Classification of Diseases and Related Health Problems, 10th Edition*

[1] American Medical Association, *Essential Guide to Depression* (New York: Pocket Books, 1998); Harold I. Kaplan and Benjamin J. Sadock, *Synopsis of Psychiatry, Eighth Edition* (Baltimore: Lippincott, Williams, & Wilkins, 1998); Maurice Victor and Allan H. Ropper, *Principles of Neurology*, *Seventh Edition* (New York: McGraw-Hill Medical Publishing Division, 2001).

[2] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, *Fourth Edition*, Text Revision, edited by Michael B. First, Fourth (Washington, D.C.: American Psychiatric Association, 2000).

(ICD-10).[3] Case studies will be presented for each, in order to clarify the unique nature of the depressive disorder and offer examples of how it may manifest clinically. Further, common therapies for each will be discussed, including psychopharmacology, talk therapies, and alternative therapies.

Medical versus psychiatric diagnosis

Before discussing the disorders themselves, it is useful briefly to consider the differences in diagnosis in psychiatry versus diagnosis in house medicine. The multi-axial system of psychiatric diagnosis offers a significantly different means of assessment than traditional medicine. Medical etiologies and diagnoses tend to be linear – while systemic dysfunctions may occur concomitantly, they are essentially grouped under one heading in the medical history – type of diagnosis is not germane or distinctive.[4] Essential hypertension will be grouped with chronic obstructive pulmonary disease, decubitus, and peripheral neuropathy. Psychiatric diagnosis is much more structured – disorders are placed into a multi-axial system which clearly delineates what can be treated on an inpatient basis versus what requires alternative modalities of therapy, as well as overall assessments of the patient's psychosocial functioning.

There are five axes in psychiatric diagnosis. Axis I encompasses the patient's clinically treatable psychiatric disorders, such as chronic paranoid schizophrenia, major depressive disorder, bipolar disorder, and other disorders which normally respond readily to psychiatric medication. For example, Sarah presents to the psychiatric emergency room; her behavior is hyperverbal and her mood and affect are labile. She relates a personal history which

[3] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, *Tenth Edition* (Geneva: World Health Organization, 1992).

[4] This statement will no doubt elicit some confusion – the author is not arguing that there is no real distinction made in medical diagnosis, but that the means of presentation of the medical history will group all medical dysfunctions together. Psychiatric diagnosis will make explicit differences in the types of disorder present, as they refer to conditions that are clinically treatable, those which are not clinically treatable, conditions falling within the two, etc.

demonstrates a cycling history of periods of extreme energy, reckless behavior, sexual promiscuity, impulsive spending, and euphoria with periods of lethargy, despair, disinterest in the affairs of her daily life, and isolation. She has no history of self-injurious behavior. The pattern that emerges is not related to any apparent social cause, but just seems to happen naturally. The psychiatrist makes a provisional diagnosis on Axis I of bipolar disorder. Sarah admits herself voluntarily for several days, and the attending psychiatrist puts her on a lithium regimen (a mood stabilizer). After several days, Sarah's behavior is more in control as a result of a structured environment and stabilized lithium levels. She is discharged with a prescription for lithium and outpatient therapy for medication management. Future medical histories will note an Axis I diagnosis of bipolar disorder.

There are, however, psychiatric conditions which do not respond readily to medication or inpatient treatment; some are not treatable at all, or require extended "talk therapy" to affect behavior change. These disorders are listed on Axis II, which encompasses personality disorders and mental retardation. There is no medication at present which can treat mental retardation or the primary effects of a personality disorder. At present, we cannot treat personality disorders with a pill – there is no anti-narcissism capsule or anti-histrionic drip to administer. While medication can be used to address secondary symptoms (for example, an anti-depressant could be used to treat the depressive symptoms of a borderline patient or an anti-anxiety medication could be used to calm an obsessive-compulsive), it cannot correct the deeper issues, which deal with either the patient's mental handicap or outlook on interpersonal interaction, self-image, or ability to assess the consequences of actions.[5] These deeper issues can lead to overt behavioral

[5] For example, the issues underlying borderline personality disorder tend to be a lack of object constancy, poor self-image, an undefined or absent sense of self, a desire for punishment, a need to constantly test and retest relationships, a concomitant desire for intimacy/dependence with a conflicting desire for independence, etc. The issues underlying histrionic personality disorder tend to be a persistent belief that relationships are much more

problems – suicidal gestures, attention seeking behavior, violence, etc., which are causes for hospitalization.

A very brief example would be Steven, who was admitted to a psychiatric inpatient unit for treatment of suicidal ideation with a plan – he had cut checkerboard patterns into his forearms after a fight with his girlfriend. The wounds were superficial, but they looked ugly. Steven stated that he had done it in front of her to punish her (he believed that she was planning to break up with him in favor of someone else). Steven stated that he wanted her to feel guilty for making him hurt himself, and by making her feel guilty he would also force her to stay in the relationship. Steven reported that he had had a difficult childhood with no real parental figures – his father had left and his mother had been involved in a series of codependent and abusive relationships afterwards. Steven disclosed that he had a history of admissions to psychiatric facilities in other states – he had been admitted a total of thirteen times over the course of the past five years, all for similar self-injurious behavior. Steven disclosed that all of his "suicide attempts" were done in public places or where friends could find him quickly. He stated that when he suffered, he wanted other people to suffer, too. This pattern of behavior and history of hospitalization led the psychiatrist to make a provisional diagnosis of borderline personality disorder. Steven suffered from an unstable self-image; he felt the need to continuously attract attention to himself and to test and retest the stability of his existent relationships. His "suicidal" gestures were simply efforts to see if his friends were still his friends. This lack of object constancy was the underlying pathology for his past and present admissions.[6] After prescribing

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intimate than they actually are, an abnormal desire to attract attention to oneself, a tendency to define oneself through the eyes of others, etc. These symptoms are not neurochemical in origin – they are cognitive processes by which the person judges relationships, their own self-worth, etc. There is no pill that can correct pathology with this etiology (unlike the anti-psychotic medications used to treat delusional behavior in a schizophrenic, for example).

[6] Jerold J. Kreisman and Hal Straus, *I Hate You, Don't Leave Me: Understanding the Borderline Personality* (New York: Avon, 1991).

antidepressants, the psychiatrist arranged for long-term cognitive-behavioral therapy to address Steven's psychosocial attitudes – by correcting his lack of object constancy and the belief structures with which he approached relationships, it was hoped that future hospitalizations could be avoided. Steven would gain greater insight into healthy relationships and a better self-image, which would prevent future suicidality.

Axis III contains all of the concomitant medical disorders a patient may be experiencing – many articles have underscored the link between physical and psychological dysfunction.[7] As the crux of psychiatric diagnosis is psychosocial stressors, clearly any current medical illness would affect the cognitive process or mood of the patient at some level. Indeed, insight into concomitant medical illness (and education and treatment thereof) can affect secondary psychiatric conditions. The link between physiological dysfunction and affective disorders will be explored in greater depth below.

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[7] T. Chow and J.L. Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 31-52; Lydia Chwastiak, et al., "Depressive Symptoms and Severity of Illness in Multiple Sclerosis: Epidemiologic Study of a Large Community Sample," *The American Journal of Psychiatry* 159, no. 11 (November 2002): 1862-68; C.A. Class, L. Schneider, and M.R. Farlow, "Optimal Management of Behavioural Disorders Associated with Dementia," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 17-29; T.J.M. Cleophas, "Depression and Myocardial Infarction: Implications for Medical Prognosis and Options for Treatment," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 77-85; G.A. Fava and N. Sonino, "Diagnostic and Therapeutic Aspects of Depression Associated with Medical Illness," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 1-16; Y. Gustafson, et al., "Post-Stroke Depression," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 63-75; P.J. McGrath, E.V. Nunes, and F.M. Quitkin, "Treatment of Depression in Alcohol-Dependent Patients: Current Concepts," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 93-104; A. Ronson and D. Razavi, "Affective and Anxiety Disorders in Patients with Cancer: Optimal Management," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 113-28; S.P. Roose and E. Spatz, "Treating Depression in Patients with Ischaemic Heart Disease: Which Agents Are Best to Use and to Avoid?" in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 87-92; T.F. Scott and C. Chieffe, "Treatment of Affective Disorders in Patients with Multiple Sclerosis," in *Depression Associated with Medical Illness*, ed. Katherine Palmer (Hong Kong: Adis International, 2000), 105-11; Victor and Ropper, *Principles of Neurology*; Rebecca Elliott, "The Neuropsychological Profile in Primary Depression," in *Cognitive Deficits in Brain Disorders*, ed. John E. Harrison and Adrian M. Owen (London: Martin Dunitz, 2002), 273-94; John B. Murray, "Depression in Parkinson's Disease," *The Journal of Psychology* 130, no. 6 (November 1996): 659-67.

Axis IV concerns a listing and overall assessment of psychosocial stressors, rating them at none, mild, moderate, or severe. This rating contains and considers financial problems, familial stressors, psychosocial difficulties, and any intercurrent medical problems. For example, if a patient presents to the psychiatric emergency room with depression following the loss of a job and the break-up of a marriage, the psychiatric dysfunction would be categorized under Axis I, and the job loss and broken marriage would be contained in a "moderate" or "severe" note on Axis IV.

Axis V represents the patient's global assessment of function – a rating from 0 to 100 which encompasses a total assessment of the patient's ability to function, ability to handle psychosocial stressors, ability to care for him- or herself, etc. Many hospitals have admissions criteria that require a specific GAF score before psychiatric inpatient admission is considered (e.g., patients with GAF scores over 35 presenting with a psychiatric emergency would be referred to an outpatient clinic or private practice psychotherapist).

To piece this all together, we can look at the case of Jeffrey, who presents to the psychiatric emergency room stating that he's giving serious thought to jumping off a bridge. During the course of his intake interview, he is rude and condescending towards the staff, states that he's the most important patient they have, states that he's feeling depressed due to a death in the family, has a depressed mood with a labile affect, has no previous self-injurious behavior, and cannot contract for safety.[8] A lot of information has been presented, but it can fit quite readily into the above diagnostic schema. Axis I would carry a differential diagnosis of major depressive disorder, with bipolar disorder and dysthymic disorder to be considered. This stems from the Jeffrey's expressed suicidality and statements of feeling depressed. Axis II would carry

[8] When a patient 'contracts for safety', he or she states that they will not engage in self-harming behavior (e.g., attempt suicide).

a diagnosis of Personality Disorder, NOS (Not otherwise specified), with notes to consider Narcissistic Personality Disorder and Borderline Personality Disorder. This is based on the grandiose statement "I'm the most important patient you have" as well as Jeffrey's attitude towards the staff. Axis III would be left empty or marked "Deferred" as there is no previous medical history. Axis IV would carry a note of Moderate or Severe, due to the death in the family. While there is a certain subjectivity to the assessment of Jeffrey's Global Assessment of Function, it is safe to say that it would fall into the range set by the care facility for inpatient treatment (35 or lower), especially in light of his inability to contract for his own safety were he to be discharged.

This is not to suggest that there is always precision in psychiatric diagnosis. Quite frequently an Axis II disorder manifests itself as an Axis I disorder. Borderline personality disorder, for example, can and has frequently been mistaken for bipolar disorder or episodic depression. Further, unless a patient is floridly psychotic or neurotic, diagnosis tends to be blurry until behavior can be observed for extended periods. Additional complications arise in that the ease with which a patient can manufacture illness is much greater in psychiatry than in house medicine. While it is possible to mimic the signs and symptoms of a heart or kidney dysfunction, tests can disprove claims of inadequate function or damage. My claims of a heart condition can be verified or disproved by a transesophageal echocardiogram, magnetic resonance imaging, etc. Psychiatric dysfunction, however, is more difficult to prove or disprove.

To a certain extent, psychiatric illness is a matter of neurochemistry and physiology – specific neurotransmitters in excess or in absence can cause corresponding changes in mood and perception, and can be measured by laboratory tests and other diagnostics. If a patient is expressing depressive symptomology, the body can be checked for physical causes and

verification. If I feel down because of hypothyroidism, I can receive pharmacotherapy for a physical problem (a more complete listing of physical and pharmacological causes of depression can be found later in this chapter). However, there is no real lab test for psychosocial causes of mood disorders. There is no enzyme for marital difficulties, no hormone for unemployment, and no neurotransmitter for the death of a loved one. Consequently, there is no means to test whether a patient is malingering[9] or is presenting with a factitious disorder.[10] When these types of activities are suspected, other metrics must be employed to discern how much of the patient's presentation is genuine psychopathology and how much is a manufactured "illness". The Minnesota Multiphasic Personality Inventory, for example, is the most common resource used in clinical and correctional settings for the detection of malingering.[11]

DEPRESSIVE DISORDERS

The DSM-IV-TR notes a four-way split in the division of the depressive disorders. It makes a distinction between the unipolar depressive disorders (e.g., Major Depressive Disorder, Dysthymic Disorder, etc.), the bipolar disorders (e.g., Bipolar I Disorder, Bipolar II Disorder, Cyclothymia, etc.), depressive disorders secondary to medical illnesses (e.g., depression

[9] The DSM describes malingering as "the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution or obtaining drugs." American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 739.

[10] The DSM describes factitious disorder as being "characterized by physical or psychological symptoms that are intentionally produced or feigned in order to assume the sick role. The judgment that a particular symptom is intentionally produced is made both by direct evidence and by excluding other causes of the symptom." American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 513.

[11] See, for example, R.M. Bagby, R. Rogers, and T. Buis, "Detecting Malingered and Defensive Responding on the MMPI-2 in a Forensic Inpatient Sample," *Journal of Personality Assessment* 62, no. 2 (April 1994): 191-203, C.B. Gacano, et al., "A Clinical Investigation of Malingering and Psychopathy in Hospitalized Insanity Acquittees," *Bulleting of the American Academy of Psychiatry and the Law* 23, no. 3 (1995): 387-97, G.L. Iverson and L.M. Binder, "Detecting Exaggertion and Malingering in Neuropsychological Assessment," *Journal of Head Trauma Rehabilitation* 15, no. 2 (April 2000): 829-58, J.L. Lewis, A.M. Simcox, and D.T. Berry, "Screening for Feigned Psychiatric Symptoms in a Forensic Sample by Using the MMPI-2 and the Structured Inventory of Malingered Symptomatology," *Psychological Assessment* 14, no. 2 (June 2002): 170-76, and E.A. Wise, "Relationships of Personality Disorders with MMPI-2 Malingering, Defensiveness, and Inconsistent Response Scales Among Forensic Examinees," *Psychological Report* 90, no. 3 pt 1 (June 2002): 760-66.

secondary to neurological disorders like stroke or Huntington's chorea or medical disorders like hypothyroidism), and substance-induced mood disorders (e.g., depressive reactions to alcohol, prescription medications, etc.).[12] This dissertation concerns itself with the cognitive aspects of three of these four categories – unipolar, bipolar, and secondary to medical illness. Substance-induced mood disorders, while prevalent, are significantly more transient than the first three categories, and tend, in this author's experience, to resolve themselves much more frequently than other forms of endogenous/exogenous depressions.

This four-way split is necessary due to the wide differential diagnosis for depressive disorders – depressions can be caused by exogenous factors (e.g., loss of a job, divorce, death of a loved one, etc.), endogenous factors (e.g., monoamine dysregulation, chronically elevated cortisol levels, etc.), medical factors (e.g., hypothyroidism, myasthenia gravis, cerebral vascular accident, etc.), and legal/illegal substances (e.g., alcohol, illicit drugs, abuse of prescription medication, etc.).

Epidemiology of affective disorders

The World Health Organization in their landmark study *The Global Burden of Disease* noted that unipolar depression (ICD-10 classification; "Major Depressive Disorder" in the DSM-IV-TR) is the second most prevalent cause of disability in industrialized nations – behind only ischemic heart disease.[13] The National Institute of Mental Health notes concurs, noting that "Major depression is the leading cause of disability in the U.S. and worldwide."[14] In fact, it is estimated that 18+% of the population will experience a mood disorder in their lifetimes. Given

[12] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 345

[13] World Health Organization, *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020.*, edited by Christopher J.L. Murray and Alan D. Lopez, Global Burden of Disease and Injury, vol. 1 (Cambridge: Harvard University Press, 1996).

[14] National Institute of Mental Health, *The Invisible Disease: Depression* (Washington, D.C.: National Institute of Mental Health, 2001), p. 1

the rapid change and extreme psychosocial stressors faced by most members of society, this number is not surprising. It is tied to a variety of stressors; economic concerns, psychosocial conflicts, health problems, and family crises all contribute to the depressive epidemic.

The prevalence of depressive disorders in society varies dependent upon what is being examined. The disease rate is dependent upon whether we are examining the general population, endogenous depressions, or depressions secondary to other conditions. Murray and Lopez estimated that in the year 1990, established market economies saw approximately 18,499,000 new cases of depression, or approximately 2,319 people affected per 100,000.[15] Globally, these numbers are significantly higher, with 109,486,000 new cases in 1990, despite a lower overall incidence of 2,079 per 100,000 people.[16] In addition to the 18% lifetime prevalence noted by the World Health Organization, the National Institute of Mental Health indicates that *in any given year*, about 9.5% of the American adult population is affected by a depressive disorder.[17] The statistics are telling, and there is a genuine concern that the number of depressive disorders is actually being underestimated. Pliska notes that depressive disorders are not necessarily limited to the adult population – while not as common as anxiety disorders, depressive disorders can be found in childhood. He offers a more conservative estimate than the World Health Organization, noting that while the risk of developing a depressive or anxiety disorder increases significantly by adulthood, the lifetime rate of depressive and anxiety disorders is approximately 10% of the

[15] John B. Murray and Alan D. Lopez, *Global Health Statistics: A Compendium of Incidence, Prevalence and Mortality Estimates for Over 200 Conditions*, Global Burden of Disease and Injury Series, vol. 2 (Cambridge: Harvard University Press, 1996), p. 601.
[16] Murray and Lopez, *Global Health Statistics: A Compendium of Incidence, Prevalence and Mortality Estimates for Over 200 Conditions*, p. 603.
[17] National Institute of Mental Health, *The Invisible Disease: Depression*, p. 1

adult population. [18] This risk varies with gender; he notes that "10-25% of women and 5-12% of men will suffer an episode of major depressive disorder (MDD) at some point in their lives."[19]

Peruzzi, et al., have noted that primary physicians frequently have difficulty recognizing the symptomology of depressive disorders, and Plovin, et al., have noted that "difficulties in diagnosing affective disorders create ambiguity in ascertaining base rates in the population."[20,21] As such, we are reliant upon predictive studies and correlational studies to estimate the actual numbers of depressed patients. Green suggests that the primary symptoms of a depressive disorder may even be mistaken as an underlying medical illness, which can complicate diagnosis and treatment.[22] Victor and Ropper note that it is much more likely that primary care physicians will be the first to encounter a depressed patient, and that they are more likely to misdiagnose the depressive disorder as one of myriad somatic and psychosocial difficulties:

> Depressive states are so often associated with obscure physical symptoms that they are more likely to come to the attention of general physicians and internists than are other psychiatric entities. Moreover, they are frequently misdiagnosed, the symptoms being mistakenly attributed to anemia, low blood pressure, hypothyroidism, migraine, tension headaches, a chronic pain syndrome, chronic infection, emotional problems, worry, and stress.[23]

As noted above, there are a variety of physical conditions that can give rise to clinical depression – it is chilling to consider the vast numbers of patients experiencing depression who are being treated for physical complaints while their emotional issues remain unresolved.

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[18] Steven R. Pliszka, *Neuroscience for the Mental Health Clinician* (New York: Guilford Press, 2003), p. 130
[19] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 200
[20] Nico Peruzzi, Andrew Canapary, and Bruce Bongar, "Physician-Assisted Suicide: The Role of Mental Health Professionals," *Ethics & Behavior* 6, no. 4 (1996): 353-66.
[21] Robert Plomin, J.C. DeFries, and G.E. McClearn, *Behavioral Genetics* (New York: W. H. Freeman and Company, 1990), p. 378
[22] Stephen A. Green, "Supportive Psychological Care of the Medically Ill: A Synthesis of the Biopsychosocial Approach in Medical Care," in *Human Behavior: An Introduction for Medical Students*, ed. Alan Stoudemire (New York: Lippincott-Raven Publishers, 1998), 495-514, p. 506
[23] Victor and Ropper, *Principles of Neurology*, p. 1608

There are a variety of depressive disorders to consider, and each presents different epidemiological rates and health concerns. The DSM-IV-TR notes the following depressive disorders: Major Depressive Disorder (single episode and recurrent, with several modifying features [e.g., psychotic, melancholic, etc.]), Dysthymic Disorder, Depressive Disorder NOS, Bipolar I/II Disorders, Cyclothymic Disorder, Bipolar Disorder NOS, Mood Disorder Due to a General Medical Condition, Substance-Induced Mood Disorder, Adjustment Disorder, and Mood Disorder NOS; Minor Depression is listed in an appendix under research conditions (i.e., there was insufficient evidence to warrant inclusion as a definitive diagnosis, but criteria are proposed that would enable future study and possible inclusion in future editions of the DSM).

Severity of the Symptoms

Both the DSM-IV-TR and the ICD-10 note that there is a clinically relevant difference in the possible presentations of the family of depressive disorders. As such, they both divide the major disorders into categories of mild, moderate, and severe symptoms. The DSM notes that a classification of 'mild' involves those conditions in which more than half of the criteria for a given diagnosis are met, as well as requiring that the presenting symptoms cause either a mild disability or require more effort to achieve normal function.[24] The ICD-10, which lists similar symptomology for the relevant conditions but groups them differently, notes that a mild condition would present with two or three of the diagnostic criteria, but should be able to continue to function relatively normally with most activities.[25] In contrast to this, a classification of a 'severe' condition would require most of the diagnostic criteria and "clear-cut, observable disability (e.g., inability to work or care for children)."[26] The ICD-10 again parallels this

[24] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 412
[25] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 336
[26] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 412

distinction, noting that most of the diagnostic symptoms are present and distressing to the patient, an in the case of depression, may be accompanied by feelings of worthlessness and guilt, as well as possible somatic complaints and suicidal ideation.[27] 'Moderate' cases of these conditions fit in between these two extremes.

Major Depressive Disorder

There are explicit diagnostic features of a major depressive disorder. Both the DSM-IV-TR and the ICD-10 note that the depressive illnesses are syndromal – the patient can exhibit a range of behaviors that fit into the spectrum of depressive illness. The ICD-10 categorizes depressive episodes as containing worsened mood, decreased energy, and decreased activity. The patient has a decreased capacity for enjoyment, exhibits general disinterest, and inability to concentrate. The patient typically experiences decreased sleep and appetite, and frequently has excessive feelings of guilt or worthlessness. These periods extend for several days, and tend to remain at the same level throughout the episode.[28] Major depressive disorder is syndromal – patients can present with a variety of symptoms. In general, patients must present with at least five out of nine symptoms, including a depressed mood, loss of interest or pleasure, significant weight loss or gain not explained by diet, insomnia or hypersomnia nearly every day, psychomotor agitation or retardation nearly every day (observable by others), fatigue or anergia nearly every day, feelings of worthlessness or inappropriate guilt nearly every day, diminished cognitive abilities, or recurrent thoughts of death. Either the depressed mood or the loss of interest or pleasure must occur, and the overall symptoms must occur more often than not for at least two weeks. These symptoms must not be better accounted for by a mixed episode (see below), must cause a significant impairment in functioning, must not be the result of a medical

[27] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 336
[28] World Health Organization, p. 335

condition (see below) or substance use, and the symptoms must not be better accounted for by bereavement.[29]

The DSM notes that major depression is associated with a high mortality rate – the suicide rate has been estimated at up to 15%; the death rate in MDD appears to be linked to age as well, with a "fourfold increase in death rates in individuals with Major Depressive Disorder who are over age 55 years."[30] In general medical settings, MDD is also associated with increased pain and physical illness, as well as diminished physical and social functioning.[31] Age appears to affect the course of the illness and its presentation; the DSM notes that atypical depression tends to be more prevalent in younger patients, while depression with melancholic features is more common in elderly patients (see below); advanced age is also associated with worse response to treatment.[32] The epidemiological rates for MDD are about a 15% lifetime risk in the general population, with approximately 6%-8% of patients in care settings experiencing depression.[33] As will be noted later, this rate of comorbid depression will vary significantly depending upon the medical illness in question, and the initial cause of depression appears to be multifactorial, taking in genetic risks, family history, biochemical dysfunction, physical and psychological stress, and other psychosocial factors. Depression tends to have an onset around age 28, and is characterized by repeated episodes of depression (there is a significant risk of future episodes following an initial depressive illness).[34] There seems to be a genetic predisposition to the development of depressive disorders; the literature suggests that the

[29] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 356
[30] *DSM*, p. 371
[31] *DSM*, p. 371
[32] *DSM*, p. 372
[33] "Neurologic Disorders," in *Diseases*, ed. Joanne M. Bartelmo, et al. (Springhouse: Springhouse Corporation, 2001), 716-81, p. 50
[34] Eric R. Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders," in *Principles of Neural Science*, ed. Eric R. Kandel, James H. Schwartz and Thomas M. Jessell (New York: McGraw-Hill Health Professions Division, 2000), 1209-26, p. 1210

condition can run in families, with first-degree relatives of individuals experiencing depression at 1.5 to 3 times the rate of the general population.[35] Pliska notes that first-degree relatives of individuals experiencing major depression have an 8-17% chance of developing depression as well.[36] Without active treatment for depression, the illness tends to last 4-12 months.[37]

Minor depression

The DSM-IV-TR offers some diagnostic criteria for minor depression, but suggests that it needs more research before being included in the canon of psychiatric diagnosis, while the ICD-10 includes it. The DSM suggests that the requisite features of minor depression is one or more periods of depressive symptomology of the same duration as major depression, but without all of the same features (i.e., the same diagnostic criteria are used, but an episode of minor depression does not have as many presenting symptoms as an episode of major depression).[38] These symptoms may or may nor impair social and occupational functioning, but they will require the person to exert more effort to remain at normal levels of function.[39] The specific epidemiology is unknown, but the suspicion is that it is a relatively common phenomenon, especially in primary medical care and behavioral health settings; there is some suggestion that it is associated with several primary medical conditions.[40] It appears that there are age correlates in the epidemiology of minor depression. Lavretsky and Kumar note that there is some consensus that the prevalence of minor depression changes with age, peaking when the individual is in her 30s, falling off, then peaking again in her 80s.[41] They also suggest that minor depression is especially prevalent in acute and long-term care facilities, in greater numbers than major depression (up to

[35] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 373
[36] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 202-3
[37] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1210
[38] *DSM*, p. 775
[39] *DSM*, p. 775
[40] *DSM*, p. 775
[41] Helen Lavretsky and Anand Kumar, "Clinically Significant Nonmajor Geriatric Depression," *Psychiatric Services* 54, no. 3 (March 2003): 297-99, p. 298

50% in long-term care, up to 25% in primary care, and up to 70% in the institutionalized elderly).[42]

Dysthymia

The ICD-10 defines dysthymia as a persistent depressive mood of several years duration which is not severe enough to warrant diagnosis as a mild, moderate, or severe depressive disorder.[43] The DSM-IV-TR suggests more explicit criteria, stating that the depressed state must last at least for at least two years (one year for children), have no symptom-free periods lasting more than two months, and include at least two of the following symptoms: "poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, and feelings of hopelessness."[44] Individuals with dysthymia tend to simply become used to their symptoms, and tend to state that this is "normal for them" – in fact, they tend not to report these symptoms unless they are directly asked.[45] After carrying a diagnosis of dysthymia for at least two years, the individual can also experience episodes of major depression, periods which are described as "double depression."[46] The lifetime prevalence of dysthymia has been estimated at 6%, with a point prevalence of 3%.[47] Dysthymia frequently begins at an early age, with an insidious onset and chronic duration. Most individuals will seek help for a depressive episode superimposed on a dysthymic state, rather than seeking treatment for primary dysthymia. Spontaneous remission occurs, but the DSM notes that "evidence suggests the outcome is significantly better with active treatment."[48] Dysthymia tends to be

[42] Lavretsky and Kumar, "Clinically Significant Nonmajor Geriatric Depression.", p. 298
[43] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 338
[44] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 377
[45] *DSM*, p. 377
[46] *DSM*, p. 377
[47] *DSM*, p. 379
[48] *DSM*, p. 379

more frequent in close biological relatives of individuals with a major depressive disorder than in the general population.[49]

Seasonal Affective Disorder

As the name suggests, seasonal affective disorder is linked with seasonal patterns. In addition to the diagnostic criteria for a major depressive episode, the DSM-IV-TR notes that it's "essential feature is the onset and remission of Major Depressive Episodes at characteristic times of the year. In most cases, the episodes begin in fall or winter and remit in spring."[50] Many have suggested that the cause of this disorder is levels of sunlight – in the winter, the sun rises later and sets earlier – and credence has been given to this theory by the efficacy of light therapy (the individual with seasonal affective disorder is exposed to bright light for an extended period, and generally reports an elevation in mood thereafter). While an individual may possess a predisposition towards depressive episodes, to carry a diagnosis of seasonal affective disorder, the seasonal episodes of depression must significantly outnumber the off-season episodes.[51] The DSM further notes that women tend to comprise the SAD patient base (60-90%), but it is unclear whether gender is a specific risk factor.[52] The type of depression manifested is also in question – some research has suggested that less severe forms of depression are also seasonal (i.e., there may be a seasonal pattern for depressions other than a major depressive episode).[53]

Atypical Depression

Atypical depression denotes a period of depression with signs less commonly associated with depression. In a typical major depressive disorder, the patient may present with an inability to sleep, loss of appetite with weight loss, general dysthymic disposition, feelings of guilt, apathy

[49] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 379
[50] *DSM*, p. 425-6. There are atypical forms of SAD that result in summer depressions as well.
[51] *DSM*, p. 425-6
[52] *DSM*, p. 426
[53] *DSM*, p. 426

etc. Some of these symptoms may also present in a case of atypical depression, but the patient may present with hypersomnia, a leaden paralysis, or increased appetite with weight gain. The individual also tends to exhibit some uncommon psychosocial symptomology, including mood reactivity and extreme sensitivity to perceived interpersonal rejection.[54] The DSM defines mood reactivity as the "capacity to be cheered up when presented with positive events,"[55] which can mean feeling less sad to feeling happy (euthymic), even for extended periods of time (so long as the outside stimuli remain positive). "Leaden paralysis" refers to feeling like one's body (generally one's arms or legs) is heavy or weighted down for at least an hour.[56] Perceived rejection sensitivity is not tied specifically to any one period in the depressive episode, but tends to be manifested more often during depressive periods. The individual tends to respond to perceived rejection with maladaptive behavior, like substance use, leaving work early, etc.,[57] and as a result of a desire to avoid rejection, the individual may avoid relationships altogether. Atypical depression tends to manifest at an earlier age than normal depression (many patients report a change in mood beginning in high school), and tends to have a more chronic course (unlike the more acute major depressive disorder).[58]

Mixed State

The major diagnostic features of a mixed episode involve meeting the diagnostic criteria for both manic and depressive episodes in rapid succession, but without meeting the minimum duration (i.e., more than a week but less than two weeks). The mood disturbance must cause the patient in social and occupational functioning or demonstrate a danger to oneself or others (with potential psychotic features), and the symptoms must not be due to a general medical condition

[54] *DSM*, p. 420-1
[55] *DSM*, p. 420-1
[56] *DSM*, p. 420-1
[57] *DSM*, p. 420-1
[58] *DSM*, p. 421

or substance use.[59] Mixed episodes can last up to several months; periods of remission can occur with little to no symptoms, or the episode can develop into a major depression.[60] The DSM suggests that mixed episodes tend to appear more often in younger or elderly patients (over age 60) and may preferentially affect men more than women.[61] Laboratory findings in mixed episodes tend to parallel the findings in major depression.[62]

Bipolar Disorder I

The DSM-IV-TR suggests the clinical criteria of Bipolar I Disorder as multiple occurrences of Manic or Mixed Episodes, with an occasional episode of depression.[63] There is a significant risk of suicide in individuals with Bipolar I Disorder, frequently occurring in a depressive episode (estimated at 10%-15%), as well as a risk of violent behavior towards others in severe manic or psychotic episodes.[64] There appear to be some physiological correlates, in that imaging studies have shown a higher rate of right-hemisphere or bilateral lesions in the frontal lobe.[65] The age of onset tends to be about 20 years old, and the DSM notes that first episodes after age 40 suggest the symptoms may be explained by a comorbid medical condition like hypothyroidism.[66] There does not appear to be a race or gender difference in the incidence of Bipolar I Disorder, but there does seem to be a difference in the initial clinical presentation. Men tend to present first with manic episodes, while women tend to present first with depressive

[59] *DSM*, p. 365
[60] *DSM*, p. 363
[61] *DSM*, p. 363
[62] *DSM*, p. 363
[63] *DSM*, p. 382. The DSM requires that these episodes not be substance-induced, not be due to a medical condition, and not be better accounted for by a psychotic disorder.
[64] *DSM*, p. 384
[65] *DSM*, p. 384-5. See also R. Joseph, "Frontal Lobe Psychopathology: Mania, Depression, Confabulation, Catatonia, Perseveration, Obsessive Compulsions, and Schizophrenia," *Psychiatry* 62, no. 2 (Summer 1999): 138-72.
[66] *DSM*, p. 385

episodes.[67] This gender differentiation seems to carry over into the course of the illness – men tend to have more manic episodes, while women tend to have more depressive episodes.[68] The DSM estimates the lifetime prevalence of Bipolar I Disorder in community samples at 0.4%-1.6%.[69] The illness is chronic, and many individuals experience multiple episodes of mania or depression, sometimes with psychotic features. Epidemiological evidence and twin studies suggest a greater incidence of bipolar disorder and major depressive disorder in first-degree biological relatives of individuals with Bipolar I disorder than in the general population (Bipolar I Disorder: 4%-24%; Bipolar II Disorder: 1%-5%; Major Depressive Disorder: 4%-24%).[70] There are a variety of treatments for bipolar disorders; treatment tends to involve the use of lithium or anti-psychotic medication. For a compelling narrative on bipolar disorder, Kay Redfield Jamison's *An Unquiet Mind* offers a marvelous personal account of her experience with the illness.[71]

Bipolar Disorder II

The DSM-IV-TR suggests criteria for Bipolar II Disorder as multiple episodes of major depression accompanied by at least one hypomanic episode.[72] In light of the above discussion of Bipolar I Disorder, it should be clear that Bipolar I accounts for mood lability that is essentially "up" while Bipolar II Disorder accounts for mood lability that is essentially "down." These recurrent down periods with occasional euthymia must not occur with an incidence of genuine mania or a mixed episode, must not be better accounted for by a psychotic disorder, must cause significant impairment in daily life, must not be due to substance-use, and must not be due to a

[67] *DSM*, p. 385
[68] *DSM*, p. 385
[69] *DSM*, p. 385
[70] *DSM*, p. 386
[71] Kay Redfield Jamison, *An Unquiet Mind* (New York: Vintage Books, 1995).
[72] *DSM*, p. 393

general medical condition, as these would warrant other diagnoses.[73] Suicide is a genuine risk with these patients – the DSM notes successful suicide in approximately 10%-15% of patients with Bipolar II Disorder.[74] The age of onset for Bipolar II Disorder is similar to that for Bipolar I Disorder (around age 20); likewise, if a clinician notices late-onset Bipolar II, she should suspect the possibility of an underlying medical condition (e.g., thyroid dysfunction) or substance use.[75] There appears to be a gender difference in the epidemiology of Bipolar II Disorder, with women more frequently carrying the diagnosis.[76] Further, there appear to be gender differences in the course of the illness, with men experiencing more hypomanic episodes and women experiencing more depressive episodes.[77] Overall, the DSM suggests that the lifetime prevalence of Bipolar II Disorder is approximately 0.5%,[78] and there appears to be a greater risk for Bipolar II Disorder in first-degree biological relatives of individuals with the disorder than for other individuals in the general population.[79]

Cyclothymia

The ICD-10 defines cyclothymia as a chronic lability of mood with numerous periods of alternating dysthymia and hypomania, neither of which is severe enough to warrant diagnosis of a bipolar disorder or recurrent depression.[80] Cyclothymia is frequently found in the relatives of individuals with bipolar disorder. The DSM-IV-TR suggests more specific criteria, including: multiple periods of hypomanic and dysthymic symptoms, symptom-free episodes that do not last

[73] *DSM*, p. 393
[74] *DSM*, p. 394
[75] *DSM*, p. 394
[76] *DSM*, p. 394-5. There are some concerns with this statement – for example, it has been suggested by epidemiologists that men are more likely to view depression as a character flaw rather than as an illness, and as such, the numbers may be misrepresentative.
[77] *DSM*, p. 394-5
[78] *DSM*, p. 395
[79] *DSM*, p. 395
[80] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 338

longer than two months (in the two-year period necessary for the diagnosis), no full-blown depressive, manic, or mixed episodes, symptoms that are not better accounted for by a psychotic disorder, the symptoms are not substance induced, and the presence of a notable impairment of functioning as a result of the illness.[81] In addition to the symptoms discussed above, there may also be a greater prevalence of substance-related disorders and sleep-continuity disturbances in individuals with cyclothymia.[82] Cyclothymia tends to begin early in life, and may represent a predisposition to the development of other mood disorders (especially bipolar disorder).[83] There seems to be no real gender difference in the incidence of cyclothymia, but women seem to come for treatment more often than men.[84] The lifetime prevalence of cyclothymia is 0.4% to 1%, while the prevalence in mood disorder clinics is between 3%-5%.[85] As in other depressive conditions, there appears to be a greater prevalence of other affective disorders in first degree biological relatives of individuals with cyclothymia than in the general population.[86]

Adjustment Disorder

The DSM notes another type of disorder that can result in depressive symptomology. "Adjustment disorder" refers to a "psychological response to an identifiable stressor or stressors that results in the development of clinically significant emotional or behavioral symptoms."[87] The specific criteria for an Adjustment Disorder include onset within 3 months of the occurrence of the stressor, marked distress or impairment in the subject, insufficient symptomology to meet the criteria for other Axis I disorders, symptoms not better accounted for by bereavement, and

[81] *DSM*, p. 398
[82] *DSM*, p. 398
[83] *DSM*, p. 399
[84] *DSM*, p. 398-9
[85] *DSM*, p. 399
[86] *DSM*, p. 399
[87] *DSM*, p. 679

abatement of the symptoms within six months of the stressor.[88] What constitutes a stressor can vary from person to person – stressors can come from every sphere of a person's life, from the environment he lives in to the termination of employment to a medical illness. The adjustment disorder puts the individual at serious risk for self-harm, as well as increasing the severity of comorbid medical illness.[89] The disorder appears to be twice as prevalent in women as in men, and the overall prevalence varies with specific target populations: between 2-8% in community samples of children/adolescents and the elderly, up to 12% in general hospital inpatients, in 10-30% of outpatient mental health patients, and up to 50% in specific medically compromised patients (e.g., following heart surgery).[90]

Other Conditions/Research Conditions

The DSM notes two other categories of interest – Depressive Disorders Not Otherwise Specified (NOS) and Depression with Melancholic Features. Depressive Disorder NOS is a general catch-all category for all disorders with depressive features not better accounted for by "the criteria for Major Depressive Disorder, Dysthymic Disorder, Adjustment Disorder with Depressed Mood, or Adjustment Disorder With Mixed Anxiety and Depressed Mood."[91] This category includes premenstrual dysphoria, minor depressive disorder (see above discussion), recurrent brief depressive disorder, post-psychotic depressive disorder (occurs during the residual phase of schizophrenia), major depression superimposed on delusional, psychotic, or schizophrenic disorders, and"[s]ituations in which the clinician has concluded that a depressive disorder is present but is unable to determine whether it is primary, due to a general medical

[88] *DSM*, p. 683
[89] *DSM*, p. 680-1
[90] *DSM*, p. 681
[91] *DSM*, p. 381-2

condition, or substance induced."[92] Depression with melancholic features is a complete loss of interest or pleasure in almost all activities or pleasurable stimuli.[93] The essential criteria for this disorder is the maintenance of a depressed state even when something desirable happens, as well as at least three additional criteria like "a distinct quality of the depressed mood, depression that is regularly worse in the morning, early morning awakening, psychomotor retardation or agitation, significant anorexia or weight loss, or excessive or inappropriate guilt."[94] This disorder is considered in cases when there is a marked difference between the current state and a normal depressed state; severe depression of longer duration is not distinct in its quality, and therefore would not be considered to have melancholic features.[95] These individuals tend to have clear precipitants to the episode, tend not to have premorbid Personality disorders, and may respond to placebo medication.[96]

In addition to the qualified categories of depressive illnesses, there are a variety of other conditions that are being researched. These proposed categories were not included in the diagnostic categories of the DSM-IV-TR because there was "insufficient information to warrant inclusion of these proposals as official categories or axes."[97] Included in this group are a discussion of an alternative criterion for dysthymic disorder, symptoms for the proposed recurrent brief depressive disorder and mixed anxiety-depressive disorder, and criteria for a depressive personality disorder. As these are research criteria for conditions, only a brief description of each will be offered.

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[92] *DSM*, p. 381-2
[93] *DSM*, p. 419
[94] *DSM*, p. 419
[95] *DSM*, p. 419
[96] *DSM*, p. 419
[97] *DSM*, p. 759. I am including them for reference to indicate directions of future research. For further information, please see the DSM-IV-TR and the DSM-V Research Agenda.

As has been already presented, dysthymic disorder is a pervasive dysphoric mood of at least two years duration. Criterion B normally requires that at least two common symptoms of major depression be present during this two year period (e.g., appetite changes, sleep disturbances, anergia, etc.). What has been proposed instead is the concomitant presence of at least *three* diagnostic criteria, including feelings of low self-esteem, pessimism or despair, general loss of interest in activities or pleasure, social withdrawal, chronic fatigue, persistent guilt or brooding, irritability or excessive anger, decreased activity or productivity, or difficulty with cognitive functioning (memory, concentration, decisiveness).[98]

Recurrent Brief Depressive Disorder has been suggested as a complement to Major Depressive Disorder. The principle difference is that the patient does not meet the time duration of MDD – Recurrent Brief Depressive Disorder lasts for at least two days but less than two weeks, and must last for several days. These episodes must recur at least once a month for twelve consecutive months, and must not be tied to the menstrual cycle.[99]

Mixed Anxiety-Depressive Disorder requires a persistent or recurrent dysphoric mood that lasts for at least one month. In addition to dysphoric mood, the patient must experience at least four additional criteria, including cognitive difficulties (e.g., in concentration or memory), sleep disturbances, fatigue, irritability, worry, hypervigilance, pessimism, low self-esteem, or feelings of worthlessness. These symptoms must be severe enough to interfere with everyday functioning. The DSM notes that current prevalence rate is about 0.8% in community samples, and 1.3%-2% in primary care.[100]

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[98] *DSM*, p. 774-5

[99] *DSM*, p. 778. There are also exclusionary signs, such as the individual not carrying a prior medical history significant for depression, etc. For further information, please see the DSM-IV-TR and the DSM-V Research Agenda.

[100] *DSM*, p. 780. There are also exclusionary signs, such as the individual not carrying a prior medical history significant for depression, etc. For further information, please see the DSM-IV-TR and the DSM-V Research Agenda.

The DSM finally discusses Depressive Personality Disorder as a possible future diagnosis. These individuals do not demonstrate any acute pathology, but rather tend to display persistent negative feelings like "dejection, gloominess, cheerlessness, joylessness, and unhappiness."[101] These individuals tend to exhibit an inability to have fun, are overly serious, tend to worry and ruminate on their negative thoughts. There tend to be feelings of inadequacy, a lack of hope for the future, low self-esteem, and chronic pessimism towards the future.[102] The DSM notes that these symptoms must not occur exclusively during periods of major depression, and must not be better accounted for by Dysthymic Disorder (the DSM notes that there may be considerable overlap between Depressive Personality Disorder and other depressive disorders); further, this disorder may appear more frequently in close relatives of an individual with major depression.[103]

The ICD-10 offers other diagnostic criteria for depressive disorders. Mood disorders, as defined by the ICD-10, contain fundamental changes in affect to a state of depression without or without associated anxiety. It notes that the mood change results in an overall decrease in activity, tends to be recurrent, and may respond to environmental triggers.[104] Persistent affective disorders refer to conditions that result in frequent affective change, but not to a degree severe enough to be labeled hypomanic or dysthymic. They last for many years, and may involve comorbid single episodes of genuine depressive or manic episodes.[105] Recurrent depressive disorder is characterized by multiple instances of depression without any manic episodes; hypomania may occasionally occur following a depressed episode (possibly caused by

[101] *DSM*, p. 788
[102] *DSM*, p. 788
[103] *DSM*, p. 788-9
[104] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 332
[105] *ICD-10*, p. 338

antidepressant treatment). These recurrent episodes can begin at any stage of life with either an acute or insidious onset, and can last from weeks to months.[106]

Etiologies

The National Institute of Mental Health suggests that diagnoses of major depression tend to fit into three categories: "major depressive disorder, dysthymic disorder, and bipolar disorder (manic-depressive illness)."[107] As has been noted above, there is a rather large grouping of disorders which fit into the depressive model.

Victor and Ropper note that there is currently some question regarding the essential nature of a depressive disorder. Specifically, the question is asked whether it is a disease state (a neurological dysfunction; the kraepelinian concept) or a psychological reaction (like grief or frustration; the meyerian concept). They argue that both can be right:

> These two disparate concepts are not irreconcilable. An eclectic position is that both are correct – i.e., that there are two basic forms of depression: exogenous and endogenous. *Exogenous (or reactive) depressions* have an overt external cause, such as the loss of a loved one, loss of one's fortune or position, or a disabling or life-threatening illness. In this framework, *grief* would exemplify a reactive or exogenous depression. In contrast, the *endogenous depressions* have no apparent external cause; they seem to occur in susceptible individuals as a response to some unknown biologic alteration.[108]

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[106] World Health Organization, *International Statistical Classification of Diseases and Related Health Problems*, p. 336-7

[107] National Institute of Mental Health, *The Invisible Disease: Depression*, p. 1. This is, of course, a simplification of a much more complex picture, but it serves as an overall summary – it suffices to note that this split is generally made in terms of acute vs. chronic onset, as well as the severity of the symptoms. Major depression and bipolar disorder have an acute onset with severe symptoms (generally seen in a matter of one or two weeks), while dysthymic disorder (dysthymia) requires a significantly longer period to diagnosis with less severe symptoms (at least two years in adults).There are other concerns regarding this generalization. Bipolar disorder is chronic – the current standard of practice involves pharmacological regulation of lithium levels throughout the patient's lifetime, and as such, bears more resemblance to the chronicity of dysthymia. However, the bipolar crises – i.e., those which require hospitalization – are acute (seen by this author much more frequently in the manic phase of the illness than in the depressive phase). As noted above, there is an entire family of depressive disorders; this dissertation does not follow the NIMH split.

[108] Victor and Ropper, *Principles of Neurology*, p. 1608

Psychosocial Factors

While it is a truism to state that environmental crises can affect us profoundly, empirical research has demonstrated that there are correlational links between specific environmental factors and resultant conditions. Pliska notes that:

> It is clear that adverse life events contribute to the development of mood and anxiety disorders. Persons who were sexually or physically abused during childhood are up to four times more likely to develop major depression or commit suicide; child abuse is associated with an earlier age of onset of depression and greater chronicity of the depression. Other factors known to be related to affective disorders are early parental loss and nonspecific life stressors. As expected from heritability figures, these environmental stressors play a larger role in unipolar depression than in bipolar disorder.[109]

Not all depressions are endogenous – there is a correlation between patterns of life events and the onset of depression and other affective disorders. Nemeroff notes that the specific interaction of the prevailing hypotheses regarding depressive etiology cannot really account for the complex interplay of the genetic, monoamine, and hormonal causes of depression.[110] He suggests that a more workable model would involve a combination of endogenous and exogenous factors, and that the "stress-diasthesis" model offers a better explanation for the prevalence of depressive disorders.[111] The stress-diasthesis model (environmental experience and inborn predisposition, respectively) suggests that there is likely an inborn quality that predisposes an individual to develop a given affective disorder, but that this alone is insufficient to trigger the onset of the condition. Rather, there must be an environmental factor which serves as the catalyst for the disorder. This would seem to offer support for twin-study data in which only one twin develops the disorder being studied – it is plausible that the affected twin may have been exposed to an environmental influence that the other did not experience (e.g., troubled marriage, abuse, trauma,

[109] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 206
[110] Charles B. Nemeroff, "The Neurobiology of Depression," in *The Scientific American Book of the Brain* (Guilford: The Lyons Press, 1999), 263-75, p. 272
[111] Nemeroff, "The Neurobiology of Depression.", p. 272

etc.). Nemeroff notes that there are cases of depressive disorder that do not fit the stress-diasthesis model; there are individuals who may have a genetic predisposition and yet never develop the condition in question, just as there may be people who do not have the predisposition and yet develop a depressive disorder.[112] Despite these aberrant cases, the model does offer a compelling explanation for depressive illness, and parallels examples from the medical model (e.g., predispositions to cancer, heart disease, diabetes, etc.).

Reductionism

There are a variety of possible etiologies of depression – psychosocial, biochemical, neuroanatomical, endocrinologic, genetic, medical, etc. Biochemical causes can involve dysregulation of one or more neurotransmitters or neuropeptides.[113] Neuroanatomical causes can include the misfiring, underdevelopment, or hyperactivity of several communicative systems, structural changes due to age or illness, or neurological trauma.[114] Endocrinologic causes can include dysregulation of myriad hormones, from growth hormone to corticotrophin-releasing hormone to thyroid-stimulating hormone, as well as suppression or abnormal responses in other hormones like prolactin or dexamethasone.[115] Genetic causes can include familial predisposition to develop an affective illness, awaiting only the correct trigger to instigate the disorder. Medical causes are myriad – as has been noted already, depression can result from, be exacerbated by, or be masked by a variety of medical conditions from metabolic disturbances to infections to gastrointestinal and genitourinary disorders. [116] None of this, however, should be taken to suggest that there is one common factor or cause.[117] Rather, it should be taken as a

[112] Nemeroff, "The Neurobiology of Depression.", p. 274
[113] *DSM*, p. 353
[114] Lavretsky and Kumar, "Clinically Significant Nonmajor Geriatric Depression.", p. 298
[115] *DSM*, p. 353
[116] "Neurologic Disorders.", p. 50;
[117] Lavretsky and Kumar, "Clinically Significant Nonmajor Geriatric Depression.", p. 298; American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, p. 353

general caution that depression is multifactorial, and the primary care physician should take care to note the subtleties of psychiatric diagnosis. Psychosocial causes of depression are fairly well known and obvious; while there are exceptions to this rule in claims of depression from bizarre or counterintuitive stimuli,[118] it should not be controversial to suggest that there is little difficulty in spotting exogenous causes of depression. What follows in the next few sections concerns endogenous depressive illnesses – concerning first reductive accounts of depression, followed by discussions of several medical disorders common in house medicine that carry a noteworthy risk of depression.

Genetic

Both Kandel and Victor and Ropper note that there is compelling evidence that depressive illness is not simply environmental – heritability studies have demonstrated that the incidence of mood disorders among biological relatives of adopted children with unipolar or bipolar depression is higher than that of biological relatives of adopted normal children.[119] Several authors have addressed the genetic aspect of depressive disorders, and the general consensus is that there is not simply one particular gene that is causative of depressive illness; rather, what is more likely is that predisposition to affective disorders (and, as some argue, all psychiatric disorders) is polygenetic.[120] Genetic predisposition towards depressive illness has been approached through a variety of means – monozygotic/dizygotic twin studies and chromosome/allele linkages have been explored, as well as stress-diasthesis considerations (genetic predisposition triggered by environmental factors). There is still significant debate

[118] In the psychiatric emergency room, this author interviewed and assessed patients who stated that they were depressed because they spent all of their money on crack cocaine; patients that were depressed because they were served macaroni and cheese for dinner; adult patients of normal intellectual function who were depressed because their mother did not buy them an action figure as a housewarming gift, etc.

[119] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212; Victor and Ropper, *Principles of Neurology*, p. 1616

[120] Nemeroff, "The Neurobiology of Depression.", p. 266; Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 134

concerning which genes are the likely culprits; research is currently directed at a handful of the chromosomes regulating human growth and development. Linkages have been proposed on chromosomes 4, 12, 13, 18 (in two places), 21, 22, and the X chromosome; thus far, the strongest evidence seems to implicate linkages on chromosomes 18 and 22. [121] Pliska notes that such suggestions ought to be taken with some caveats – these reports are preliminary, and they do not indicate that a link with a specific psychiatric disorder has been identified. Further, he notes, some conditions may be linked to mitochondrial DNA.[122] As such, the specific genetic loci of mental illnesses are still in the process of being identified.

Twin studies

Twin studies have proven to be very useful tools in ruling out environmental influences in the development of physical and mental disorders. In essence, twin studies have suggested that while environmental factors can be an influence in the onset of particular disorders, there is a specific genetic component or predisposition for the development of an illness (e.g., schizophrenia, bipolar disorder, etc.).[123] Monozygotic twins have identical DNA, and as such, can provide a model of behavior and development of identical genes in differing settings; they offer a greater degree of control and reliability than dizygotic twins, who have different genes. There is some dispute concerning the actual rate of monozygotic concordance. Kandel argues that there is a 40-60% rate of concordant affective illness in monozygotic twins reared apart, and that the concordance rate of bipolar disorder in monozygotic twins could reach 80%, versus 10% in dizygotic twins.[124] Victor and Ropper suggest that in bipolar disorder, monozygotic twins exhibit a concordance rate of 72% versus 14% concordance in dizygotic twins; for unipolar

[121] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 203
[122] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 141
[123] Nemeroff, "The Neurobiology of Depression.", p. 266
[124] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212

depression, monozygotic twins are concordant for the illness 40% of the time, while dizygotic twins are concordant 11% of the time.[125] Pliska suggests that the bipolar concordance rate in monozygotic twins is 58-70%, versus 16-24% in dizygotic twins. Overall, he suggests that bipolar disorder has a heritability of 0.8-0.9, versus 0.45 for unipolar depression.[126]

Stress-diasthesis

Genetic predisposition ought not to be interpreted as inevitability, however. Modern medicine has demonstrated that a variety of gene-linked diseases do not have to manifest, simply because a susceptibility exists. When unmanaged, phenylketonuria results is profound intellectual disability; current medical practice, however, has shown that dietary changes can prevent these intellectually crippling effects from occurring. In a like manner, environmental changes and behaviors can prevent the onset of cancers, diabetes, etc. The stress-diasthesis model has been proposed as an explanation for the onset of illness in one patient with a relative absence in another.[127] Several sources have suggested that life stressors are likely triggers for the onset of depression; this interplay is complex, and the genetic predisposition may even give rise to the environmental stressors that cause a full-blown depressive episode.[128]

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[125] Victor and Ropper, *Principles of Neurology*, p. 1616

[126] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 203. 0.8-0.9 and 0.45 are on a scale from 0 to 1, which signifies complete absence of heritability to total heritability.

[127] Victor and Ropper, *Principles of Neurology*, p. 1616; K.S. Kendler, et al., "A Longitudinal Twin Study of 1-Year Prevalence of Major Depression in Women," *Archives of General Psychiatry* 50, no. 11 (November 1993): 843-52.

[128] K.S. Kendler and L. Karkowski-Shuman, "Stressful Life Events and Genetic Liability to Major Depression: Genetic Control of Exposure to the Environment?" *Psychological Medicine* 27, no. 3 (May 1997): 539-47; O. Agid, et al., "Environment and Vulnerability to Major Psychiatric Illness: A Case Control Study of Early Parental Loss in Major Depression, Bipolar Disorder and Schizophrenia," *Molecular Psychiatry* 4, no. 2 (March 1999): 163-72.; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 207

Monoamine transport and reception

Part of the genetic predisposition to affective disorder may involve the development of monoaminergic systems.[129] Kandel suggests that:

> Certain major depressive illnesses may be the result of genetically determined defects in chemical synaptic transmission involving at least two major transmitter pathways of the brain: the serotonergic and noradrenergic systems. Although the mechanisms that cause the defects in transmission remain obscure, progress in studying allelic variations in the human genome provide hope that aspects of the molecular basis of affective disorders might soon be elucidated.[130]

There is some controversy, however. Pliska notes that studies have shown that there is no noticeable genotype difference for serotonin transporter binding alleles between depressed individuals who had committed suicide versus normal controls, suggesting that "the individual's genotype was not related to serotonin transporter binding in the brain."[131] Further, he argues that none of the genes for norepinephrine transporters or receptors have been implicated in affective disorders, nor have the enzymes that convert dopamine β hydroxylase (the chemical precursor of norepinephrine) been implicated. This, he argues, suggests that the common serotonin/norepinephrine disruption in depression may not be due to genetic reasons, but rather, these dysfunctions in neurotransmission may actually be "downstream of the actual cause of the condition."[132]

Neurochemistry

The most common reductionistic model of depressive disorders hinges upon dysregulation of monoamines, a group of neurotransmitters involved in a variety of processes and behaviors from

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[129] Monoaminergic systems involve the production, transmission, and reception of monoamines. Monoamines are neurotransmitters and/or neuromodulators, and are divided into two groups: the catecholamines (dopamine, epinephrine, norepinephrine) and serotonin. See the section on neurochemical dysfunction for the discussion on neurotransmission.
[130] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1224
[131] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 205
[132] Pliszka, p. 206

sleep and relaxation to pleasure and stimulation.[133] Nemeroff notes that a significant body of evidence suggests that "regardless of the initial triggers, the final common pathways to depression involve biochemical changes in the brain. It is these changes that ultimately give rise to deep sadness and the other salient characteristics of depression."[134] Victor and Ropper describe the importance of monoamines in the biological basis of depressive disorders when they state:

> The biogenic monoamines (norepinephrine, serotonin, and dopamine) are the key elements in these theories. Following the observations that the tricyclic antidepressants and the MAO inhibitors exert their effect by increasing norepinephrine and serotonin at central adrenergic receptor sites in the limbic system and hypothalamus and that depression-provoking drugs (such as reserpine) deplete biogenic amines at these sites, the theory followed that naturally occurring depressions might be associated with a deficiency of these latter substances. Furthermore it was observed that depressed patients and their first-degree relatives, as well as healthy individuals, develop a greatly depressed mood after dietary depletion of the monoamine precursor tryptophan.[135]

There are two classes of neurotransmitters in the monoamine group – catecholamines (dopamine, norepinephrine (noradrenaline), and epinephrine (adrenaline)) and serotonin. Dopamine, norepinephrine, and epinephrine are all derived from the common chemical precursor tyrosine, while serotonin is derived from tryptophan. These neurotransmitters are manufactured in the axon terminal of the respective monoamine neuron (e.g., certain neurons produce dopamine, others produce epinephrine, others produce serotonin, etc.), and are released into the synaptic cleft between neurons as a means of excitatory or inhibitory communication throughout the central nervous system.[136] Once the neurotransmitter has been released, the synapse is cleared

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[133] Victor and Ropper, *Principles of Neurology*, p. 1616
[134] Nemeroff, "The Neurobiology of Depression.", p. 264-5
[135] Victor and Ropper, *Principles of Neurology*, p. 1616
[136] Neurons do not physically touch one another – they are held in place by glial cells and communicate chemically with one another through the release of neurotransmitters. These neurotransmitters either polarize or depolarize the target neuron, inhibiting or exciting it, respectively. There are several varieties of neurons with different physical properties and structures.

by reuptake and enzymatic action (i.e., the neurotransmitter is reabsorbed into the releasing neurons terminal or is deactivated and disassembled by an enzyme).

There are several mechanisms that can lead to a dysregulation of monoamines. Neuronal death from a medical condition or trauma can result in monoaminergic dysregulation; insufficient neurotransmitter release, excessive enzymatic "clean-up" activity, excessive reuptake of monoamines, and neurological response to exogenous conditions are also possible causes. Medications treating depression through monoamine regulation are of several classes: monoamine oxidase inhibitors (MAOI) operate by inhibiting the enzyme that deactivates monoamines, resulting in a greater amount of the neurotransmitter in question in the synapse for a longer period of time; selective serotonin reuptake inhibitors and serotonin-noradrenaline reuptake inhibitors (SSRIs and SNRIs, respectively) operate by preventing the reabsorption of the neurotransmitter into the transmitting terminal; tricyclic antidepressants (named for their ringed structure) operate by inhibiting the reuptake of serotonin and norepinephrine.

Serotonin

There are several physiological signs that serotonin depletion is correlated with depression – serotonin-producing cells extend into some of the critical areas involved in emotion (amygdala), arousal (hypothalamus), and cognitive functions (frontal cortices);[137] the effects of hypothalamic dysfunction – and thereby the HPA axis (hypothalamus, pituitary gland, adrenal gland) – will be discussed below in the section presenting endocrinologic bases of depression. Second, serotonin metabolite levels typically are low in depressed patients, suggesting reductions in available serotonin in the brain.[138] Third, lower levels of surface molecules unique to serotonin-releasing cells are found in depressed patients, suggesting that the amount of

[137] Nemeroff, "The Neurobiology of Depression.", p. 269
[138] Nemeroff, p. 269

serotonin-releasing cells themselves is also reduced.[139] Fourth, there is an overabundance of type-2 serotonin-receptors in the brain tissue of depressed patients – up-regulation of neurotransmitter receptors is indicative of the brain's compensatory mechanism for lower levels of neurotransmitter (the brain tries to increase the amount of serotonin transmitted by increasing the number of serotonin receptors in cells).[140] Nemeroff also notes that there is a noticeable difference in the absorption of serotonin by blood platelets in depressed patients versus normal patients.[141] Last, Pliska notes that "there appear to be fewer serotonin transporters in the cortex of persons with major depression, and several studies show the number of the 5-HT_{1A} and 5-HT_{2A} receptors to be decreased."[142]

Norepinephrine

Deficiencies in serotonin can also lead to deficiencies in norepinephrine – some of the systems enervated by serotonin neurons produce and regulate the synthesis and distribution of norepinephrine.[143] If the supply of serotonin is suppressed or inadequate in a noradrenergic system, there will likely be a comorbid suppression or insufficiency of norepinephrine.

There are two pieces of evidence supporting the involvement of norepinephrine in depression. First, Nemeroff notes that the level of norepinephrine metabolites (waste products of norepinephrine use) in the urine and cerebrospinal fluid of depressed patients were low in depressed individuals in relation to 'normal' patients.[144] This suggests that there is a low level of the neurotransmitter in the brains of depressed patients. Second, postmortem studies have noted

[139] Nemeroff, p. 269
[140] Nemeroff, p. 269
[141] Nemeroff, p. 270
[142] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 209. 5-HT_{1A} and 5-HT_{2A} receptors [5-hydroxytryptomine (serotonin)] are linked to second messenger systems, which trigger a series of biochemical changes in cells.
[143] Nemeroff, "The Neurobiology of Depression.", p. 269
[144] Nemeroff, p. 268

an increase of norepinephrine receptors in the post-synaptic membranes of depressed patients – again, suggesting a compensatory mechanism for low levels of the neurotransmitter.[145]

There is some controversy as to whether the monoamine model is accurate. Pliska suggests that the monoamine hypothesis of affective disorder may have to be revisited, as "tianeptine, a substance that enhances the uptake of serotonin (the opposite effects of the SSRIs), has been shown to have antidepressant properties."[146] A stronger objection comes from Kandel, who suggests that the biogenic amine model has a likely basis, but that there are several confounding factors, including a more complex interlacing of the monoaminergic systems than is presently understood, as well as the likelihood that the categories of depressive disorders are in actuality groups of disorders with common pathologies.[147] Victor and Ropper note that there is presently no reliable biological test for depression (see below for the discussion of the dexamethasone suppression test). Rather, "further knowledge of the metabolism and physiology of transmitter function of the biogenic amines is needed before a complete theory can be developed."[148] Psychiatry is relatively new in relation to internal medicine, and undergoes paradigm shifts with greater frequency than house medicine.

Neuroanatomy

The structure of the brain itself is currently under investigation as a cause for depressive symptoms.[149] There are a variety of structures in neuroanatomy which influence mood – pleasure centers, pain centers, regulatory mechanisms, sleep mechanisms, alert centers, etc. A full treatment of the neuroanatomy of mood would require a much longer work, and several

[145] Nemeroff, "The Neurobiology of Depression.", p. 268
[146] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 209
[147] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1219
[148] Victor and Ropper, *Principles of Neurology*, p. 1617
[149] Alan F. Schatzberg, "Major Depression: Causes or Effects?" *American Journal of Psychiatry* 159, no. 7 (July 2002): 1077-79, p. 1078-9

volumes in biopsychology have already covered this topic. A brief introduction should suffice to cover the major structures in question – the structures tend to revolve around the limbic system (emotional regulation) and the frontal lobes:

> Above the cerebellum is the group of structures known as the limbic system. This part of the brain, which we share in common with all other mammals, provides us with primal urges and powerful emotions crucial for self-preservation: rage, terror, hunger, and sexual desire. The limbic system's direct connections with some of the higher brain faculties allow us both to cogitate upon what we feel emotionally, as well as have emotional reactions to that which we think about. The found major components of the limbic system are the amygdala, the hippocampus, the hypothalamus, and the thalamus. The almond-shaped amygdala, which plays a role in the emotions, especially aggression, is the basic pathway into the limbic system for nerve impulses. The hippocampus is an information processor, matching new data against those already stored in the brain. It therefore is one of the structures absolutely critical in the process of ascribing meaning to the symbols and events of our lives. The hypothalamus, integral to our moods, regulates food intake, internal water balance, and reproductive cycles. It generally acts as a liaison between the brain and the rest of the body, initiating the release of at least seven different hormones to the pituitary (or master) gland, which in turn releases other hormones into the bloodstream that influence growth, aging, and all aspects of reproduction. The thalamus, located near the center of the brain, processes all the senses except smell. It takes the incoming sensory signals and, like a switchboard, sends them to the appropriate region in the brain for interpretation.[150]

Depression exerts profound stress on the nervous system; the National Institute of Mental Health has noted that "brain imaging research is revealing that in depression, neural circuits responsible for moods, thinking, sleep, appetite, and behavior fail to function properly, and that the regulation of critical neurotransmitters is impaired."[151] Kandel notes that some of the symptoms of depression suggest a dysregulation of the pleasure/reward and fear systems.[152]

Efforts to find a specific anatomical correlate for depression have thus far been unsuccessful, but Victor and Ropper note that several structures have consistently been found to

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[150] The National Institute of Neurological Disorders and Stroke and The National Institute of Mental Health, "The Brain and Nervous System," in *The Johns Hopkins Medical Handbook: The 100 Major Medical Disorders of People Over the Age of 50*, ed. Simeon Margolis and Hamilton Moses (New York: Rebus, Inc., 1992), 96-160, p. 96

[151] National Institute of Mental Health, *The Invisible Disease: Depression*, p. 2

[152] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1216

be involved in a concomitant depressed mood. They note that the cingulate, orbitofrontal cortices, the insular cortex, amygdala, and basal ganglia have been proposed as causative bases for depression.[153] The structure that shows up most frequently in physiological studies of major depression is the left frontal cortex[154] – the frontal cortices are the basis of cognition and planning; they are the "executive" of the brain, so to speak. In patients experiencing depression, the most frequent finding is hypoactivity in the left frontal cortex. Dysfunction of the left frontal lobe produces the characteristic signs and symptoms of major depression – anhedonia, apathy, lack of volition, anergia, etc.[155] While there are a variety of neurochemical, hormonal, and structural abnormalities that can give rise to depressive states,[156] it is useful to note the importance of the hippocampus and frontal lobes, as the literature tends to revolve around their function in biopsychology.

Hippocampus

The hippocampus has attracted significant recent attention, as it may demonstrate a specific biological marker for depression.[157] A variety of studies have examined the total volume of the white and gray matter in the hippocampus, and have noted that the volume of these areas decreases in patients with depression.[158] There are some questions about these results, however. Some concern has been expressed concerning whether depletion in hippocampal volume is causative for, caused by, or correlative to major depressive episodes,

[153] Victor and Ropper, *Principles of Neurology*, p. 1617.
[154] Victor and Ropper, p. 1617.
[155] Joseph, "Frontal Lobe Psychopathology: Mania, Depression, Confabulation, Catatonia, Perseveration, Obsessive Compulsions, and Schizophrenia.", p. 138; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 216.
[156] "Neurologic Disorders.", p. 50.
[157] Schatzberg, "Major Depression: Causes or Effects?", p. 1078-9.
[158] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 214; Thomas Frodl, et al., "Hippocampal Changes in Patients With a First Episode of Major Depression," *American Journal of Psychiatry* 159, no. 7 (July 2002): 1112-18, p. 1115.

while others suggest that the depletion may simply be coincidental.[159] Pliska notes that the degree of the reduction in hippocampal volume is directly linked to the lifetime duration of the depressive disorder, and that at times it can be so marked as to "be visible to the untrained eye on the MRI scan."[160] Schatzberg suggests that decreased hippocampal volume could result in elevated levels of glucocorticoid during stressful periods, which may put the patient at an elevated risk for neural atrophy and dysfunction in the prefrontal lobes (see below).[161] Frodl notes that while depression is more frequently seen in women, men have a higher risk of depleted hippocampi, which he suggests may be attributable to gender differences in brain development and response to neurotoxins and stress.[162]

Frontal Lobes

As noted above, the frontal lobes can have a significant impact on mood and emotional functioning. The frontal lobes are the seat of executive functioning – they not only decide what will be done, but also put the plan into action (through the motor cortices on the posterior frontal lobes). They are directly connected to a variety of other processing areas, involving memory, association, volition, etc., and indirectly connected to deeper sensory and regulatory structures. Joseph offers a succinct summary of psychopathology associated with frontal lobe damage:

> The frontal lobes can be subdivided into major functional neuroanatomical domains, which, when injured, surgically destroyed, or reduced in activity or volume, give rise to signature pathological and psychiatric symptomology. A review of case reports and over 50 years of research, including magnetic resonance imaging, positron emission tomography, and single photon emission computed tomography scans, indicates that apathy, 'blunted' schizophrenia, major depression, and aphasic-perseverative disturbance of speech and thought are associated with left lateral as well as bilateral frontal (and striatal) abnormalities. Impulsiveness, confabulatory verbosity, grandiosity, increased sexuality, and

[159] Schatzberg, "Major Depression: Causes or Effects?", p. 1077; Frodl, et al., "Hippocampal Changes in Patients With a First Episode of Major Depression.", p. 1116.
[160] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 214
[161] Schatzberg, "Major Depression: Causes or Effects?", p. 1078
[162] Frodl, et al., "Hippocampal Changes in Patients With a First Episode of Major Depression.", p. 1115

> mania are associated with right frontal (as well as bilateral) disturbances. Gegenhalten, catatonia, and disturbances of 'will' are indicative of medial frontal injuries. Disinhibitory states and obsessive-compulsive perseverative abnormalities are more frequently observed with orbital frontal lobe dysfunction, including frontal-striatal disturbances. These associations, however, are not always clear-cut as patients with the same diagnosis may demonstrate different symptoms that may be due to an additional abnormality in a different region of the brain. Moreover, as the frontal subdivisions are richly interconnected, and as frontal lobe abnormalities are not always discrete or well localized, a wide array of seemingly divergent waxing and waning symptoms may be manifest, sometimes simultaneously, including manic depression and what has been referred to as the 'frontal lobe personality.'[163]

Pliska notes that positron-emitting tomography studies have shown a decrease in the activity of the dorsolateral prefrontal cortex, which may "correlate with the cognitive impairments in depression, such as decreased concentration and poor memory."[164] Further, it is noted that this decrease in activity returns to normal following treatment for depression.[165] There are also structures that appear to have hereditary weaknesses – Kandel notes that fMRI (functional magnetic resonance imaging) studies have defined an area of the prefrontal cortex beneath the corpus callosum that appears to be only affected in hereditary affective disorders.[166] The activity in the region decreases during periods of depression (in both unipolar and bipolar depression), while activity in the regions spikes during manic phases. Kandel suggests that the decrease in activity may be accounted for by a significant (45%) reduction in grey matter in that section of the prefrontal cortex.[167] Schatzberg notes that cognitive decline in the left frontal lobe correlates with depressive symptoms, and may be a marker for depression in elderly patients.[168] Left frontal dysfunction and hypoactivity suggest a possible therapeutic intervention – artificial stimulation (chemical or somatic) of left frontal structures may suffice to reverse the depressive

[163] Joseph, "Frontal Lobe Psychopathology: Mania, Depression, Confabulation, Catatonia, Perseveration, Obsessive Compulsions, and Schizophrenia.", p. 138.
[164] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 215.
[165] Pliszka, p. 215; "Neurologic Disorders.", p. 50.
[166] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212-3.
[167] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1212-3.
[168] Schatzberg, "Major Depression: Causes or Effects?", p. 1077.

symptomology (see the treatment section below concerning pharmacological and somatic treatment interventions).

Endocrinology

In addition to the neurochemical and neuroanatomical explanations of depression, answers can also be found in the endocrine system. Dysregulation of several hormones have been proposed for the symptoms found in major depression. Nemeroff suggests that dysregulation of hormones responsible for growth hormone [GH] and thyroid-stimulating hormone [TSH] produce depressive symptomology.[169] Nemeroff and Kandel have also explored dysregulation of adrenocorticotropic hormone [ACTH] in the pituitary gland.[170] This hypersecretion of ACTH leads to an overproduction of cortisol, a stress hormone (see below). Kandel notes that depression is often associated with signs of hypothalamic disturbance, leading to ACTH dysregulation.[171]

Each chemical modifying brain behavior has a receptor fitted specifically to it. Cortisol is normally secreted according to the body's circadian rhythms, with periods of peak activity in the mid-morning; in general, cortisol keeps the individual awake and alert. Cortisol production is connected to activation of the sympathetic nervous system – a branch of the autonomic nervous system that governs the body during times of exertion or conflict. When the sympathetic nervous system is activated, the body tends not to engage in activity that expends energy unrelated to fight or flight – appetite is suppressed, digestion slows, the body is more alert, etc. When an individual neuromodulator or neurotransmitter is not produced in sufficient quantities or is overproduced, the nervous system can compensate for the deficiency or excess by increasing or decreasing the number of corresponding receptors. However, in the case of the

[169] Nemeroff, "The Neurobiology of Depression.", p. 271.
[170] Nemeroff, p. 271; Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1220.
[171] Kandel, p. 1220.

overproduction of corticotrophin-releasing factor [CRF], there is an overabundance of the chemical and an increase in the number of its receptors – the opposite effect we would expect. Thus, we have too much CRF and too many receptors sensitive too it; the net result is a chronic overproduction of cortisol. When cortisol is present in excessive amounts chronically, it can produce classic signs of depression – appetite loss and weight loss, sleep continuity disturbance, loss of sexual appetite, loss of interest in outside activities and interests, anhedonia, etc. In short, the body keeps itself in a condition primed for conflict rather than relaxation, ultimately spiraling the patient into a depressive episode.[172] In some cases, the hypersecretion of ACTH swells the adrenal glands to a noticeable degree.[173] Three principle structures in neuroanatomy are implicated in the overabundance of cortisol – the hypothalamus, the pituitary gland, and the adrenal cortex, collectively referred to as the HPA axis. Nemeroff suggests that HPA-axis hyperactivity is "surely the most replicated one in all of biological psychiatry."[174]

Returning to the theme of the difficulty of corroborating a patient's complaint of depression clinically, it has been suggested by some that neuroendocrinological tests might be employed to verify a claim. The dexamethasone suppression test has been proposed as a possible solution to this need. Adrenocorticotropin normally is suppressed in individuals exposed to dexamethasone, a synthetic corticosteroid. Researchers have found that cortisol hypersecretion is not suppressed in approximately 40% of depressed individuals.[175] This approach is controversial, however, in that dexamethasone is also suppressed in dementia,

[172] National Institute of Mental Health, *The Invisible Disease: Depression*, p. 2
[173] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1220
[174] Nemeroff, "The Neurobiology of Depression.", p. 271
[175] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1220

anorexia nervosa, bulimia, alcohol withdrawal, and weight loss; as such, there is some question as to its specificity.[176]

Pharmacology

A final consideration is the possibility of developing a depressive disorder from routine medications. Again citing J.L. Cummings, Kaplan and Sadock note that there are multiple medications that can cause depressive symptomology (Figure 1)[177]:

Figure 1: Pharmacological Causes of Depression

Cardiac and antihypertensive drugs	
Bethanidine	Digitalis
Clonidine	Prazosin
Guanethidine	Procainamide
Hydralazine	Veratrum
Methyldopa	Lidocaine
Propranolol	Oxprenolol
Reserprine	Methoserpidine
Sedatives and hypnotics	
Barbituates	Benzodiazepines
Chloral hydrate	Chlormethiazole
Ethanol	Chlorazepate
Steroids and hormones	
Corticosteroids	Triamcinalone
Oral contraceptives	Norethisterone
Prednisone	Danazol
Stimulants	
Amphetamine	Diethylproprion
Fenfluramine	Phenmetrazine
Psychotropic drugs	
Butyrophenones	Phenothiazines
Neurological agents	
Amantadine	Baclofen
Bromocriptine	Carbamazepine
Levodopa	Methosuximide
Tetrabenazine	Phenytoin
Analgesics and anti-inflammatory drugs	
Fenoprofen	Phenacetin
Ibuprofen	Phenylbutazone
Indomethacin	Pentazocine
Opiates	Benzydamine
Antibacterial and antifungal drugs	
Ampicillin	Griseofulvin
Sulfamethoxazole	Metronidazole
Clortimazole	Nitrofurantoin
Cycloserine	Nalidixic acid
Dapsone	Sulfonamides
Ethionamide	Streptomycin
Tetracycline	Thiocarbanilide

[176] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1220
[177] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 531.

Antineoplastic drugs

C-Asparaginase	6-Azauridine
Mithramycin	Bleomycin
Vincristine	Trimethoprim
	Zidovudine

Miscellaneous drugs

Acetazolamide	Anticholinesterases
Choline	Cimetidine
Cyproheptadine	Lysergide
Methysergide	Mebeverine
Meclizine	Metaclopramide
Pizotifen	Salbutamol

Because depressive disorders can result from medication used to treat other conditions, it is quite possible that it can be effectively combated by changing medications to a substitute without the extra-pyramidal symptom of depression.

Treatment

There are a variety of treatments for affective disorders – psychopharmacological, psychotherapeutic, and somatic. The National Institute of Mental Health notes that 80% of depressive disorders are treatable through a combination of psychopharmacology and psychotherapy. Bipolar disorders have been shown to respond favorably to treatment with lithium and psychotherapy.[178] Electroconvulsive therapy (ECT) is a useful treatment for depressions that do not respond to other therapeutic interventions, and because of its efficacy and cost, may become the treatment of choice.[179] Each of these therapeutic modalities will be explored briefly, as full discussion of each individually would require textbook-length treatments

[178] In this author's experience, many bipolar patients have an aversion to lithium treatment – while there are therapeutic effects, many have complained of somatic disturbances, and many more find it easier to comply when they are experiencing dysthymic or depressed moods. Characteristically, patients with bipolar disorder tend not to stay on their medications during manic and hypomanic phases – they have repeatedly stated that these periods "simply feel too good to stop." The hypomanic and manic phases are subjectively described as periods of increased intelligence, increased insight and wisdom, greater intuition, energy, etc. During the manic phase, patients will frequently take on more projects than can be handled, as well as engage in behaviors that are dangerous and potentially self-injurious (e.g., hypersexuality, impulsive spending, etc.). For an excellent subjective account of bipolar disorder, see Kay Redfield Jamison's *An Unquiet Mind*.

[179] Patients undergoing ECT report some memory loss following the procedure, however, which may present an unacceptable side effect. In addition, there is still a stigma towards the treatment – in this author's experience, many patients have expressed an aversion based upon popular media presentations of much older methods of "shock therapy."

(specific treatment pathways, underlying medical and psychosocial etiologies, discussion of ethical considerations like informed consent, etc.).

Psychopharmacology

There are several classes of medications employed in the treatment of depression – Kandel notes that depression can be combated through the use of antidepressants, mood stabilizers (lithium), and some anticonvulsants.[180] The antidepressants, generally the first "line of defense" against depression, are further divided into tricyclics (TCA, like amitriptyline (Elavil), doxepin (Sinequan), imipramine (Tofranil), etc.), monoamine oxidase inhibitors (MAOIs, like phenelzine (Nardil), isocarboxazid (Marplan), etc.), selective serotonin reuptake inhibitors (SSRIs, like citalopram (Celexa), fluoxetine (Prozac), sertraline (Zoloft), etc.), serotonin-noradrenaline reuptake inhibitors (SNRIs, like nefazadone hydrochloride (Serzone), venlafaxine (Effexor), etc.), and atypical antidepressants (like trazodone, mirtazapine (Remeron), etc.). For many of these medications, the specific means of their therapeutic effect involves correcting monoamine insufficiencies, while for others the specific means of their therapeutic effect are unknown. As noted above in the discussion of neurochemistry, monoamine deficiencies can result from a variety of etiologies – insufficient production of the monoamines in question, enzymatic deactivation, terminal reuptake, etc. The tricyclic antidepressants prevent the reuptake of serotonin and norepinephrine back into the axon terminal, thus allowing more of them to bind with their corresponding receptors on the post-synaptic membrane and thereby increasing their effect (SNRIs operate in a similar manner, but have a different chemical structure – the tricyclics are so named for their common "three rings" shape). Monoamine oxidase inhibitors operate by preventing the enzyme monoamine oxidase from deactivating the neurotransmitter, allowing more to bind to the corresponding receptor on the post-synaptic

[180] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1213

membrane. Like tricyclics and SNRIs, selective serotonin reuptake inhibitors prevent the reuptake of serotonin into the axon terminal, allowing for greater amounts to bind to serotonin receptors on the target neuron.

Mood stabilizers like lithium are more suited for individuals with a bipolar disorder – these patients have a delicate neurochemistry which tends to react unfavorably to certain medications. For example, if a patient with bipolar disorder is given an antidepressant, it is quite likely that they will not return to baseline – rather, they will enter a manic phase, with a corresponding shift in behavior and affect.[181] Kandel notes that lithium is occasionally used in treating unipolar depression, but only when used in conjunction with an MAOI, TCA, or SSRI, as the lithium augments the therapeutic effects of these antidepressants).[182] The efficacy of the monoamines is noteworthy; Kandel states that "the monoamine oxidase inhibitors and the tricyclic antidepressants produce remission or marked improvement in about 70% of patients with major depressions. When optimal doses are given, the success rate with tricyclic drugs and the specific serotonin reuptake inhibitors may reach 85%, almost as effective as ECT."[183] Anticonvulsants currently in use include carbamazepine (Carbatrol, Tegretol), gabapentin (Neurontin), lamotrigine (Lamictal) and valproate (Depakene).

Many of these medications have side effects, and some carry other concerns – with MAOIs, for example, there are dietary considerations, as patients must avoid a high tyramine diet (as a result, there are restrictions on wine, cheeses, etc.). The side effects are frequently a barrier to medication compliance; further, in patients with concomitant substance abuse problems, medication compliance suffers due to the psychopharmacologic properties of alcohol, street drugs, and prescription drugs used improperly. In dually diagnosed patients – patients with

[181] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1214
[182] Kandel, p. 1216
[183] Kandel, p. 1214

comorbid psychiatric disorders and substance abuse disorders – medication compliance frequently takes second priority to self-medication. Self-medication is frequently used as a coping mechanism for other psychosocial and medical problems (e.g., a patient who drinks because he has pancreatitis, and thereby exacerbates his condition, or a patient with schizophrenia who uses cocaine to stop the voices, only to discover that the voices get worse[184]).

Psychotherapy

There are a variety of mechanisms utilizing psychotherapies ("talk therapies"). A full presentation of the available talk therapies is well beyond the purview of this dissertation; such a project would require a book in and of itself.[185] There are, however, several popular approaches, which can be covered succinctly.[186]

Cognitive-behavioral therapy, developed by Alfred Beck, enjoys widespread practice, and a body of literature exists suggesting that it is an effective means of treating depression. Cognitive-behavioral therapy (CBT) suggests that the emotional reactions a patient has towards stressful situations is not a result of the situation itself, but rather the result of a specific thought the patient had. In essence, the patient would automatically interpret a situation or event, creating a thought reaction to the event. This reaction gives rise to an emotional response (anger, frustration, depression, anxiety, etc.). CBT suggests that by exploring the reaction (an

[184] An excess of dopamine frequently causes the onset of auditory and visual hallucinations (a side effect found while exploring therapeutic vs. toxic doses of L-dopa in Parkinson's disease); cocaine is dopaminergic, causing a spike in already high dopamine levels, which makes the auditory/visual hallucinations worse.

[185] E.g., Gerald Corey, *Theory and Practice of Counseling and Psychotherapy*, Sixth (Belmont: Brooks/Cole, 2001); Corey, *Theory and Practice of Counseling and Psychotherapy*; *Combined Treatments for Mental Disorders*, edited by Morgan T. Sammons and Norman B. Schmidt (Washington, D.C.: American Psychological Association, 2001).

[186] The treatment modalities discussed below are in no way indicative of the limits of available therapies; these have been selected from the author's personal experience as being appropriate for consideration in an acute care facility. Psychoanalytic and psychodynamic perspectives, family therapy perspectives, etc. are all germane and useful therapeutic interventions, but tend to require longer periods of intervention. Cognitive-behavioral and individual psychotherapies can be introduced and taught on a short-term basis, offering rapid psychological treatment to compound pharmacological treatments. Acute care facilities may consider group therapies useful in a variety of house medicine situations (e.g., in oncology units, neurological units, internal medicine wards, etc.) as a supplement to the medical care being offered, in light of the demonstrated prevalence of comorbid depression in common medical illnesses and its effects on recovery and immunology.

'automatic thought' like "They're laughing at me" or "They don't like me"), we can see whether it represents a true assessment of a given situation or whether it represents what Beck calls a "cognitive distortion" – a misrepresentation of the situation to oneself. CBT suggests that depression is the result of cognitive distortions, and leads to a negative bias in decision-making; by addressing the underlying distortions, the patient can improve her mood.[187] As discussed later, CBT is one of the recommended psychotherapeutic interventions for new stroke patients.

Goal-oriented interventions (especially Alfred Adler's Individual therapy) are also suggested in combating depression and self-destructive behavior. The therapist is non-judgmental, and attempts to build rapport with the patient, exploring the patient's life goals (conscious and subconscious). The basis of belief is that the patient wishes to move from a subjectively inferior to a subjectively superior position (to get better physically, get a better job, earn more money, earn more respect, develop a healthy relationship from a dysfunctional one, etc.), and it is the therapist's role to explore these goals, and the means to them, with the patient. If barriers to these goals can be identified, life changes can result which will allow the patient to overcome those challenges. In a depressive disorder, the patient may not necessarily be able to objectively assess her situation; the therapist can provide a means of finding solutions to the problem presently inaccessible to the patient as a result of her condition.

Group therapies and process-oriented therapies are also popular in many acute care and outpatient clinics. A supportive environment is provided to several patients experiencing common problems, and discussion of personal challenges, individual progress and regress, and problem-solving solutions are discussed. Access to group and process treatments may not

[187] We will return to the theme of cognitive bias and distortion in later discussions on depressive realism, which suggests that individuals with mild and moderate forms of depression can actually produce more realistic assessments of control than individuals with severe depression, mania, hypomania, or baseline mood and affect.

necessarily be available to patients in critical care settings, and as such, may not be appropriate recommendations.

Somatic interventions

The type of mechanical intervention most referenced and stigmatized is electroconvulsive therapy. So-called "shock therapy" is the oldest mechanical intervention, and that which gave rise to the current interest in non-pharmacological, non-psychotherapeutic interventions. Contemporarily, ECT is significantly different from its rather barbarous past. Patients are anaesthetized, with the exception of one foot, before the shock is administered (usually a fraction of a second). Following the shock, the patient experiences a surge of electrical activity initiated in the left frontal lobe (ECT generally involves electrode placement in one of two places – either one on the posterior left forehead and the other on the center of the forehead, or one on the posterior left forehead and the other on the posterior right forehead. In both cases, the current travels through the left frontal lobe. As mentioned in the section describing neuroanatomical causes of depression, left frontal lobe lesions and/or hypoactivity are both linked to depressive symptomology. Consequently, artificial stimulation of this area of the frontal cortex can cause significantly improved mood. Kandel notes:

> ECT has been used for the longest period of time, over 50 years. Although antidepressants are generally the first choice in the treatment of major depression, ECT is very effective. It produces full remission or marked improvement in about 85% of patients with well-defined major depression…On average, six to eight treatments given at two-day intervals over a period of 2-4 weeks usually suffice to produce a complete remission of symptoms.[188]

Rush, et al., estimate that approximately 100,000 patients annually receive electroconvulsive therapy.[189] While the procedure is generally safe, there are some cardiac concerns – ECT is

[188] Kandel, "Disorders of Mood: Depression, Mania, and Anxiety Disorders.", p. 1213

[189] A. John Rush, et al., "Vagus Nerve Stimulation (VNS) for Treatment-Resistant Depressions: A Multicenter Study," *Biological Psychiatry* 47 (2000): 276-86, p. 276-7

associated with activation of the parasympathetic autonomic system (principally through increases in vagus nerve tone [see below]), it can disrupt or suppress heart rate, causing arrhythmias and brief asystole.[190]

An alternative controversial somatic intervention proposed for treatment-resistant depressions is stimulation of the vagus nerve. Originally developed as a treatment for epilepsy, direct vagus nerve stimulation involves surgical placement of an electrical stimulator beneath the skin in the patient's upper left chest/shoulder. A second incision is made nearer to the patient's neck to attach the stimulating device to the left vagus nerve. A controlled shock is then sent, stimulating the nerve. The afferent nerve fibers in the vagus activate the parasympathetic autonomic system (responsible for returning the body to rest), along with a variety of other CNS structures.[191] The treatment was found to be effective for patients experiencing epilepsy, but researchers also noted that a significant number began to experience an elevation in mood that was not adequately accounted for by the reduction in seizure activity.[192] It was hypothesized that stimulation of the vagus nerve could have antidepressant effects.[193]

The vagus nerve travels extensively, and connects to several structures in the forebrain, as well as deeper structures involved in autonomic responses and emotional regulation. The exact therapeutic methodology of vagus nerve stimulation is presently idiopathic, but it is hypothesized that artificial stimulation of the vagus nerve leads to changes in neurotransmission, specifically in serotonin, norepinephrine, gamma-aminobutyric acid (GABA), and glutamate, all

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[190] Mustafa M. Husain, et al., "Safety of Vagus Nerve Stimulation with ECT," *American Journal of Psychiatry* 159, no. 7 (July 2002): 1243, p. 1243

[191] Mark S. George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy," *Biological Psychiatry* 47 (2000): 287-95, p. 288

[192] George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy.", p. 292; Rush, et al., "Vagus Nerve Stimulation (VNS) for Treatment-Resistant Depressions: A Multicenter Study.", p. 284

[193] George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy.", p. 293

of which are "implicated in the pathogenesis of major depression."[194] While there have been cases in which vagus nerve stimulation has had the desired anti-depressive effect, there are also cases in which no significant reduction in depressive features was realized, and at least one case in which the implant may have caused a depressive reaction. Clearly there is more to be researched, and the role of the vagus nerve in influencing mood ought to be more fully explored.[195] Other mechanical interventions relying on direct stimulation of the nervous system (like deep brain stimulation, in which "a thin electrode is inserted directly into the brain and different currents are applied at varying depths until the desired effects are found"[196]) have been suggested as possible future therapeutic interventions. Therapeutic interventions that are less invasive (e.g., transcranial magnetic stimulation) are also presently being explored.[197]

Depression in Medical Illness

There are a variety of medical conditions which give rise to depressive illnesses. Comorbid depression can be caused by neurological conditions, cerebrovascular conditions, metabolic conditions, endocrine conditions, autoimmune conditions, infections, and neoplasms.[198] Citing J.L. Cummings[199], Kaplan and Sadock note a variety of medical dysfunctions that can cause the onset of depression (Figure 2)[200]:

[194] George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy.", p. 292
[195] For a more detailed description of the vagus nerve hypothesis and its strengths/weaknesses, please see George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy.", Rush, et al., "Vagus Nerve Stimulation (VNS) for Treatment-Resistant Depressions: A Multicenter Study.", John F. Prater, "Recurrent Depression with Vagus Nerve Stimulation," *American Journal of Psychiatry* 158, no. 5 (May 2001): 816-17, and Husain, et al., "Safety of Vagus Nerve Stimulation with ECT.", among others.
[196] George, et al., "Vagus Nerve Stimulation: A New Tool for Brain Research and Therapy.", p. 287
[197] George, et al., p. 287
[198] *DSM*, p. 403
[199] J.L. Cummings, *Clinical Neuropsychiatry* (Orlando: Grune & Stratton, 1985).
[200] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 530.

Figure 2: Neurological and Medical Causes of Depression

Neurological Disorders
Extra pyramidal diseases
- Parkinson's disease
- Huntington's disease
- Progressive supranuclear palsy

Alzheimer's Disease
Cerebrovascular disease
Cerebral neoplasms
Cerebral trauma
CNS infections
Dementia
Migraine
Multiple sclerosis
Epilepsy
Narcolepsy
Hydrocephalus
Sleep apnea
Wilson's disease

Systemic disorders
Infections
- Viral
- Bacterial

Endocrine Disorders
Adrenal (Cushing's, Addison's diseases)
Hyperaldosteronism
Menses-related
Parathyroid Disorders (hyper- and hypo-)
Postpartum

Thyroid Disorders (hypothyroidism and apathetic hyperthyroidism)

Inflammatory disorders
Systemic lupus erythematosus
Rheumatoid Arthritis
Temporal arteritis
Sjögren's syndrome

Vitamin deficiencies
Folate
Vitamin B_{12}
Niacin
Vitamin C
Thiamine

Other Disorders
Cancer
Cardiopulmonary disease
Renal disease and uremia
Systemic neoplasms
Porphyria
Klinefelter's syndrome
Acquired immune deficiency syndrome (AIDS)
Postpartum mood disorders
Postoperative mood disorders

Further, depression comorbid with a medical condition can greatly increase the risk of suicide.[201] The DSM notes that the prevalence of affective disorders in medical illness is rather high – on

[201] *DSM*, p. 402

average, 25%-40% of individuals with a neurologic condition (such as Parkinson's disease, Huntington's disease, multiple sclerosis, stroke, Alzheimer's disease, etc.) will develop a depressive illness during the course of their illness. In general medical conditions, this rate fluctuates greatly, with an 60% estimated prevalence of comorbid depression in Cushing's syndrome to an 8% estimated prevalence of comorbid depression in end-stage renal disease.[202] Comorbid depression is frequently masked by medical illnesses, but there seem to patterns in symptomology – complaints of fatigue and lassitude have frequently been linked to some manner of psychiatric illness. Victor and Ropper note that:

> In one series, 85 percent of persons admitted to a general hospital and seen in consultation by a psychiatrist for the chief complaint of chronic fatigue were diagnosed, finally, as having anxious depression or anxiety neurosis. In a subsequent study, Wessely and Powell found similarly that 72 percent of patients who presented to a neurologic center with unexplained chronic fatigue proved to have a psychiatric disorder, most often a depressive illness.[203]

They note that patients presenting with medical or neurological illness rarely discuss feelings like sadness or despair without mentioning concomitant physical disabilities or symptoms (e.g., fatigue, anxiety, loss of appetite, sleep continuity disturbances, etc.). When these conditions are present, they suggest that there may be a concomitant depressive episode.[204] In fact, they strongly suggest that endogenous depression should be suspected in *every* chronic case of illness or disability.[205] What follows is a general discussion of commonly presenting medical conditions which can give rise to comorbid affective illness.

MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is a disease affecting the myelin sheath of axons. Symptoms include motor weakness, partial paralyzation (paraparesis), abnormal sensations or lack of

[202] *DSM*, p. 403
[203] Victor and Ropper, *Principles of Neurology*, p. 526-527
[204] Victor and Ropper, p. 1610
[205] Victor and Ropper, p. 535

sensation (parasthesias), visual impairment, double vision (diplopia), abnormal oscillations of the eyes (nystagmus), difficulty in speech production (dysarthria), intention tremor, loss of balance (ataxia), impairment of deep sensation, and bladder dysfunctions.[206] Early-onset symptoms include weakness or numbness in one or more limbs, a tingling feeling during passive flexion of the neck, pain in the lower back or limbs, partial or complete vision impairment/loss (optic neuritis), inflammatory and demyelinative lesions of the spinal cord (transverse myelitis), loss of balance or stability in movement (cerebellar ataxia), vertigo, facial pain or numbness, paresthesias, and disorders of urination (micturation).[207]

Epidemiology

In the United States, MS has a prevalence that varies from 6-14 per 100,000 to 30-80 per 100,000, dependent upon one's geographic location[208] - the overall rate in western countries is as high as 1 in 1,000.[209] Chwastiak et al note that it is "the most common chronic disabling CNS [central nervous system] disease in young adults."[210] MS tends to affect younger adults more than the elderly – two thirds of MS cases first present symptoms between the ages of 20 to 40.[211] There is a genetic link, approximately 15% of MS cases have an affected relative (siblings with the same parents have the highest risk (two to three times higher than siblings with only one parent in common).[212]

Course of the Illness

MS results in progressive neurological damage and dysfunction. While some remission occurs after each attack (even complete remission in some cases), the morbid and mortal nature

[206] Victor and Ropper, *Principles of Neurology*, p. 955
[207] Victor and Ropper, p. 961-2
[208] Victor and Ropper, p. 957. There is a higher incidence of MS in the northern states than in southern.
[209] Chwastiak, et al., "Depressive Sumptoms and Severity of Illness in Multiple Sclerosis: Epidemiologic Study of a Large Community Sample.", p. 1862
[210] Chwastiak, et al., p. 1862
[211] Victor and Ropper, *Principles of Neurology*, p. 958. Of the remaining third, most cases are below the age of 20.
[212] Victor and Ropper, p. 958

of the illness is undeniable. MS assaults the patient in a series of attacks, each resulting in greater levels of CNS deterioration as the lesions accumulate.[213] These attacks are episodic, and tend not to be a steady decline from the initial onset of the condition (only about 10% of the cases are a continuous decline from the first attack).[214] As these attacks accumulate, the patient's cognitive and adaptive abilities decline, increasing the difficulties they face:

> Impairment could be seen whether neurological involvement was mild or severe, suggesting that cognitive impairments were sometimes present early in the disease. Peyser *et al*. stress that cognitive impairments in multiple sclerosis can be subtle, need not be apparent to the patient, and are liable to be missed in the routine neurological evaluation. From the above it seems clear that patients with the disease have not only to adapt to progressive physical disability, but must often do this against a background of progressively diminishing intellect and impaired adaptive capacity.[215]

Patients typically survive over 30 years – only a small number of patients die within the first few months or years.[216]

Rates of depression

The incidence of comorbid depression in MS is significant. Chwastiak et al note that studies have indicated as many as 1 in 2 patients with MS will experience an episode of major depression:

> Depression may be more common in multiple sclerosis than in other chronic neurological conditions. Epidemiologic studies of patients at specialty clinics have indicated that the lifetime risk of major depression in multiple sclerosis is between 22.8% and 54.0%. Small studies also suggest a wide range for the point prevalence of depression in multiple sclerosis, 27%-54%. These studies have used a variety of screening measures to detect depression and have been conducted in specialty clinics, which may not be representative of the underlying population. Risk factors for major depression in multiple sclerosis were identified

[213] Victor and Ropper, *Principles of Neurology*, p. 970
[214] Victor and Ropper, p. 970
[215] William Alwyn Lishman, *Organic Psychiatry* (Boston: Blackwell Scientific Publications, 1987), p. 592
[216] Victor and Ropper, *Principles of Neurology*, p. 971

> in one study and included female gender, age less than 35 years, family history of major depression, and a high level of stress.[217]

These numbers are indicative of only one of the disorders within the spectrum of depressive symptomology. The overall prevalence of depressive disorders is likely higher as the inclusion criteria are widened.[218] While the disease is a chronic condition of long duration, the risk of depression is not principally linked with the amount of time the patient carries the diagnosis. While there is some risk of comorbid depression in newly diagnosed patients, the likelihood of a major depressive episode decreased as the time since the diagnosis increased. Chwastiak et al note that "the lower prevalence of severe depressive symptoms in patients with longer durations of multiple sclerosis suggests that patients may adapt to illness over time. Subjects who were within 1 year of the diagnosis of multiple sclerosis were more often severely depressed."[219] As such, while physicians ought to concern themselves with newly diagnosed cases of MS, they should not be as concerned with patients who have carried the diagnosis for longer periods of time. Instead, comorbid depression seems to be primarily dependent upon the severity of the resulting dysfunction:

> In this large community sample of persons with multiple sclerosis, severity of multiple sclerosis was more strongly associated with depressive symptoms than was duration of illness or pattern of progression…These findings are consistent with longitudinal findings on chronic mental illness and normal aging that suggest that depressive symptoms and major depressive disorder develop as functional impairment increases.[220]

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[217] Chwastiak, et al., "Depressive Sumptoms and Severity of Illness in Multiple Sclerosis: Epidemiologic Study of a Large Community Sample.", p. 1862

[218] If inclusion criteria for dysthymia, atypical depression, and minor depression are included, patients with depressive symptoms who do not meet the criteria for a major depressive episode could be considered. For example, dysthymia requires periods of sadness for two years, which is well within the purview of a chronic illness typically lasting for thirty years. It does not require a significant leap in reasoning to imagine a patient having a protracted period of sadness and difficulty adjusting to the fact that he has a progressive, fatal neurological disorder.

[219] Chwastiak, et al., p. 1866

[220] Chwastiak, et al., p. 1866

The overall risk of comorbid depression is markedly different for patients with mild, moderate, and severe resultant dysfunctions. They note that in one study "the odds of depressive symptoms in subjects with intermediate illness was three times that of the group with minimal severity of multiple sclerosis, and the odds for those with advanced severity was six times as high."[221] Last, the amount of social support enjoyed by the patient with MS directly affects their reaction to their condition. Patients with a larger network of support (friends, family, support groups, etc.) tend to be more resilient in weathering illness. As such, the most important clinical risks for depression are recent diagnosis, major loss of function, and limited social support.[222]

Treatment options

There are two principal methods of treatment: corticosteroids and interferon injections.[223] Corticosteroids attempt to treat the symptoms of MS, while interferons attempt to alter the course of the illness. There are risks and benefits to both. Corticosteroids have been shown to offer relief of many of the symptoms of MS, but there is a significant number of patients for whom this treatment is of no benefit. Further, it is to be considered a short-term treatment, as "there is no evidence that steroids have a significant effect upon the ultimate course of this disease or that they prevent recurrences, so there is little justification for steroid treatment over a period of many months or years."[224] In fact, some of the common side effects of corticosteroid treatment are insomnia and depressive or manic symptoms.[225] In light of what has been said above, the irony

[221] Chwastiak, et al., "Depressive Sumptoms and Severity of Illness in Multiple Sclerosis: Epidemiologic Study of a Large Community Sample.", p. 1864
[222] Chwastiak, et al., p. 1867
[223] There are alternative therapies proposed (such as immunosuppressive medications, synthetic polypeptides, hyperbaric oxygen, diet changes, and linoleate supplementation), but the data presented thus far has not supported them as advised or reliable treatments. There is at least one trial study that has shown some benefit in monthly immunoglobulin infusions (Victor and Ropper, *Principles of Neurology*, p. 974).
[224] Victor and Ropper, *Principles of Neurology*, p. 973
[225] Victor and Ropper, p. 973

of these side effects is that a patient may become depressed as a result of both his condition and his treatment for it.

The other principal treatment is the use of interferons, which can "give promise of modestly altering the natural history of the disease."[226] Betaseron (interferon beta-1b) has been shown to decrease the frequency and severity of relapses by approximately one-third, as well as decreasing the number and size of new lesions; further, it has been demonstrated to slow the progression of the disease over extended periods.[227] Avonex (interferon beta-1a) has been shown to be equally effective, and does not require injections of the same frequency as Betaseron.[228] Copolymer I (glatiramer acetate) has been shown to be effective in patients who have developed a resistance to interferon beta.[229] There are several potential detriments to interferon treatment, however. The side effects include "flu-like symptoms" and "malaise," questions still remain about the long-term effects of the treatment, and the health effects are "not overwhelming."[230]

PARKINSON'S DISEASE

Parkinson's disease is a condition in which dopaminergic neurons in the central nervous system are progressively damaged, leading to progressively worsening muscle rigidity and tremors. It is idiopathic, but several possible etiologies have been proposed, including environmental influences, infections, and genetic predispositions. The classic triad of parkinsonian symptoms consists of tremor, muscular stiffness, and bradykinesia.[231] The principle marker of Parkinson's disease is damage to the substantia nigra (specifically the pars compacta)

[226] Victor and Ropper, *Principles of Neurology*, p. 974
[227] Victor and Ropper, p. 974
[228] Victor and Ropper, p. 974
[229] Victor and Ropper, p. 974
[230] Victor and Ropper, p. 974
[231] Roger C. Duvoisin, *Parkinson's Disease: A Guide for Patient and Family* (New York: Raven Press, 1991), p. 1; Victor and Ropper, *Principles of Neurology*, p. 1131. Both Duvoisin and Victor & Popper note that there are a variety of etiologies for parkinsonism: drug induced, postencephalitic (secondary to an inflammation of the brain), arteriosclerotic, symptomatic (e.g., from intoxication, poisoning, brain injury, etc.), and secondary to other neurological insults.

– a dopamine creation, storage, and transport mechanism.[232] The substantia nigra transports dopamine to various locations in the corpus striatum (deep gray matter in the central nervous system). Dopamine elevations and deficiencies have been correlated to depression, euphoria (as in cocaine intoxication), and psychosis (extreme excess of dopamine). When the corpus striatum has a severely reduced dopamine level, the affected brain cells are prevented from performing their normal inhibitory function.[233] Parkinsonian symptoms then develop, leading some to label parkinsonism as a state of "brain dopamine depletion."[234]

Epidemiology

In the United States, Parkinson's disease affects approximately 1% of the population over the age of fifty (approximately 500,000 people in 1991,[235] approximately 1,000,000 in 2002[236]). Overall, the prevalence is approximately 100 per 100,000 people less than fifty years old and 1,100 per 100,000 for people older than eighty.[237] It rarely affects individuals under the age of forty.[238] Chow and Cummings note that it is the most commonly encountered extrapyramidal movement disorder.[239] Men tend to develop the condition more than women,[240] and the disease apparently is not isolated to any one particular region or environment.[241] Race does seem to play a factor in susceptibility; the prevalence in white populations tends to be higher than in other

[232] Chow and Cummings also include loss of dopaminergic neurons in the pars compacta as a cardinal manifestation of Parkinson's disease. Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 31.
[233] "Neurologic Disorders.", p. 763-4.
[234] Duvoisin, *Parkinson's Disease: A Guide for Patient and Family*, p. 2-3; Mahlon R. DeLong, "The Basal Ganglia," in *Principles of Neural Science*, ed. Eric R. Kandel, James H. Schwartz and Thomas M. Jessell, Fourth (New York: McGraw-Hill Health Professions Division, 2000), 853-67, p. 864.
[235] Duvoisin, p. 5.
[236] DeLong, "The Basal Ganglia.", p. 862.
[237] Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 31.
[238] Duvoisin, p. 5.
[239] Chow and Cummings, p. 31.
[240] "Neurologic Disorders.", p. 763.
[241] Duvoisin, p. 5. Duvoisin notes that there is some question about comparing different countries, as methodologies may differ.

races.[242] As the disease remains idiopathic, it cannot be stated with absolute certainty the means by which a patient develops the condition. Twin studies have demonstrated that there may be a genetic component to the development of the disease,[243] but this is not absolute or beyond question;[244] current literature suggests that approximately 10% of Parkinson's is familial with specific genetic defects.[245]

Course of the illness

The difficulty of properly diagnosing Parkinson's disease is in its gradual onset. The symptoms may appear so mild as to escape detection or to be misdiagnosed. Duvoisin notes that "The beginning is usually so insidious and the progression so gradual that it can rarely be dated precisely…Indeed, progression is so gradual that little if any changes can be seen from one year to the next."[246] The duration of Parkinson's varies, but most patients tend to be disabled within 5 to 7.5 years;[247] only one-third of Parkinson's patients have mild and/or stable symptoms beyond 10 years. [248] Parkinson's is highly debilitative and ultimately fatal; death typically results from aspiration pneumonia or other infections.[249]

Parkinson's disease can be exacerbated by dementia, and there is a significant amount of comorbidity dependent upon age. Victor and Ropper note that while the frequency is dependent upon the patients selected and type of testing, an average of 10-15% of Parkinson's patients have

[242] Victor and Ropper, *Principles of Neurology*, p. 1128
[243] Victor and Ropper, p. 1128
[244] Duvoisin, *Parkinson's Disease: A Guide for Patient and Family*, p. 6
[245] L. Dawson and Ted M. Dawson, "Parkinson's Disease," in *Fundamental Neuroscience*, ed. Larry R. Squire, et al. (New York: Academic Press, 2003), 830, p. 830
[246] Duvoisin, p. 5
[247] Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 31; , "The Basal Ganglia.", p. 862. Victor and Ropper cite two earlier studies to defend the 7.5 year prognosis (Victor and Ropper, *Principles of Neurology*, p. 1131).
[248] Victor and Ropper, p. 1131
[249] "Neurologic Disorders.", p. 763

comorbid dementia, and this frequency increases to nearly 65% above 80 years of age.[250] They also note that MRI studies have demonstrated lesions in cerebral white matter in patients with comorbid dementia.[251] Harrison, et al., found a similar rate of comorbid dementia.[252] Kaplan and Sadock suggest that the rate is higher (20-30%), and that measurable cognitive impairment can be found in an additional 30-40% of patients.[253]

Rates of Depression

As noted above, Parkinson's affects the levels of dopamine in the corpus striatum by damaging and inhibiting the dopaminergic qualities of the substantia nigra; dopamine insufficiency has been linked to depressed mood and anhedonia.[254] Chow and Cummings note that:

> According to a two-pronged model of dopamine deficiency in depression, dysfunction in the dopamine-mediated reward system induces anhedonia (the 'reduced capacity to experience reward or pleasure'), while the dysfunction of serotonin pathways exacerbates abnormalities in the levels of dopamine via neurotransmitter interactions. Anhedonia may reflect a dysfunction in positive reward mechanisms, whereas negative reward systems are mediated by opioid receptors and may contribute to the dysphoric component of depression.[255]

Parkinson's disease leads to depletion in both dopamine and serotonin, as such, it can lead to depression by multiple pathways.

The rate of depression in Parkinson's disease varies greatly, dependent upon the source, scope of depressive criteria, diagnostic criteria employed (e.g., DSM-III-TR or DSM-IV-TR), and reporting methodology. Chow and Cummings cite one community-based study which

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[250] Victor and Ropper, *Principles of Neurology*, p. 1130
[251] Victor and Ropper, p. 1130
[252] John E. Harrison, Isabel Stow, and Adrian M. Owen, "Parkinson's Disease," in *Cognitive Deficits in Brain Disorders*, ed. John E. Harrison and Adrian M. Owen (London: Martin Dunitz, 2002), 197-215, p. 202
[253] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 331
[254] Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 35. They also note that Parkinson's disease causes decreased levels of norepinephrine (noradrenaline), serotonin, and acetylcholine. Norepinephrine and serotonin depletion have also been linked with depressed mood.
[255] Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 35

reported depressive symptoms meeting DSM-III-TR standards for Major Depressive Disorder in only 7.7% of the patients, but noted that approximately 46% demonstrated mildly depressive symptoms.[256] They also note that "the estimated rate of clinically important depression in patients with PD (37%) is more than twice that seen in medically ill patients."[257] They note further that the rate of major depression increases dramatically in cases of advanced Parkinson's patients who are not receiving antiparkinsonian treatment; 40-60% experience major depression "regardless of the duration of illness or degree of physical disability."[258] Kaplan and Sadock rate the incidence of comorbid depression at 30% overall,[259] and 50% in elderly Parkinson's patients.[260] Lavretsky and Kumar suggest that the prevalence falls somewhere between 20-40%; half of these patients meet the criteria for Major Depressive Disorder, while the remainder exhibit features consistent with minor depression and Dysthymia."[261] Victor and Popper set the incidence of major depression at approximately 25-30%.[262] Chwastiak, et al., note that the use of a structured psychiatric interview demonstrated in several studies yielded comorbid depression in 41-49% of patients with Parkinson's disease.[263] Harrison, et al., note a range of 23-50% of comorbid depression, dependent upon the cohort and testing methodology.[264] In light of the above figures, for the purposes of this dissertation it seems safe to set a prevalence of comorbid depression at approximately 40%.

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[256] Chow and Cummings, "Depression in Parkinson's Disease: Pharmacological Characteristics and Treatment.", p. 31.
[257] Chow and Cummings, p. 31.
[258] Chow and Cummings, p. 32.
[259] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 87.
[260] Kaplan and Sadock, p. 1295.
[261] Lavretsky and Kumar, "Clinically Significant Nonmajor Geriatric Depression.", p. 298.
[262] Victor and Ropper, *Principles of Neurology*, p. 1612; see also p. 1132.
[263] Chwastiak, et al., "Depressive Sumptoms and Severity of Illness in Multiple Sclerosis: Epidemiologic Study of a Large Community Sample.", p. 1866.
[264] Harrison, Stow, and Owen, "Parkinson's Disease.", p. 202.

The difficulty of assessing rates of depression is complicated by the symptoms – they can mimic or be masked by the signs of Parkinson's. Weakness and fatigue are present in both depression and Parkinson's, and apathy and anergia can produce bradykinesia. Further, the most prevalent therapeutic intervention for Parkinson's – L-dihydroxyphenylalenine (L-dopa) – can produce psychiatric dysfunction, including depression.[265] Further, they note that:

> Paucity of movement, unchanging attitudes and postural sets, and a slightly stiff and unbalanced gait may be observed in patients with an anergic or hypokinetic ('retarded') type of depression. Since as many as 25 to 30 percent of parkinsonian patients are depressed, the separation of these two conditions may be difficult. The authors have seen patients who were called parkinsonian by competent neurologists whose movements became normal when antidepressant medication or electroconvulsive therapy was given.[266]

Clearly it seems necessary that proper diagnostic measures must include assessment for depression in addition to medical examination and history of the illness.

Treatment

As noted above, Parkinson's is a fatal condition – at present, there is no curative treatment. Rather, "the goal of treatment is to relieve symptoms and keep the patient functional as long as possible."[267] The current standard of care is treatment with L-dihydroxyphenylalenine (L-dopa). L-dopa has produced the most consistent results and has been demonstrated to be the most effective treatment for Parkinson's disease in comparison with other drug therapies (dopamine agonists, anticholinergics, and antihistamines).[268] L-dopa acts as a dopamine replacement; while the levels of dopamine are depleted, the remaining nigral cells can still convert L-dopa to dopamine (the neurons in the striatum that respond to the nigral cells remain

[265] Victor and Ropper, *Principles of Neurology*, p. 1612
[266] Victor and Ropper, p. 1132
[267] "Neurologic Disorders.", p. 764
[268] Victor and Ropper, p. 1133

receptive to dopamine).[269] L-dopa is frequently administered with carbidopa, which maximizes the amount of L-dopa to reach the nigral cells (L-dopa can be converted to dopamine in peripheral tissues (outside the nigral cells), which can decrease the total amount of dopamine that reaches the striatum).[270]

While this therapy is effective for most patients, there are several concerns. First, the number of dopamine-converting nigral cells diminishes, and the striatal target neurons become overly receptive to dopamine. The consequence of this is a decreased response to L-dopa and excessive dyskinesias.[271] Second, the most common consequence of L-dopa therapy is the inevitable appearance of secondary disorders. Victor and Ropper note:

> The most common and troublesome effects of L-dopa, requiring individualization of therapy, are end-of-dose failure, the "on-off" phenomenon, and the induction of involuntary movements – restlessness, head wagging, grimacing, lingual-labial dyskinesias, and choreoathetosis [continuous movements] and dystonia [painful posturing] of the limbs, neck, and trunk. *The on-off phenomenon* refers to an unpredictable change in the patient, in a matter of minutes or from one hour to the next, from a state of relative mobility to one of complete or nearly complete immobility. These disorders eventually appear in about 75 percent of patients within 5 years. Above a certain daily dose, which varies from patient to patient, very few patients escape these effects, forcing a reduction in dosage. [italics in the original; definitions are the author's][272]

Further, L-dopa itself can cause psychiatric reactions in 15 to 25% of patients (particularly the elderly) ranging from depression to psychosis, delusions, and suicide.[273]

Alternate therapies have been suggested that target acetylcholine instead of dopamine. Anticholinergic medications seek to decrease the amount of acetylcholine in relation to dopamine. This treatment is less common, but it appears to manage the symptoms of patients who cannot tolerate L-dopa as their primary pharmacological intervention. Victor and Ropper

[269] Victor and Ropper, *Principles of Neurology*, p. 1133
[270] Victor and Ropper, p. 1133; "Neurologic Disorders", p. 764
[271] Victor and Ropper, p. 1133
[272] Victor and Ropper, p. 1135
[273] Victor and Ropper, p. 1134

note that successful results can be achieved with a variety of anticholinergics in treating Parkinson's (trihexyphenidyl (Artane), benzotropine mesylate (Cogentin), Amantadine (an antiviral agent with anticholinergic properties), and ethopropazine (Parsidol)).[274] Some antihistamines are also employed to augment treatment, such as diphenhydramine and phenindamine.[275] There are, however, side effects with anticholinergic treatment – memory impairment, confusion, auditory/visual hallucinations, and bradyphrenia (slowing mental processes) – which can deter their use.[276]

A third alternative has been surgical treatment – a neurosurgeon will create lesions on specific areas of the brain. This surgery involves stereotactic damage either to the globus pallidus or subthalamic nucleus, and tends to be used in the treatment of patients with advanced disease or who respond poorly to medications.[277,278] The surgery has the best results on patients with unilateral tremor or rigidity, and has the least effect on "postural imbalance and instability, paroxysmal akinesia, bladder and bowel disturbances, dystonia, and speech difficulties."[279] The long-term benefits of surgery are unclear – the procedure only relieves some of the symptoms, while the disease itself continues to advance. Patients have found that some benefits are lost within a year or two.[280]

[274] Victor and Ropper, *Principles of Neurology*, p. 1135
[275] Victor and Ropper, p. 1136
[276] Victor and Ropper, p. 1135
[277] Victor and Ropper, p. 1136
[278] Delong, "The Basal Ganglia.", p. 863
[279] Victor and Ropper, p. 1136
[280] Victor and Ropper, p. 1136

DEMENTIA

Dementia is a disorder frequently associated with advancing age. It frequently involves a progressive loss of mental faculties with a variety of etiologies.[281] Victor and Ropper describe it as follows:

> [T]he term dementia (literally, an undoing of the mind) denotes a deterioration of all intellectual or cognitive functions with little or no disturbance of consciousness or perception. Implied by the word is the idea of a gradual enfeeblement of mental powers in a person who formerly possessed a normal mind.[282]

The most common associated sign of dementia is a progressive loss of short and long-term memory.[283] Butler, et al., note that these changes can also hallmark a demonstrable shift in other aspects of the individual, including changes in "judgment, intellectual abilities, activities of daily living, and in some cases, personality"[284]; Kaplan and Sadock expand upon this list, noting changes in "general intelligence, learning and memory, language, problem solving, orientation, perception, attention and concentration, judgment, and social abilities."[285] The DSM notes that these deficits cannot simply be minor inconveniences – they must represent a significant impairment in social or occupational functioning, and they must represent a significant change in the person's ability to function.[286] The specific changes are variable dependent upon the type of dementia and areas of the brain which are affected. Rapp and Bachevalier note that there are, at

[281] This is not a universal statement – some dementias are static, instead of progressive, and there are dementias that are reversible.
[282] Victor and Ropper, *Principles of Neurology*, p. 432
[283] This is, perhaps, a misnomer – in psychology, the information processing approach to memory argues that the "storage" aspect of memory is unchanged, but the "retrieval" of memory is not what it once was.
[284] Robert N. Butler, Myrna I. Lewis, and Trey Sunderland, *Aging and Mental Health: Positive Psychosocial and Biomedical Approaches* (Boston: Allyn and Bacon, 1998), p. 129. They note several other types of non-Alzheimer's dementia, dividing them generally into categories of those due to structural causes (e.g., vascular dementia, Parkinson's disease, Lewy Body dementia, brain tumors, etc.), metabolic causes (e.g., hypothyroidism, Hypercalcemia, chronic alcohol use, pernicious anemia, hepatic encephalopathy, etc.), or infectious causes (e.g., tuberculosis, Creutzfeld-Jacob, Neurosyphilis, HIV-related dementia, etc.).
[285] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328
[286] *DSM*, p. 148

present, approximately fifty disorders known to cause dementia.[287] Most of these dementing illnesses are of insidious onset – the change is gradual and protracted; often it is difficult to note when the first symptoms appeared.

Knowledge deficits seem to be a natural consequence of advancing age – neurons die off at a significant rate without any environmental causes. Some sources rate this loss as high as nearly 20 million neurons per year.[288] This loss, however, does not immediately translate into deficits in "crystallized" intelligence. This is to say that knowledge we have acquired over the years is not affected. The deficit appears, however, when we attempt to learn something new – "fluid" intelligence diminishes with aging.[289] As such, the onset of senility is not an inevitable result of advancing age. Neuron death is selective – there are regions of the brain that remain relatively unaffected by aging – the cerebellum and the brainstem incur very little to no neuronal death, while the cortex can experience daily losses of up to 50 thousand neurons.[290] Fortunately, however, there are several million "reserve neurons" which can take up the responsibilities of the dead cells. The only instances in which abnormal loss occurs usually involves trauma (injury), infection, neurological insult (e.g., stroke), or nutritional deficits (e.g., malnutrition, alcoholism, etc.).[291]

Dementia typically manifests itself first in the fifth or sixth decade of life, followed by a progressive deterioration.[292] The age and rate of deterioration vary with the type of dementia encountered, and approximately 10 to 15 percent of dementias can be stopped or reversed.[293,294]

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[287] R. Rapp and Jocelyne Bachevalier, "Cognitive Development and Aging," in *Fundamental Neuroscience*, ed. Larry R. Squire, et al. (Boston: Academic Press, 2003), 1167-200, p. 1195.
[288] The National Institute of Neurological Disorders and Stroke and The National Institute of Mental Health, "The Brain and Nervous System.", p. 97 [hereafter NIND & NIMH].
[289] NIND & NIMH, "The Brain and Nervous System.", p. 97
[290] NIND & NIMH, p. 97.
[291] NIND & NIMH, p. 97.
[292] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 341.
[293] Kaplan and Sadock, p. 341.

In patients with Alzheimer's disease – the most common degenerative disease of the brain[295] – the mean survival rate is approximately eight years (this can vary, and has been estimated to vary considerably – as short as one year and up to twenty years).[296] In general, Alzheimer's disease involves a series of neurological lesions – an abnormal aggregation of senile plaques and neurofibrillary tangles in and around the neurons of several CNS structures, particularly the neocortex, entorhinal region, hippocampus, amygdala, nucleus basalis, anterior thalamus, and areas of the brain stem.[297] These lesions have significant neurological consequences – entorhinal cortex, medial temporal cortex, and hippocampal abnormalities have been suggested as the basis of the memory impairment in Alzheimer disease. [298] These abnormalities are complicated by the functional impairments associated with insult to the association areas of the neocortex and the basal forebrain cholinergic system.[299] Behavioral and affective disturbances have been linked to insult to the limbic cortex (the limbic system is the basis of emotion), amygdala, thalamus, and the brain stem. [300]

In addition to the lesions described above, marked brain atrophy is suggestive of Alzheimer's disease. The brain normally atrophies with age, but this tends not to be inimical of any particular dysfunction (see the discussion of normal neuronal death above). When this atrophy is severe and diffuse, however, credence is lent to a suggested diagnosis of Alzheimer's disease. [301] Certainty in the diagnosis of Alzheimer's disease normally is only obtained post-mortem, when an examination of the patient's brain is possible. However, criteria have been

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[294] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328
[295] Victor and Ropper, *Principles of Neurology*, p. 1109
[296] Kaplan and Sadock, p. 341
[297] L. Price, "Aging of the Brain and Dementia of the Alzheimer Type," in *Principles of Neural Science*, ed. Eric R. Kandel, James H. Schwartz and Thomas M. Jessell (New York: McGraw-Hill Health Professions Division, 2000), 1149-61, p. 1153
[298] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1154
[299] Price, " p. 1154
[300] Price, " p. 1154
[301] Victor and Ropper, p. 1109

proposed for research purposes and to establish inclusive/exclusive criteria for diagnosis. Victor and Ropper note:

> The National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the Alzheimer's Disease and Related Diseases Association (ADRDA) have proposed the following diagnostic criteria: (1) dementia defined by clinical examination, the Mini-Mental Scale, the Blessed Dementia Scale, or similar mental status examination; (2) the age of patient (over 40 years); (3) deficits in two or more areas of cognition and progressive worsening of memory and other cognitive functions – such as language, perception, and motor skills (praxis); (4) absence of disturbed consciousness; and (5) exclusion of other brain diseases…Using these criteria, the correct diagnosis is achieved in more than 85 percent of patients.[302]

Drislane, et al., also suggest that "elevated tau protein and low Ab-42 levels in the cerebrospinal fluid (CSF) have been suggested as early diagnostic markers for AD,"[303] while Price suggests that CT and MRI scans of the medial temporal lobe may reveal structural abnormalities and decreased blood flow, both of which can be predictive of Alzheimer's.[304] Victor and Ropper also note that in the later stages of the disease, MRI scans can reveal profound atrophy of the hippocampus, which they suggest lends credence to an Alzheimer's diagnosis.[305] There are, however, difficulties in the differential diagnosis of Alzheimer's disease. They note that the most frequent barrier to appropriate diagnosis is distinguishing Alzheimer's disease and other dementias from late-life depression – both can involve symptoms like anergia, memory loss, sleep disturbances, etc. This is further complicated by occasional comorbidity – both dementia and depression can be found in the same patient, which makes it difficult to distinguish which conditions is responsible for which neurological/psychosocial symptoms.[306]

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[302] Victor and Ropper, *Principles of Neurology*, p. 1112-3
[303] Drislane, et al., p. 95-6
[304] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1152-3
[305] Victor and Ropper, p. 1113
[306] Victor and Ropper, p. 1117

Epidemiology

Alzheimer's disease is the most common cause of dementia in older populations,[307] but the prevalence of dementia varies with age and location. In 1992, the National Institute of Neurological Disorders and Stroke and the National Institute of Mental Health estimated that 5% of the population age 65 and over are severely demented, with an addition 10% mildly to moderately demented.[308] Half of these dementias were of the Alzheimer type.[309] In 1996, Murray and Lopez suggested a point prevalence of 888 cases of dementia per 100,000 people in established market economies (resulting in a prevalence of about 0.88%),[310] compared with a point prevalence of 357 cases per 100,000 people globally (0.36% globally).[311] Seven years later, Zubenko, et al., suggested that the rate of Alzheimer's disease is significantly higher – 8% to 15% of the current population over the age of 65, with more expected as the current population ages.[312] Bird's estimates fall within this range,[313] while other studies assess the overall rate of Alzheimer's as significantly higher – many have suggested prevalence as high as 17%-40% of the population over the age of 80-85.[314] This dissertation follows the epidemiological assessment by Kaplan and Sadock, as it represents a middle ground in the estimates provided. They suggest that:

> In the United States, approximately 5 percent of people older than age 65 have severe dementia and 15 percent have mild dementia. Of those older that age 80,

[307] S. Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease," *American Journal of Psychiatry* 160, no. 5 (May 2003): 857-66, p. 857
[308] NIND & NIMH, "The Brain and Nervous System.", p. 98
[309] NIND & NIMH, p. 98
[310] Murray and Lopez, *Global Health Statistics: A Compendium of Incidence, Prevalence and Mortality Estimates for Over 200 Conditions*, p. 616
[311] Murray and Lopez, p. 618
[312] Zubenko, et al., p. 865
[313] D. Bird, "Alzheimer's Disease and Other Primary Dementias," in *Harrison's Principles of Internal Medicine*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 2391-98, p. 2391
[314] "Neurologic Disorders.", p. 743; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196; Bird, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1152; G. Waxman and Jack deGroot, *Correlative Neuroanatomy* (Norwalk: Appleton & Lange, 1995), p. 280-1; Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328.

> approximately 20 percent have severe dementia. Of all patients with dementia, 50 to 60 percent have the most common type of dementia, dementia of the Alzheimer's type. About 5 percent of everyone who reaches age 65 has dementia of the Alzheimer's type, compared with 15 to 25 percent of everyone age 85 or older.[315]

The incidence of Alzheimer's disease increases with age, and several sources have suggested that it will pose a growing concern in the next 15 years. [316]

Most of those affected by Alzheimer's disease are over the age of sixty, but cases have been found in every period of life.[317] The physiological manifestations of Alzheimer's disease parallel the neurological effects of other conditions, suggesting a similar process occurs in a younger age with individuals affected by trisomy 21 (Down syndrome – see below) and other familial forms of early-onset Alzheimer's disease. Further, risk may be sex-linked, as women have a disproportionate incidence of the disease.[318]

Physiology

Alzheimer's disease presents with both behavioral and physiological signs and symptoms. Looking at the behavioral/psychosocial manifestations first, the patient typically presents with signs of dementia (significant enough to interfere with social/occupational functioning), an insidious onset of symptoms (progressive, irreversible, and so gradual as to make difficult any particular date of onset), and an absence of other potential dementias (established by history, physical examination, and testing).[319] The physiology of Alzheimer's disease is marked by reductions in brain volume, widening of sulci (fissures in the brain), decrease in the levels of specific neurotransmitters, and the presence of neurofibrillary tangles and neuritic plaques – atrophy is particularly marked in the cortex, hippocampus, amygdala, and

[315] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328.
[316] Butler, Lewis, and Sunderland, *Aging and Mental Health: Positive Psychosocial and Biomedical Approaches*, p. 133; Drislane, et al., *Blueprints in Neurology*, p. 95; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 257.
[317] Victor and Ropper, *Principles of Neurology*, p. 1109.
[318] Victor and Ropper, p. 1109.
[319] NIND & NIMH, "The Brain and Nervous System.", p. 104.

thalamus.[320] The effects on the hippocampus lead to perhaps the most marked psychosocial changes – because the hippocampus is a central structure in memory formation and recall, changes in its function can adversely affect a person's ability to recall old and form new memories.[321] Alzheimer's disease is normally of insidious onset, and memory disturbances are generally the first signs to appear. There are also concomitant changes in neurochemistry – deficits can be found in the enzymes which synthesize the neurotransmitters dopamine, norepinephrine, and, to a lesser extent, acetylcholine.[322] Similar to the reduction in hippocampal volume, a decreased level of choline acetyltransferase, the enzyme which synthesizes acetylcholine, can affect memory – a common side effect of anticholinergic medications.

Zubenko, et al., suggest that because the etiology of Alzheimer's disease is heterogeneous, it may be more accurate to describe our current understanding of Alzheimer's not as a particular disease, but rather as a syndrome.[323] The cause of Alzheimer's is presently idiopathic, but a variety of explanations have been proposed, including "neurochemical factors, such as deficiencies of the neurotransmitters acetylcholine, somatostatin, substance P, and norepinephrine; environmental factors, such as aluminum and manganese; trauma; genetic factors; and viral factors such as slow-growing central nervous system viruses."[324] There appear to be genetic ties to some forms of Alzheimer disease, linked to the interaction of amyloid precursor protein (APP) and Apolipoprotein alleles (ApoE – discussed below), referred to as the amyloid hypothesis. Victor and Ropper suggest that there are legitimate questions which remain

[320] Drislane, et al., *Blueprints in Neurology*, p. 95-6; Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 329; Victor and Ropper, *Principles of Neurology*, p. 1113; Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1151.
[321] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 257; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2392.
[322] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1151; Waxman and deGroot, *Correlative Neuroanatomy*, p. 279-280.
[323] Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease.", p. 857.
[324] "Neurologic Disorders.", p. 744.

to be answered concerning the presence of neurofibrillary tangles and neuritic plaques – depending upon the pathogenesis of Alzheimer's, the tangles and plaques may be secondary characteristics of the condition, rather than causative characteristics.[325] Regardless, they note that the amyloid hypothesis remains the most compelling explanation.

Plaques/Tangles/Amyloid Protein

In addition to the behavioral signs of dementing illness, the hallmark pathophysiological signs of Alzheimer disease are atrophy of the brain, neurofibrillary tangles, and neuritic plaques, each of which is summarized below. In general, there seems to be a dysfunction of neuroanatomy and neurochemistry; there is a marked dysfunction of tau protein in the case of neurofibrillary tangles and of amyloid beta-protein in the case of the neuritic plaques.[326] The tangles and plaques can adversely affect the inter- and intracellular communication of neurons. The tangles and plaques seem to preferentially affect the hippocampus and entorhinal cortex initially, later spreading to other areas of the CNS. These plaques and tangles are normal signs of aging, but when present in an abnormal amount, are indicative of a deeper pathology.[327] Similar processes of neuritic plaques and neurofibrillary tangles occur in other diseases, but specific attention has been paid to trisomy-21 (Down Syndrome), due to the presence of immature plaques in youth and mature neuritic plaques and tangles in the patient's thirties and forties.

[325] Victor and Ropper, *Principles of Neurology*, p. 1115.
[326] Drislane, et al., *Blueprints in Neurology*, p. 96; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2391.
[327] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393; Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 329; Waxman and deGroot, *Correlative Neuroanatomy*, p. 282; Victor and Ropper, *Principles of Neurology*, p. 1114.

Neurofibrillary tangles [NFTs] are intercellular aggregations of phosphorylated tau protein that surround the neural nucleus and extend towards the dendrites.[328] Bird notes that tau "may function to assemble and stabilize the microtubules that convey cell organelles, glycoproteins, and other important materials through the neuron."[329] The tangles consist of paired helical filaments constructed from cytoskeletal protein in the neuron. Alterations in cytoskeletal structure can disrupt the cells ability to traffic material and neurotransmission, resulting in dysfunction and neuronal death.[330] These filaments are not explicitly linked with Alzheimer's – there are several neurological disorders in which they manifest (e.g., progressive supranuclear palsy, frontotemporal dementia, etc.).[331] However, in combination with the neuritic plaques (see below), they are one of the hallmark signs of Alzheimer's disease.

Neurofibrillary tangles and neuritic plaques (see below) seem to be normal signs of aging. The key difference (i.e., the mark of a dementing illness) is found in the number of these tangles and plaques; the higher the number, the greater the severity of the dementia.[332] These filaments are so numerous and dense that the cell body can appear swollen with a displaced nucleus.[333] The NFTs normally first affect the hippocampus and entorhinal cortex, suggesting that they initially target the anatomical bases of declarative memory, leading to a disruption of memory of ongoing events.[334] As the disease progresses, the tangles expand into areas responsible for "language, semantic knowledge, abstract reasoning, and other capacities."[335]

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[328] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 257-8; J. Selkoe, "Amyloid Protein and Alzheimer's Disease," in *The Scientific American Book of the Brain* (Guilford: The Lyons Press, 1999), 251-62, p. 253; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196-7; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393; Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1154.
[329] Bird, p. 2393.
[330] Rapp and Bachevalier, p. 1196-7.
[331] Selkoe, p. 253; Victor and Ropper, p. 1114.
[332] Pliszka, p. 257-8.
[333] Rapp and Bachevalier, p. 1196-7.
[334] Rapp and Bachevalier, p. 1197
[335] Rapp and Bachevalier, p. 1197

One of the hallmark signs of Alzheimer disease is the presence of neuritic plaques; these plaques are protein fragments – approximately 40 amino-acid long collections of amyloid-beta-protein (Aβ protein).[336] These plaques were first observed by Alois Alzheimer scattered throughout the cortices of his patient. These neuritic plaques are extracellular; unlike the neurofibrillary tangles, they are found outside of the neuron, typically surrounded by dystrophic neurites.[337] The dystrophic neurites are malformed dendrites and axons (sections of the neuron which receive synaptic transmissions and which carry the nerve impulse, respectively; axons release neurotransmitters into a junction between the axon and the dendrite of another neuron to transmit an inhibitory or excitatory signal). At the core of these plaques, one typically finds microglial cells (inflammatory cells), while around the outside one typically finds reactive astrocytes – glial cells found most often in injured areas of the CNS.[338] The core of the plaque can contain several different forms of Aβ, including $A\beta_{42}$ which is prone to aggregation.[339]

There are a variety of diseases that result in deposition of amyloid filaments (called amyloidoses); these filaments are constituted by differing types of protein fragments, dependent upon the disease in question. [340] Selkoe argues that while the identity of the cell types which produce beta-APP [amyloid precursor protein] are unknown, plausible causes can be found in platelets, endothelial cells, neurons, and glial cells. He suggests:

> If these cells synthesized either excess or altered forms of beta-APP, some of those molecules might be broken down by an alternative enzymatic pathway, thereby liberating large fragments that contain the amyloid beta-protein. Over time, I suspect, these fragments are further cleaved by proteases to release the intact amyloid beta-protein, which then accumulates in the extracellular spaces of the brain in the form of diffuse plaques. Because of local tissue factors in the

[336] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 252; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196.
[337] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1155; Rapp and Bachevalier, p. 1196.
[338] Selkoe, p. 253
[339] Rapp and Bachevalier, p. 1196
[340] Selkoe, p. 253

> cerebral cortex and other brain regions important for cognitive function, a minority of these diffuse plaques becomes increasingly filamentous and compact. The addition of so-called beta-amyloid-associated proteins – some of which have already been identified – and the activation of nearby microglia and astrocytes probably contribute to the maturation of the plaques.[341]

Waxman and deGroot note that Aβ is neurotoxic, and suggest that "recent studies suggest (but have not yet conclusively proved) that deposition of abnormal amyloid beta protein triggers neuronal death in Alzheimer's disease."[342] APP has been shown to have a variety of effects, both neurotrophic and neuroprotective.[343]

Several authors have noted a link between trisomy-21 (Down Syndrome[344]) and Alzheimer's disease.[345] Upon their death, many individuals with Down Syndrome exhibit similar plaques in their CNS; while they are not the mature neuritic plaques exhibited in Alzheimer's disease, it has been suggested that these patients ultimately would exhibit similar pathophysiology.[346] These plaques occur at a significantly younger age than the average age of onset for Alzheimer's disease; Victor and Ropper note their presence in the third and fourth decade of life,[347] while Selkoe has noted them in patients in their teens and twenties.[348] It has been suggested (and will be explored in greater detail below) that specific alleles of apolipoprotein on chromosome 21 can be linked with a greater prevalence of Alzheimer's disease.

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[341] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 261.
[342] Waxman and deGroot, *Correlative Neuroanatomy*, p. 282.
[343] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393.
[344] Down Syndrome occurs when an individual inherits an extra copy of chromosome 21; it results in profound physical and cognitive dysfunctions. Down syndrome is one of the potential complications of older motherhood.
[345] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 257; Victor and Ropper, *Principles of Neurology*, p. 1118.
[346] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 257.
[347] Victor and Ropper, *Principles of Neurology*, p. 1118.
[348] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 257.

Neurochemistry

There are a variety of chemical changes that occur as a result of Alzheimer's disease. Neurotransmission is fundamentally affected, as both the enzymes which synthesize neurotransmitters as well as the neurotransmitters themselves are reduced. Specific deficiencies have been noted in the neurotransmitters acetylcholine, somatostatin, substance P, norepinephrine, serotonin, cholecystokinin, and corticotrophin, as well as the enzyme choline acetyltransferase (which synthesizes acetylcholine).[349] Along with the neurochemical deficits, functional losses abound, included inhibited uptake and functioning of a variety of receptors (e.g., nicotinic cholinergic receptors, GABAergic functions, etc.).[350] The cholinergic deprivation is noteworthy, given that a common side effect of anticholinergic medications (medications regulating and reducing the level of acetylcholine) is memory loss. This anticholinergic effect is compelling, but not causative. Pliska notes that therapies aimed at increasing the cholinergic functions of the brain have been shown to be of limited benefit in the treatment of Alzheimer's (see below), but that they do not alter the prognosis or course of the illness.[351] Structural changes have been attributed to the loss of specific neurotransmitters – Zubenko, et al., note that:

> Projections from the dorsal and median raphe nuclei provide extensive serotonergic innervation of the forebrain. The noradrenergic cells of the locus ceruleus project axons widely to both the neocortex and the hippocampus. Alzheimer's disease is associated with the loss of neuronal cells from both of these nuclei, and a substantial fraction of those that remain develop neurofibrillary tangles. The neurochemical correlates of this process include decrements in the levels of these amine neurotransmitters and their metabolites, their respective biosynthetic enzymes, and in the presynaptic reuptake of both neurotransmitters in their projection areas.[352]

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[349] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 259; "Neurologic Disorders.", p. 744; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393; Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 329-30; Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease.", p. 858; Victor and Ropper, *Principles of Neurology*, p. 1115-6.
[350] Victor and Ropper, p. 1115-6.
[351] Pliszka, p. 259.
[352] Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease.", p. 858.

Hence, in the process of disrupting cognitive function and memory recall, Alzheimer's disease affects the cells ability to synthesize, transmit, and receive both neurotransmitters as well as their enzyme precursors.

Genetics

Significant research has gone into explorations of the human genome in explaining the prevalence of Alzheimer's disease. Current research is focusing on marker genes at several locations (alleles on chromosomes 1, 12, 14, 19, and 21).[353] Evidence suggests that up to 40%-70% of Alzheimer patients have a familial history of the disease, lending credence to the genetic hypothesis.[354] Further, twin studies have demonstrated that there is a significantly higher correlation of Alzheimer's disease between monozygotic versus dizygotic twins (43 percent versus 8 percent, respectively).[355] Price notes that, in general:

> Five principal genetic risk factors for Alzheimer disease are known: (1) mutations in the APP gene on chromosome 21; (2) mutations in the *presenilin 1* gene on chromosome 14; (3) mutations in the *presenilin 2* gene on chromosome 1; (4) alleles for the *ApoE* positioned on the proximal long arm of chromosome 19; and (5) possibly a mutation or polymorphism in a gene on chromosome 12 that encodes alpha-2 macroglobulin. Any of the first three mutations is associated with early onset of the disorder in the third through sixth decades.[356]

It is also possible that these genetic risk factors may interact – for example, it is possible that an allele of ApoE may influence production of beta-amyloid; dysfunctions in this process could give rise to the neuritic plaques and neurofibrillary tangles evidenced in Alzheimer's disease and older adults with Down syndrome (see below). Contrary to Price, however, Victor and Ropper

[353] The National Institute of Neurological Disorders and Stroke and The National Institute of Mental Health, "The Brain and Nervous System.", p. 102; Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1156.
[354] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328-9.
[355] Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 328-9.
[356] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1156.

suggest that the only potential genetic marker for Alzheimer disease is the abnormal apolipoprotein allele.[357]

Presenilins/APP Mutations

Close attention is currently being paid to the genes responsible for the production of the amyloid precursor protein. Selkoe suggests that there is a causative role between beta-amyloid abnormalities and the onset of Alzheimer diseases.[358] Bird notes that there is correlation between APP abnormalities on chromosome 21 and Alzheimer's disease in adult Down syndrome patients, supporting Selkoe's optimistic assessment.[359] In fact, multiple mutations in the APP gene have been correlated with a small percentage of the cases of Alzheimer disease.[360] Despite Selkoe's optimism and Bird's support, the role of normal amyloid in the brain is unclear; however, it has been suggested that while "the normal amyloid stimulates neuron proliferation and enhances the effects of nerve growth factors, the abnormally long amyloid produces the mutation that causes neuron death."[361]

In addition to the APP abnormalities noted above, two candidate genes called presenilins have been suggested as causative of early onset forms of Alzheimer disease. All of the known mutations of presenilin-1 and presenilin-2 increase the production and deposit of the longer amyloid protein.[362] Price notes that approximately 30% of the cases of early-onset Alzheimer disease are linked to the presenilin-1 gene, but the mechanism by which the mutations in presenilins lead to Alzheimer disease is not yet known.[363] There are, however, specific proteins

[357] Victor and Ropper, *Principles of Neurology*, p. 1117.
[358] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 256.
[359] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393.
[360] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 258.
[361] Pliszka, p. 258.
[362] Pliszka, p. 258.
[363] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1156.

derived from presenilin mutations which are present in higher quantities in affected patients than in controls.[364]

Apolipoprotein E

Links between the gene apolipoprotein-E (a regulator of lipid metabolism, found on chromosome 19) and Alzheimer disease are currently being explored.[365] Several sources have linked ApoE ε4 and the formation of neurofibrillary tangles and neuritic plaques, suggesting that ApoE it forms binds with tau protein or APP.[366] Bird notes that "in a group of AD patients, approximately 40 to 65% have at least one ε4 allele, a highly significant difference compared with controls,"[367] but cautions that "ε4 is neither necessary nor sufficient as a cause of AD."[368] There are several alleles of the ApoE gene; the most prevalent form in Caucasians is the ε3 allele, found in 75% of the Caucasian population, followed by alleles ε4 and ε2 at 15% and 10%, respectively.[369] The risk of Alzheimer's disease appears to be dependent upon which allele an individual inherits. It has been suggested that individuals with one copy of the ε4 allele carry a risk four times higher than the general population, while individuals with two copies of the ε4 allele are eight times as likely to develop Alzheimer's.[370] Several sources have noted that the ApoE ε4 allele is not a proven cause of later development of Alzheimer disease, but it can be noted as a risk factor. [371]

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[364] Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1156
[365] Drislane, et al., *Blueprints in Neurology*, p. 95; Pliszka, *Neuroscience for the Mental Health Clinician*, p. 258-9; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1197-8.
[366] Pliszka, *Neuroscience for the Mental Health Clinician*, p. 258-9; Victor and Ropper, *Principles of Neurology*, p. 1116; Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2394.
[367] Bird, p. 2394
[368] Bird, p. 2394
[369] Pliszka, p. 258-9
[370] Rapp and Bachevalier, p. 1197-8
[371] Bird, p. 2394; Victor and Ropper, p. 1116.

Familial Alzheimer's Disease

Part of the current research into the genetics of Alzheimer's disease involves exploring family-linked early-onset forms of Alzheimer's disease. Mutations in the presenilin-1 gene (PS-1, on chromosome 14) are causally linked with approximately 40% to 70% of early-onset familial Alzheimer's disease (FAD).[372] PS-1 mutations tend to cause onset around the age of 45 with a mean duration of 6-7 years, while presenilin-2 (PS-2, on chromosome 1) mutations tend to cause onset of the disease around the age of 53 years with a duration of 11 years.[373] The normal functions of the proteins these genes encode and the effect they have on Alzheimer's disease are currently unknown.[374] Mutations in the gene is associated with higher levels of Aβ amyloid, suggesting a link with the production of APP[375] and, perhaps, with a higher risk for developing neuritic plaques. Defects on chromosome 21 involving the beta-APP (amyloid precursor protein) have also been linked to familial Alzheimer's disease, despite it's correlation with only a small percentage of proved familial cases.[376]

Course of the Illness

As indicated above, Alzheimer's disease results in progressive loss of executive and motor function, culminating in a state of extreme dependency. It is characterized by an insidious loss of memory, judgment, and emotional stability. Difficulties in assessing the onset of Alzheimer's disease are magnified by the likelihood of a significant "pre-clinical" phase in which the symptoms are unnoticed or not present.[377] Estimates of its duration range from 4-12

[372] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2393; Victor and Ropper, *Principles of Neurology*, p. 1116.
[373] Bird, p. 2393; Victor and Ropper, p. 1116.
[374] Bird, p. 2393.
[375] Bird, p. 2393.
[376] Waxman and deGroot, *Correlative Neuroanatomy*, p. 282; Victor and Ropper, p. 1116.
[377] Victor and Ropper, p. 1111.

years[378] to as high as 10-15 years[379], but wide variations exist – Bird for example notes that the duration can vary from 1 to 25 years.[380] Some patients show a consistent pattern of decline, while others have periods in which their degeneration plateaus; ultimately, however, the end is the same. The duration is also contingent upon outside factors such as when the diagnosis is made (i.e., early, middle, or late stage), as well as the type of care the patient receives after the diagnosis is made. Attention paid to details such as proper nutrition and hydration, as well as attention to skin integrity in bed-bound patients, hygiene, wound care, urinary tract infections, etc. can lengthen the survival of an Alzheimer's patient. These are especially important as death frequently results not from the dementing illness itself, but rather from secondary infections, medical conditions, or malnutrition, thus, as Butler states "people do not die of Alzheimer's *per se*…they die *with* Alzheimer's."[381]

Early

The early stages of Alzheimer's disease typically involve a mild loss of memory and difficulty learning new tasks.[382] Most frequently this is attributed to age, and is not given much notice, unless it is pronounced or happens to occur at roughly the same time as a head injury, medication change, or illness.[383] These memory difficulties typically involve recent memories, older memories remain relatively intact. The patient's behavior typically becomes more docile and apathetic – patients tend to exhibit difficulty in motivating themselves to do novel or

[378] Selkoe, "Amyloid Protein and Alzheimer's Disease.", p. 252.
[379] NIND & NIMH, "The Brain and Nervous System.", p. 103.
[380] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2392.
[381] Butler, Lewis, and Sunderland, *Aging and Mental Health: Positive Psychosocial and Biomedical Approaches*, p. 137.
[382] NIND & NIMH, p. 102; "Neurologic Disorders.", p. 744; Bird, p. 2391-2; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196.
[383] Victor and Ropper, *Principles of Neurology*, p. 1110.

unfamiliar tasks, and may withdraw from social situations.[384] Personal hygiene may also begin to deteriorate.[385]

Middle

As the disease progresses to the middle stage, there is increased impairment in cognitive function (e.g., in abstract thinking), memory, and language.[386] The patient is no longer able to work and requires supervision. Routine behaviors may be unaffected, as well as social interaction and superficial conversation, but there is frequent difficulty in expression – the patient finds it increasingly difficult to say what they want to say. Verbal instructions may not be followed, and it may not be clear as to whether the instructions were forgotten or misunderstood.[387] Behavioral shifts occur – the patient may become more irritable or aggressive, with symptoms worsening towards the evening (referred to as "sundowning").[388] Personal attention may wane, and the patient may require increased assistance with dressing and personal hygiene.

Late

Late in the course of the illness, profound and broad cognitive and psychosocial impairment exists.[389] The patient is frequently bed-ridden, and there occasional seizures in about 5% of patients.[390] They require increasing assistance with bathing, dressing, voiding, and other activities of daily life.[391] The patient may forget how to use common objects while retaining the

[384] NIND & NIMH, "The Brain and Nervous System.", p. 102.
[385] "Neurologic Disorders.", p. 744.
[386] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2392; NIND & NIMH, p. 102; Victor and Ropper, *Principles of Neurology*, p. 1110.
[387] Victor and Ropper, p. 1110.
[388] NIND & NIMH, p. 102, Drislane, et al., *Blueprints in Neurology*, p. 95.
[389] Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196.
[390] Victor and Ropper, p. 1111.
[391] Bird, p. 2392.

motor coordination necessary to use the objects.[392] It has been this author's experience that simple objects like utensils present profound challenges to the Alzheimer's patient – the spoon is used backwards, upside-down, like a knife, etc. Dressing the patient appropriately is a challenge, as the patients in this author's care will resist efforts to disrobe and wash soiled clothing, preferentially putting clean clothing directly over the soiled clothing, becoming very agitated in the process. Basic language skills are gone, as well as recent memory and most remote memories.[393] The patient frequently is mute and incontinent as bowel and bladder control is frequently lost.[394] Only the most habitual and automatic actions are retained – patients cannot execute verbal, written, or imitative commands (termed "ideational" and "ideomotor apraxia").[395] It is at this state that secondary infections and comorbid illnesses most frequently cause death.[396]

Treatment

There is presently no curative treatment for Alzheimer's disease.[397] Treatment currently revolves around management of secondary conditions (medical problems, psychosocial and behavioral problems, etc.) as well as therapies designed to address specific physiological difficulties (e.g., hyperbaric oxygenation of the brain) and extend plateau periods (e.g., postponing memory loss for a few months). Identification of psychosocial stressors can prevent unnecessary escalation of a patient's behavioral problems, as well as facilitating care and allowing for more effective treatment.[398] Some patients show a modest response to

[392] Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1196; Victor and Ropper, *Principles of Neurology*, p. 1110.
[393] Rapp and Bachevalier, p. 1196
[394] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2392
[395] Victor and Ropper, p. 1110
[396] NIND & NIMH, "The Brain and Nervous System.", p. 103; Drislane , et al., *Blueprints in Neurology*, p. 95.
[397] Rapp and Bachevalier, p. 1198; "Neurologic Disorders.", p. 745; Bird, p. 2394; Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1158.
[398] NIMD & NIMH, "The Brain and Nervous System.", p. 105-6.

acetylcholinesterase inhibitors (12-20%), which can stabilize – but not improve – a patient's condition for several months, but these drugs are not beneficial in later stages.[399] Future strategies are examining hormone replacement, reduction of oxidative damage, as well as prevention of the formation of the neuritic plaques.[400]

Comorbid Depression

There is a significant rate of comorbid psychiatric dysfunction in conjunction with dementing disorders, principally involving depression and psychosis.[401] Rates of comorbid depression historically have varied. Class, et al., suggest that comorbid depression can be found in approximately 10% to 30% of patients with Alzheimer's disease, [402] while Zubenko, et al., note historical studies with a range as great as 0% to 86%.[403] Zubenko, et al., suggest generally that approximately half of Alzheimer's patients experience major depression in their lifetime, and that approximately one-third develop major depression after the onset of cognitive impairment.[404] They further suggest that "the major depressive syndrome of Alzheimer's disease may be among the most common mood disorders of late life."[405] Class, et al., note that it is likely that depression may go unnoticed in long-term care settings, because the concomitant passivity is not seen as a behavioral problem.[406] It has been this author's experience that most commitments (voluntary and involuntary) from long-term care facilities are due to overt

[399] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2394; Rapp and Bachevalier, "Cognitive Development and Aging.", p. 1198; Price, "Aging of the Brain and Dementia of the Alzheimer Type.", p. 1158; Victor and Ropper, *Principles of Neurology*, p. 1117.
[400] Rapp and Bachevalier, p. 1198; Price, p. 1158.
[401] Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease.", p. 857-8; Bird, p. 2394.
[402] Class, Schneider, and Farlow, "Optimal Management of Behavioural Disorders Associated with Dementia.", p. 24.
[403] Zubenko, et al., p. 857-8.
[404] Zubenko, et al., p. 865.
[405] Zubenko, et al., p. 865.
[406] Class, Schneider, and Farlow, p. 24.

behavioral problems (e.g., violence towards staff or over threats of suicide), rather than unvoiced depression.

Victor and Ropper note that depression in Alzheimer's makes it difficult to posit what symptomology is attributable to which condition, as memory impairment is common to both.[407] Further, depression has a variety of consequences that complicate an existing medical diagnosis. Zubenko, et al., note that it "increases the suffering of patients and their families, produces excess disability, promotes institutionalization, and hastens death."[408] They note, however, that there may be mitigating factors – the physiological changes that occur may actually prevent future episodes of depression:

> This last observation [that patients with Alzheimer's disease have relative preservation of the cholinergic neurons in the basal forebrain than innervate the hippocampus and neocortex] is interesting in the context of Alzheimer's disease, since the progression of the central cholinergic deficit that occurs in this disorder may interact with the pathophysiology of depression to limit the development of major depressive episodes in later stages of this disorder. Several lines of evidence from published autopsy studies suggest that these neuropathological and neurochemical correlates of major depression in Alzheimer's disease have relative specificity for this mood disorder and differ from those associated with psychosis, exposure to psychotropic medications, and the neurodegeneration in Alzheimer's disease more generally.[409]

As such, while the disease may lead to depressive disorders initially, as the patient is both aware of the loss and of the progressive nature of the disease, as it progresses the physiological damage it inflicts may salve the psychosocial and emotional trauma of earlier stages.

[407] Victor and Ropper, *Principles of Neurology*, p. 1612.
[408] Zubenko, et al., "A Collaborative Study of the Emergence and Clinical Features of the Major Depressive Syndrome of Alzheimer's Disease.", p. 857-8.
[409] Zubenko, et al., p. 858.

Other Behavioral Disturbances

There are other complicating features of Alzheimer's disease – other aberrant behaviors can develop as the disease progresses.[410] In addition to the depression noted above, Alzheimer patients may develop auditory/visual hallucinations, paranoid ideation, and other psychoses.[411] It has been this author's experience that Alzheimer disease markedly increases aggressiveness in patients, particularly after sunset, accompanied by verbalized statements of fear, anger, and/or accusations of persecution or theft of property. Butler, et al., suggest that "up to 70% of patients with dementia develop symptoms of psychosis within seven years of getting the diagnosis."[412] Concomitant psychosis exacerbates incapacity and cognitive dysfunction in Alzheimer's disease, and compounds the difficulties in intellectual function already experienced, leading to conditions referred to as "excess disability states."[413]

STROKE

An overview of a cerebral vascular accident (CVA, or stroke) must begin with a quick summary of neural vasculature; the brain consumes a significant portion of the total blood volume – approximately 18% of the total blood volume goes to the brain,[414] despite its minute percentage of the total body weight. The brain also uses approximately one-fifth of all the oxygen absorbed in the lungs, and requires a constant supply of oxygen – irreparable brain damage occurs within five minutes of cerebral anoxia.

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[410] Butler, Lewis, and Sunderland, *Aging and Mental Health: Positive Psychosocial and Biomedical Approaches*, p. 132; NIMD& NIMH, "The Brain and Nervous System.", p. 106-7.
[411] Bird, "Alzheimer's Disease and Other Primary Dementias.", p. 2392; Kaplan and Sadock, *Synopsis of Psychiatry, Eighth Edition*, p. 335-7.
[412] Butler, Lewis, and Sunderland, p. 132.
[413] NIMD & NIMH, p. 106-7.
[414] Waxman and deGroot, *Correlative Neuroanatomy*, p. 172.

The brain is supplied by two sets of arteries – two internal carotid and two vertebral arteries.[415] The internal carotid arteries supply the cerebrum with blood and oxygen, with each carotid dividing into an anterior and middle cerebral artery. The middle cerebral artery supplies the bulk of the lateral surface of the cerebrum, while the anterior cerebral artery supplies the majority of the sagittal surface of the brain (brain regions on the midline area of the cerebral hemispheres).[416] Occlusion of a cerebral artery results in contralateral deficits (e.g., occlusion of the right carotid artery causes weakness and sensory loss on the left side of the body). Occlusion of the anterior cerebral artery affects sensorimotor functions in the lower part of the body, while occlusion of the middle cerebral artery affects sensorimotor functions in the upper parts of the body.[417] The vertebral artery supplies the cerebellum and the ventral portions of the cerebrum, but it undergoes several name changes along the way.[418] The two vertebral arteries fuse into a single artery called the basilar artery. The basilar artery splits into two posterior cerebral arteries at the base of the circle of Willis (a roughly circular collection of cerebral and communicating artery pairs near the pituitary gland), and branches out throughout the cerebellum and ventrolateral cerebra. These branches are referred to as the superior cerebellar arteries, anterior inferior cerebellar arteries, and the posterior inferior cerebellar arteries.[419] Occlusion of the basilar artery results in total blindness, as it supplies blood to the visual cortex. Occlusion of one vertebral artery may not have any resultant deficits, as additional blood is supplied by the other vertebral artery.[420] Occlusion of the cerebellar arteries result in brainstem damage.[421] There are

[415] Stephen Goldberg, *Clinical Neuroanatomy Made Ridiculously Simple* (Miami: MedMaster, Inc., 2000), p. 8. It should be noted that these two paragraphs are simply a quick summary of the most major features of neurovasculature – the actual neurophysiology is significantly more complex.
[416] Goldberg, *Clinical Neuroanatomy Made Ridiculously Simple*, p. 9.
[417] Goldberg, p. 9.
[418] Goldberg, p. 8.
[419] Goldberg, p. 13.
[420] Goldberg, p. 12.
[421] Goldberg, p. 13.

a host of veins that return blood to the heart – perhaps the most familiar is the jugular vein, into which the other veins empty. There are, however, veins emptying from all over the pia mater, cerebrum and cerebellum. Less attention will be given to blood return, as the primary pathophysiology of stroke involves the arteries.

When this blood flow is compromised, either by damage or hemorrhage, the patient suffers an ischemic or hemorrhagic accident.[422] Ischemia causes a rapid loss of ATP, resulting in a buildup of potassium in the extracellular space in the central nervous system. This overabundance of potassium depolarizes the neurons, resulting in massive release of a variety of neurotransmitters (including excitatory neurotransmitters), which leads to a toxic buildup of calcium, a proposed explanation of neuronal cell death.[423] If this ischemic attack is brief, the damage may be reversible; if, however, it is prolonged, the neurons die and neurologic deficits result (the specific deficit will depend on the location of the infarct; see below). Several terms are used to refer to cerebral vascular accidents – transient ischemic attack (TIA), stroke, and reversible ischemic neurologic deficit (RIND) – but Drislane, et al., note that these labels are "arbitrary and somewhat meaningless," as there is no clear correlation for the rough time interval they are meant to denote.[424]

Most infarcts are caused by vascular narrowing or occlusion, cerebral embolism, prolonged hypertension, hypotension, drugs (illicit drugs like cocaine and amphetamines, as well as prescription drugs like heparin or warfarin), vascular malformation, disease (like amyloid angiopathy, which affects blood vessel walls), or inflammation.[425] Most of the atherosclerotic

[422] Waxman and deGroot, *Correlative Neuroanatomy*, p. 178-9; Drislane, et al., *Blueprints in Neurology*, p. 105; Victor and Ropper, *Principles of Neurology*, p. 822.
[423] Waxman and deGroot, p. 181.
[424] Drislane, et al., p. 105
[425] Waxman and deGroot, p. 181; Drislane, et al., p. 106-7.

changes in cerebral arteries occur in the neck and brain.[426] It is a progressive condition, and has been linked to disturbances in metabolism and blood pressure.[427] In the case of embolism or occlusion, the blood supply is interrupted by a foreign particle (clot, fat, tumor, etc.) which plugs or constricts the artery, resulting in infarction and cell death – a common cause is atrial fibrillation.[428] Further, high blood pressure can result in vasculature change; the blood vessel can become distended in smaller branches – an increase in blood pressure can then breach these distensions, resulting in cerebral hemorrhage.[429] The resultant hemorrhage can damage surrounding tissue as the blood clots, compressing the tissue beneath, causing further neurological deficits.[430] Hemorrhages can occur in a variety of locations in the brain and the protective tissues surrounding it.

In addition to the bone of the skull, the brain is protected and supported by several layers of meninges. The outermost layer is referred to as the dura mater, a tough, somewhat flexible tissue. Beneath the dura matter is the arachnoid layer, separated from the lowest layer – the pia mater – by the subarachnoid space, a porous layer with filaments resembling a spider's web. The pia mater conforms to the folds and fissures of the brain, both covering the brain's exterior surface as well as the surface of the ventricular spaces within. Hemorrhages in the subarachnoid space are normally found in individuals with normal blood pressure; the aneurysms leading to hemorrhage usually are the result of minor trauma, congenital defects, or infections. Complications of subarachnoid hemorrhages can lead to increased pressure on the brain and

[426] Waxman and deGroot, *Correlative Neuroanatomy*, p. 183.
[427] Waxman and deGroot, p. 183.
[428] Waxman and deGroot, p. 183; Drislane, et al., *Blueprints in Neurology*, p. 106; Victor and Ropper, *Principles of Neurology*, p. 822.
[429] Waxman and deGroot, p. 185-6; Drislane, et al., p. 107.
[430] Waxman and deGroot, p. 185-6.

cerebral infarction.[431] Subdural hemorrhage (hemorrhage between the brain surface and the dura) can result from even minor trauma (children and adults with cerebral atrophy are at particular risk because of age-related vascular properties). While the bleeding from the hemorrhage may be reabsorbed, there are risks that it may become encapsulated or calcified. [432] Epidural hemorrhage (outside the dura) typically results from major trauma and/or skull fracture resulting in an arterial bleed. If this bleeding is not controlled, it can put pressure on the brain, leading to significant neurological deficits.[433] Profound neurologic deficits can also result from low blood pressure (hypotension) as a result of hypovolemia or cardiac failure – this type of damage tends to be more perfused than that of a localized ischemic event, low blood volume affects a variety of areas at once.[434]

The symptoms of a stroke can vary greatly. Individuals suffering a cerebral vascular accident can present with the most commonly associated symptoms like slurred speech, blurred vision, hemiparesis or weakness, or headache, but can also experience symptoms as diverse as seizures, progressive focal deficits, aphasia of several sorts, sensorimotor deficits or loss, neglect of one side, auditory/visual/olfactory/tactile deficits, subjective distortion of space, limb alienation, memory loss, ataxia, apraxia, vomiting, decreased arousal, neck stiffness, and syncope. In fact, the type of symptoms presenting can aide the diagnosing clinician greatly in isolating the brain region affected by the stroke (e.g., sensorimotor loss would localize the stroke to the contralateral sensorimotor cortex; the type of aphasia experienced would suggest

[431] Drislane, et al., *Blueprints in Neurology*, p. 106; Waxman and deGroot, *Correlative Neuroanatomy*, p. 186; Victor and Ropper, *Principles of Neurology*, p. 823.
[432] Waxman and deGroot, p. 186.
[433] Waxman and deGroot, p. 186-7.
[434] Drislane, et al., p. 106; Victor and Ropper, p. 822.

localization to either Broca's or Wernicke's areas, etc.), as well as whether the patient had suffered arterial versus ventricular hemorrhage.[435]

Epidemiology

Stroke is a major cause of death in the United States, only heart disease and cancer are more lethal. Victor and Ropper note that "among all the neurologic diseases of adult life, the cerebrovascular ones clearly rank first in frequency and importance. At least 50 percent of the neurologic disorders in a general hospital are of this type."[436] Waxman and deGroot estimate that every year in the United States there are 500,000 strokes (175,000 of the patients will die), and that 15% of admissions to chronic care facilities are due to stroke.[437] Drislane, et al., and Smith, et al., offer a more morbid prognosis, suggesting that approximately 25%-33% of individuals suffering a stroke will die as a result (about 187,500 to 250,000 people each year).[438] The World Health Organization estimates the prevalence of stroke at 9,467,000 cases in established market economies in the year 2000, with a global prevalence of 30,872,000 – of these, 870,000 and 5,580,000 will die, respectively.[439] Robinson and Smith, et al., warn that the prevalence of stroke increases with each decade of life.[440]

Drislane, et al., note that "risk factors for stroke include older age, male sex, family history, hypertension, diabetes, smoking, hypercholesterolemia, heavy alcohol use, and cardiac

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[435] Drislane, et al., *Blueprints in Neurology*, p. 107.
[436] Victor and Ropper, *Principles of Neurology*, p. 821.
[437] Victor and Ropper, p. 821.
[438] Drislane, et al., p. 105; Wade S. Smith, Stephen L. Hauser, and J. Donald Easton, "Cerebrovascular Diseases," in *Harrison's Principle's of Internal Medicine*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 2370-91, p. 2370.
[439] World Health Organization, *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020*, p. 655 [Established Market Economies]; Health Organization, *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020*, p. 657 [Global].
[440] G. Robinson, "Neuropsychiatric Consequences of Stroke," *Annual Review of Medicine* 48 (1997): 217-29, p. 217; Smith, Hauser, and Easton, "Cerebrovascular Diseases.", p. 2370.

or peripheral vascular disease."[441] Victor and Ropper add that atrial fibrillation, hyperlipidemia, hypercoagulable state diseases, and the use of birth control pills also contribute to stroke liability.[442]

Course of the Illness

The onset of a stroke is acute – while the stenosis, occlusion, or aneurysm leading up to the event may develop over time, the resulting deficits are of rapid onset (thrombotic strokes tend to be of more insidious progression than embolic strokes).[443] These lesions tend to lead to focal disturbances of cognitive or sensorimotor function (e.g., blurred vision, slurred or absent speech, hemiplegia, etc.[444] The specific means of neuronal destruction can vary – some intracranial bleeds result in pressure on the cortices, some result in cerebral anoxia, etc.; many strokes result in cerebral hemorrhage, which can yield cerebral dysfunction "through a variety of mechanisms, including destruction of tissue, mass effect, and compression of blood vessels leading to ischemia."[445] Cerebral infarction involves two pathological processes – oxygen and glucose deficits from occlusion, and changes in cellular metabolism which destroys cellular membranes.[446] The severity of the stroke varies widely – some result in neurologic dysfunctions so slight as to not warrant medical attention, some are so severe as to result in hemiplegia, coma, and death.[447]

The resultant symptoms of the stroke can locate the location of the accident.[448] Left hemisphere lesions tend to produce contralateral weakness and neglect, aphasia, and potential

[441] Drislane, et al., *Blueprints in Neurology*, p. 105.
[442] Victor and Ropper, *Principles of Neurology*, p. 825.
[443] Victor and Ropper, p. 823-4.
[444] Waxman and deGroot, *Correlative Neuroanatomy*, p. 181.
[445] Drislane, et al., p. 106.
[446] Victor and Ropper, p. 827-8.
[447] Victor and Ropper, p. 823-4.
[448] Victor and Ropper, p. 824.

deficits in reading, writing, and calculation.[449] Right hemisphere lesions tend to result in contralateral weakness, neglect, and cortical sensory deficits.[450] Left posterior cerebral artery lesions tend to result in contralateral visual field deficits, difficulty naming colors presented visually, and potential contralateral sensory deficits and difficulty reading.[451] Right posterior cerebral artery lesions tend to result in contralateral sensory/visual losses and neglect.[452] Vertebrobasilar artery occlusion can result in both cerebellar and brain-stem dysfunction. The patient may experience vertigo, diplopia, nystagmus, extremity weakness, ataxia, vomiting, headache, or sensory dysfunctions.[453] Pure motor strokes (usually resulting from a lesion in the interior capsule or pons) and pure sensory strokes (usually resulting from thalamic lesions) involve contralateral deficits in motion/control or sensation, respectively, but tend not to produce other deficits (e.g., a pure motor stroke should not present with sensory deficits and vice versa).[454] Computerized tomography (CT) scans are useful in finding localizations of the neurological deficits, but magnetic resonance imagine (MRI) tends to be more useful, as bone can obscure CT results.[455]

As noted above, not all strokes require medical attention or surgical resolution. Sometimes the neurological deficits reverse themselves within a few hours to days; more often, however, the effects can take weeks to months to reverse themselves, and can leave the patient considerably disabled.[456] The severity of the attack is dependent upon the degree of the occlusion and the duration of the attack. Victor and Ropper note that:

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[449] Drislane, et al., *Blueprints in Neurology*, p. 110.
[450] Drislane, et al., p. 110.
[451] Drislane, et al., p. 110.
[452] Drislane, et al., p. 110.
[453] Drislane, et al., p. 110.
[454] Drislane, et al., p. 110.
[455] Victor and Ropper, *Principles of Neurology*, p. 824.
[456] Victor and Ropper, p. 824.

> [C]ertain major vessels (carotid, vertebral, and less often a cerebral artery at its origin) can sometimes be occluded with little or no disturbance of neurologic function, and at autopsy there may be complete integrity of the tissue in the territory of the occluded vessel. Moreover, if infarction has occurred, it usually involves a zone that is smaller than the anatomic territory of supply of the artery in question. The margins of the infarct are hyperemic, being nourished by meningeal collaterals, and here there is only minimal or no parenchymal damage. The necrotic tissue swells rapidly, mainly because of excessive intracellular and intercellular water content. Since anoxia also causes necrosis and swelling of cerebral tissue (although in a different distribution), oxygen lack must be a factor common to both infarction and anoxic encephalopathy. Obviously the effects of ischemia, whether functional and reversible or structural and irreversible, depend on its degree and duration.[457]

They note that other influences can affect the severity of the injury.[458] Specifically, the speed of the occlusion and the patient's blood pressure can affect the opening of collateral channels; these channels can shunt off excess fluid, preventing anoxia and pressure damage. Altered blood viscosity and osmolality may also play a role, but their influence is difficult to infer presently. Further, each patient has variations in their neural vasculature, and as such, there may be anatomical factors which alleviate or worsen the effect of a stroke. Current research is examining the role of excitatory neurotransmitters during ischemia. It has been found that glutamate and aspartate are released by ischemic cells, which then excite surrounding cells, leading to massive influxes of sodium and calcium, which it is suggested are responsible for cell injury and death.[459]

Depression and Cognitive Dysfunction

Depression following stroke is a serious comorbid illness. Starkstein and Robinson note that "post-stroke depression is one of the most frequent complications of brain injury, and approximately 40% of patients with an acute stroke lesion will show either a major or a minor

[457] Victor and Ropper, *Principles of Neurology*, p. 828.
[458] Victor and Ropper, p. 825-7.
[459] Victor and Ropper, p. 828.

depression."[460] After analyzing patient data from a variety of sources, Robinson has found two types of depression following a stroke – major depression, occurring in up to 25% of post-stroke patients, and minor depression, occurring in approximately 10% to 30% of post-stroke patients.[461] There is a significantly higher mortality among stroke patients experiencing depression. Previous studies have indicated that individuals experiencing a depressive disorder following a stroke are 3.5 times more likely to die during the first two to three years following the stroke.[462] Robinson notes two key factors that influence post-stroke depression – treatment with antidepressant medication and the location of the stroke lesion (see below for discussions of each).[463]

Kneebone and Dunmore suggest that there are significant problems in attempting diagnosis of depression in patients who have had a stroke. They note that:

> Features of some strokes, such as pathological emotionalism, lethargy and memory impairment, can suggest depression when in fact they are common results of such neurological assault. Assessment is most obviously a problem with those who have communication difficulties due to dysphasia or other cognitive losses. In the latter case lack of awareness can also compromise PSD assessment. For some individuals this is so frank that it extends to a complete denial of the stroke itself. Up to 45% of those having suffered a stroke approximately 1 year previously have been found to minimize their mood disorder substantially when their responses were compared with a structured clinical interview modified to allow better assessment of depression in brain-damaged individuals. Such lack of awareness has lead writers in the area to suggest specialist assessment strategies are required for PSD, perhaps involving a family member or caregiver in the process.[464]

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[460] Sergio E. Starkstein and Robert G. Robinson, "Depression in Cerebrovascular Disease," in *Depression in Neurologic Disease*, ed. Sergio E. Starkstein and Robert G. Robinson (Baltimore: The Johns Hopkins University Press, 1993), 28-49, p. 46.
[461] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 218.
[462] Robinson, p. 222.
[463] Robinson, p. 218.
[464] I. Kneebone and Emma Dunmore, "Psychological Management of Post-Stroke Depression," *The British Journal of Clinical Psychology* 39 (March 2000): 53-65, p. 54.

Lyketsos, et al., suggest that two causal explanations for post-stroke depression need to be delineated.[465] First, post-stroke depression can result from the psychosocial impairments of the disease process. Second, the individual may feel depressed because of the specific pathophysiology of the neurological insult – critical pathways affecting mood may become damaged, giving rise to feelings of depression. They suggest that both models are compelling and warrant further research. Nicholl, et al., found similar results, and suggest that the potential split between the endogenous model and the exogenous model of post-stroke depression makes diagnosis much more complicated.[466] Predicting post-stroke depression is not easy; Lyketsos, et al., note that the manifestation secondary to stroke does not have a unique clinical manifestation – when present, it tends to parallel the manifestation of a primary depression.[467] Post-stroke depression can be masked by some of the typical signs associated with physical infirmity, e.g., fatigue, anergia, etc. Starkstein and Robinson note that difficulty with activities of daily living (ADLs) is not a good predictor of post-stroke depression. Once post-stroke depression developed, however, the individual's recovery of ADL functioning suffered.[468] Ramasubbu, et al., echo this ADL deficiency, noting that "post-stroke depression during the acute phase of recovery adversely affects functional abilities, short-term physical therapy outcome, and long-term functional recovery."[469]

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[465] G. Lyketsos, et al., "Does Stroke Cause Depression?" *The Journal of Neuropsychiatry and Clinical Neurosciences* 10, no. 1 (Winter 1998): 103-07, p. 103.
[466] Catherine R. Nicholl, et al., "Cognitions and Post-Stroke Depression," *The British Journal of Clinical Psychology* 41 (September 2002): 221-31, p. 222.
[467] Lyketsos, et al., "Does Stroke Cause Depression?", p. 104.
[468] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 39.
[469] Rajamannar Ramasubbu, et al., "Functional Impairment Associated with Acute Poststroke Depression: The Stroke Data Bank Study," *The Journal of Neuropsychiatry and Clinical Neurosciences* 10, no. 1 (Winter 1998): 26-33, p. 26.

Epidemiology

Starkstein and Robinson suggest that the prevalence of depression in stroke patients in acute stroke units, general hospitals, rehabilitation centers, and community studies falls between 30% to 50%.[470] Ramasubbu, et al., note that approximately 20% to 50% of stroke patients may have a demonstrable depression;[471] of these 20% to 50%, only 15% of depressed stroke patients were identified as carrying the disease, and only 10% of those patients received antidepressant medication.[472] Ramasubbu, et al., also note that white patients tend to be more depressed than other ethnic group (as per the United States Data Bank). Hachinski suggests that depression occurs in approximately 50% of stroke patients.[473] Van de Wag, et al., note that the reported rates of post-stroke depression (PSD) vary significantly, with a range from 25% to 79%, with the most conservative estimate of the incidence being 500,000 people with PSD in 1999 alone.[474] They suggest a general prevalence of 35% in the post-stroke patient population.[475] Nicholl argue that despite the wide disparity in the prevalence rate, it is important to identify and treat depression in its early stages.[476] Tateno, et al., place the rate of post-stroke depression at 23%-40% of patients, while 7% to 57% of patients with a traumatic brain injury experience depression.[477]

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[470] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 30.
[471] Ramasubbu, et al., "Functional Impairment Associated with Acute Poststroke Depression: The Stroke Data Bank Study.", p. 27.
[472] Ramasubbu, et al., p. 29.
[473] Hachinski, "Post-Stroke Depression, not to Be Underestimated," *The Lancet* 353 (May 22 1999): 1728, p. 1728.
[474] FB van de Wag, DJ Kulk, and GJ Lankhorst, "Post-Stroke Depression and Functional Outcome: A Cohort Study Investigating the Influence of Depression on Functional Recovery from Stroke," *Clinical Rehabilitation* 13 (1999): 268-72, p. 269.
[475] van de Wag, Kulk, and Lankhorst, "Post-Stroke Depression and Functional Outcome: A Cohort Study Investigating the Influence of Depression on Functional Recovery from Stroke.", p. 268.
[476] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 221-2.
[477] Amane Tateno, Yuichi Murata, and Robert G. Robinson, "Comparison of Cognitive Impairment Associated With Major Depression Following Stroke Versus Traumatic Brain Injury," *Psychosomatics* 43, no. 4 (July/August 2002): 295-301, p. 295.

Starkstein and Robinson suggest that there are two principle risk factors for post-stroke depression. Specifically subcortical atrophy and genetic markers for depression were found to be more frequent in patients with left hemisphere lesions.[478] Nicholl, et al., expand on this list, noting that:

> Other risk factors that have been associated with an increased prevalence of post-stroke depression are: subcortical atrophy, structural brain asymmetries, lesion volume, female gender, family or previous history of mood disorder, neuroticism trait, younger age, greater impairment in activities of daily life, impaired social support (especially support from spouse), and negative life events. Some of them independently increase the prevalence of diagnosed depression following stroke, while others have been shown to have an additive effect. Thus, the cause of PSD probably includes several mechanisms that vary with premorbid as well as post-stroke factors.[479]

Paradiso and Robinson note that gender may play a role in the manifestation of depression following a stroke, simply by virtue of a greater overall prevalence of depression in the female population:

> One might expect, given these gender-based differences in brain organization, that brain injury would affect women and men differently. A common psychopathological manifestation after stroke is mood disorder. Mood disorders following stroke have been proposed as a model to study affective illness in general. If women are biologically more vulnerable to developing a depressive disorder, one might expect that a precipitating factor common to men and women (such as a cerebrovascular accident) would provoke more depression in females than males. One would also predict post-stroke depression to be associated with clinical variables indicating a greater biological predisposition in females. On the other hand, depression in males might be associated with nonbiological factors such as severity of physical and psychosocial impairment.[480]

Women, they note, tend to respond more negatively towards impairments affecting social interaction, while men tend to respond more negatively to impairments in motor coordination. They suggest that as a result of specific correlates in the data, women with post-stroke depression

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[478] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 41-2.
[479] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 429.
[480] Sergio Paradiso and Robert G. Robinson, "Gender Differences in Poststroke Depression," *The Journal of Neuropsychiatry and Clinical Neurosciences* 10, no. 1 (Winter 1998): 41-47, p. 41-2.

may respond more to psychological or somatic therapies, while men may respond more to physical therapy.[481]

Major versus Minor Depression

Starkstein and Robinson note that both major and minor depression tend to manifest following stroke, but the duration and remission vary.[482] They link the presence of major depression with the location of the lesion (see below). Paradiso and Robinson follow the DSM discussion of major versus minor depression and dysthymic disorder, noting that both present for the same duration, but that minor depression presents with fewer symptoms of major depression.[483] While noting some disparities in the literature,[484] they suggest that there are differences in the physiognomy of the younger patients, stating that they "found that minor depression was associated with younger age, left-hemisphere lesion location, and more caudal hemisphere lesions than found in nondepressed control patients. The more caudal lesion location also held true when minor depression and major depression were compared."[485] They also found the prevalence of comorbid anxiety to be higher than non-depressed controls and lower than patients with major depression.

Functional Impairment

Several studies have explored the link between depression and functional impairments following a stroke. Robinson noted that in a controlled study equalized in terms of lesion variables, demographics, rehabilitation efforts, and acute stroke treatment, stroke patients with depression showed significantly less recovery in activities of daily living than non-depressed

[481] Paradiso and Robinson, "Gender Differences in Poststroke Depression.", p. 46-7.
[482] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 29.
[483] Paradiso and Robert G. Robinson, "Minor Depression After Stroke: An Initial Validation of the DSM-IV Construct," *The American Journal of Geriatric Psychiatry* 7, no. 3 (Summer 1999): 244-51, p. 244
[484] Paradiso and Robinson, "Minor Depression After Stroke: An Initial Validation of the DSM-IV Construct.", p. 245
[485] Paradiso and Robinson, p. 248

stroke patients.[486] Ramasubbu, et al., noted that this was a controversial topic, in that the literature to date yielded inconsistencies, but found similar ADL deficits in depressed patients (but did not find an ADL difference between major and minor depression).[487] They also noted that age negatively affected ADL performance, and that a significant contributor to functional impairment is motor loss.[488] Van de Wag, et al., found a prevalence of 35% for post-stroke depression, and noted that the results of their study supported a hypothesis linking the presence of a depressive disorder with increased disability on two rehabilitation tests at both admission and follow-up.[489] They also noted a significant negative effect of depression on the rehabilitation process.[490]

Lesion Location/Physiological Correlation

Several studies have been conducted exploring a possible link between the location of the neurological insult and any resulting affective disorder.[491,492] Robinson, et al., state that:

> The clinical correlates of post-stroke depression include younger age, greater impairment in activities of daily living, social impairment, premorbid personality, prior personal or family history of psychiatric disorders, nonfluent aphasia, cognitive impairment, and enlarged ventricle-to-brain ratio. Perhaps the most interesting and controversial correlate of post-stroke depression, however, has been lesion location.[493]

They note that previous studies have suggested an inverse relation between the severity of the depressive episode and the distance of the lesion from the anterior edge of the left frontal lobe.[494]

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[486] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 220-222.
[487] Ramasubbu, et al., "Functional Impairment Associated with Acute Poststroke Depression: The Stroke Data Bank Study.", p. 29.
[488] Ramasubbu, et al., p. 32.
[489] van de Wag, Kulk, and Lankhorst, "Post-Stroke Depression and Functional Outcome: A Cohort Study Investigating the Influence of Depression on Functional Recovery from Stroke.", p. 270.
[490] van de Wag, Kulk, and Lankhorst, p. 272.
[491] Daniel Rogers, "Functional Depression Viewed as Neurologic Disease," in *Depression in Neurologic Disease*, ed. Sergio E. Starkstein and Robert G. Robinson (Baltimore: The Johns Hopkins University Press, 1993), 13-27, p. 23.
[492] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 38.
[493] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 218-9.
[494] Robinson, p. 219.

Others have found similar evidence for the influence of lesion location. Lyketsos, et al., have noted that left prefrontal cortical abnormalities can be found in patients with primary depression. They argue that "with the exception of the neuropathology of depression in Alzheimer's disease, this correlation of severity of depression with proximity of the stroke lesion to the frontal pole is perhaps the most consistently replicated clinical-pathological correlation in psychiatry."[495] Further, MRI scans have identified hyperintensitites in subcortical white matter and in the basal ganglia, which they suggest shows overlap in the anatomical bases of primary and post stroke depression.[496] Paradiso and Robinson found comorbid depression in both left and right frontal lobe lesions, but found a greater instance of depressive disorders in patients with left-sided lesions, and greater severity of symptoms when the lesions were closer to the anterior edge of the left frontal lobe.[497] They also note that minor depression was associated with more posterior-oriented lesions, which may involve a different mechanism of depression.[498] In a previous study, Paradiso and Robinson also suggested a possible gender link in post-stroke depression, as they found a greater instance of left hemispheric lesions in women than in men.[499] In a 2-year longitudinal study, Nicholl, et al., found the severity of the depressive episode to be linked to lesion location, but they also suggested that the severity is time-linked, with greater depression occurring within six months of the accident.[500]

Starkstein and Robinson also suggested monoaminergic disruption (i.e., lesions affecting the structures receiving the neurotransmitter, the delivery mechanism, and/or the production

[495] Lyketsos, et al., "Does Stroke Cause Depression?", p. 105.
[496] Lyketsos, et al., p. 106.
[497] Paradiso and Robinson, "Minor Depression After Stroke: An Initial Validation of the DSM-IV Construct.", p. 245.
[498] Paradiso and Robinson, p. 249.
[499] Paradiso and Robinson, "Gender Differences in Poststroke Depression.", p. 44.
[500] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 428.

mechanism).[501] They also suggest that there may be a lateralized biogenic amine response in unilateral lesions. Nicholl, et al., note that rat studies also suggest significant disruption of monoamine distribution in anterior lesions – specifically, the pathways for norepinephrine and serotonin delivery seem to be especially vulnerable.[502] Starkstein and Robinson also suggest metabolic disruptions in depression following left anterior frontal lobe lesions, citing earlier studies demonstrating significantly lower glucose metabolism (indicating decreased neuronal activity) and dexamethasone suppression.[503]

The overall duration of the depressive reaction tends to vary depending on lesion location and type of depressive disorder. Startstein and Robinson note:

> In conclusion, major depression lasts for approximately 1 year, while minor depression lasts for more than 2 years. Lesion location is an important factor influencing the duration of PSD, and patients with subcortical or cerebellar/brainstem lesions have significantly briefer depressions than patients with lesions in the MCA [middle cerebral artery] territory.[504]

Treatments for underlying depression and cognitive dysfunction (see below) ought to take into consideration this lengthy duration and is morbid effects on recovery.

Cognitive Dysfunction

Tateno, et al., note that several researchers have found cognitive impairments to be more severe in patients with major depression compared with patients with minor depression or no depression.[505] They noted a variety of intellectual dysfunctions, and reported that while the most severe phase of the depression was during the acute stroke period, the depressive episode was

[501] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 44.
[502] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 429.
[503] Starkstein and Robinson, p. 34; see also p. 43.
[504] Starkstein and Robinson, p. 32-33.
[505] Tateno, Murata, and Robinson, "Comparison of Cognitive Impairment Associated With Major Depression Following Stroke Versus Traumatic Brain Injury.", p. 295.

present for up to a year following the cerebral accident.[506] They also suggest that aging itself influences cognitive function, noting that Mini Mental Status Exam scores without brain injuries tend to be lower than younger subjects.[507] They reported that the same age-based score discrepancy occurred when comparing young versus elderly stroke patients with depression – depression did not result in cognitive deficits in younger stroke patients.[508] Starkstein and Robinson note that the deficits:

> [W]ere most severe in tasks assessing orientation, language, visuoconstructional ability, executive motor functions, and frontal lobe tasks; however, no significant differences in cognitive performance were found between eight patients with major depression after right hemisphere lesions and 19 patients with a similar lesion location but no depression.[509]

They suggest that lesion location might not necessarily yield different cognitive effects. Nicholl, et al., suggest that stroke patients experience more negative and less positive cognitions than non-depressed stroke patients.[510]

Other Neuropsychiatric sequelae

There are a number of potential neuropsychiatric sequelae to stroke,[511] including depression, anxiety, apathy, and fatigue. These can fundamentally affect the morbidity and mortality of the neurological condition, and they can exert a powerful effect on the patient's ability to recover. Robinson notes that in a previous study, stroke has been linked with general anxiety disorder, and that a majority of patients in question also had major or minor depression.[512] Depression with anxiety was linked with left cortical lesions, depression alone

[506] Tateno, Murata, and Robinson, "Comparison of Cognitive Impairment Associated With Major Depression Following Stroke Versus Traumatic Brain Injury.", p. 295.
[507] Tateno, Murata, and Robinson, p. 299. See also the section on dementia concerning neurological and cognitive functions and aging.
[508] Tateno, Murata, and Robinson, p. 300.
[509] Starkstein and Robinson, "Depression in Cerebrovascular Disease.", p. 40.
[510] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 229.
[511] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 225.
[512] Robinson, p. 224-5.

was linked with left subcortical lesions, and anxiety alone was linked with right cortical lesions.[513] Robinson also discusses a study that 22% of patients in one study displayed symptoms of either apathy alone or apathy with depression.[514] He noted an apparent link between the age of the patient and the frequency of basal ganglia lesions, as well as a greater decrement in activities of daily life functioning in patients with both depression and apathy than in patients with depression or apathy alone. Staub and Bogousslavsky suggest that fatigue, a common complaint in neurological patients, may be related to, be masked by or exacerbate comorbid depression.[515] They suggest that fatigue in neurological patients (e.g., patients with multiple sclerosis, Parkinson Disease, stroke, etc.) be considered a potentially separate neurological sequela of the condition – i.e., fatigue on top of a potential depressive disorder.[516]

Treatment

At present, treatment focuses on the neurological damage concomitant to the stroke – if the cause of the cerebral infarction is a stenosis (e.g., more than 70% of the carotid artery), the course of treatment would suggest carotid endarterectomy.[517] If there is no evidence of a stenosis, antiplatelet agents tend to be employed (e.g., aspirin); if there is concomitant coronary fibrillation, anticoagulants may be used, though there is a risk of further cerebral hemorrhage.[518] Therapy tends to involve limitation of the extent of the damage by blocking the NMDA (N-methyl-D-aspartate) channel, a calcium channel receptive to glutamate that opens in ischemia, creating a series of secondary reactions which culminate in neuronal death. This treatment is not

[513] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 224-5.
[514] Robinson, p. 225.
[515] Fabienne Staub and Julien Bogousslavsky, "Post-Stroke Depression or Fatigue?" *European Neurology* 45 (January 2001): 3-5, p. 4.
[516] Staub and Bogousslavsky, "Post-Stroke Depression or Fatigue?", p. 4.
[517] Drislane, et al., *Blueprints in Neurology*, p. 112.
[518] Drislane, et al., p. 112.

always effective, as it is suggested that other calcium channels are dysfunctional in addition to the NMDA channel.[519]

Treatment for post-stroke depression (PSD) parallels that of traditional depressive disorders – a combination of psychotherapeutic and pharmacological approaches tends to yield the best results.[520] PSD tends to be associated with a number of psychosocial variables. Kneebone and Dunmore note that PSD risk factors include "institutionalization, divorce and pre-stroke alcohol consumption, younger age and activities of daily living impairment and perception of social support."[521] They note that the relative lack of attention that the recognition of and treatment for PSD have received has profoundly negative impacts on recovery.[522] Ramasubbu, et al., note that comorbid depression is frequently missed in the diagnosis and treatment of stroke.[523] Given the effects of depression on recovery, they suggest that "depression should be taken into account in the evaluation and treatment of functional abilities of all stroke patients," and that patients with depression have greater improvement in their activities of daily life if they are treated with antidepressants.[524] Robinson notes that benzodiazepines (anti-anxiety medications) may be used in treatment of post-stroke depression, but suggests that caution must exercised, as post-stroke patients are vulnerable to side effects like sedation, ataxia, disinhibition and confusion.[525] He also suggests that tricyclic antidepressants may be effective. Nicholl, et al., suggest that antidepressant therapy can be contraindicated in some stroke patients, and suggest that cognitive-behavioral therapy (CBT) be employed as a possible supplement or

[519] Victor and Ropper, *Principles of Neurology*, p. 828.
[520] There is some controversy about this, however. Kneebone and Dunmore suggest that there are potential side effects of antidepressant medication that may affect an individual's recovery.
[521] Kneebone and Dunmore, "Psychological Management of Post-Stroke Depression.", p. 54.
[522] Kneebone and Dunmore, p. 55.
[523] Ramasubbu, et al., "Functional Impairment Associated with Acute Poststroke Depression: The Stroke Data Bank Study.", p. 32.
[524] Ramasubbu, et al., " p. 32.
[525] Robinson, "Neuropsychiatric Consequences of Stroke.", p. 225.

replacement to pharmacological interventions.[526] Kneebone and Dunmore note that there "is some evidence that PSD is more a 'depression of dementia' than a 'dementia of depression', that is depression develops as a result of cognitive impairment, rather than is caused by it post stroke."[527] Therapies directed at this cognitive impairment (e.g., problem solving) can greatly improve the family function difficulties experienced by many post-stroke patients.[528] They note that other forms of therapy can compliment CBT, noting that "research with depressed older caregivers of persons with dementia has identified interpersonal psychotherapy as more useful early in the process, CBT later."[529]

CANCER

Cancer is a genetic aberration, but this is not meant in the sense of something uncommon or out of the realm of normal human experience. Cancer is an aberration in the sense of the cell's function; as a neoplastic disorder, it describes a condition in which the cells grow abnormally, out of sync or proportion with the host's body or natural cycle of cell replacement. In a patient with cancer, it can lead to "a sense of betrayal by one's own body."[530] A cancer diagnosis generally relies most on tissue biopsy – it is not recommended to make the diagnosis absent this invasive but necessary test.[531] It is critical to identify malignancies early; early detection makes treatment easier, as well as increasing the odds of surviving. Longo notes that "the curability of a tumor usually is inversely proportional to the tumor burden."[532] Given a variety of therapeutic interventions (including surgery, chemotherapy, radiotherapy, and

[526] Nicholl, et al., "Cognitions and Post-Stroke Depression.", p. 222.
[527] Kneebone and Dunmore, "Psychological Management of Post-Stroke Depression.", p. 61-2.
[528] Kneebone and Dunmore, p. 61-2.
[529] Kneebone and Dunmore, p. 62-3.
[530] Daniel L. Longo, "Approach to the Patient with Cancer," in *Harrison's Principles of Internal Medicine (15th Edition)*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 491-97, p. 491.
[531] Longo, "Approach to the Patient with Cancer.", p. 492.
[532] Longo, p. 492.

biological therapy), more than half of all patients diagnosed with cancer will be cured.[533] There are, however, a wide variety of psychological sequelae to the diagnosis, involving changes in self-image, psychosocial function, and long-term goals.[534]

Several authors have noted that nearly all cancers stem from a single cell, but that multiple triggers are necessary for a cell to convert from a normal to a malignant phenotype.[535] Like other illnesses, it is suggested that cancer follows the stress-diasthesis model of pathology. Most if not all cancer has a genetic component, but there must be some other factor present to set the malignancy in motion.[536] Further, latent susceptibility to cancer does not follow strictly Mendelian patterns, and specific risks to particular types of cancers can vary in differing populations.

Several types of genes are involved in the production of cancer. Genes that promote normal cell growth are called protooncogenes; once the protooncogene is activated through mutation or dysregulation, it is converted into an oncogene.[537] Protooncogenes can be converted to oncogenes through several mechanisms – point mutations, DNA amplification, and chromosomal alterations can all cause upregulation of protooncogenes to active oncogenes.[538] At the opposite end of the genetic spectrum are the genes which inhibit cell growth, called tumor suppressor genes.[539] Tumors are produced when the tumor suppressor genes are dysfunctional –

[533] Longo, "Approach to the Patient with Cancer.", p. 491.
[534] Longo, p. 491.
[535] Francis S. Collins and Jeffrey M. Trent, "Cancer Genetics," in *Harrison's Principles of Internal Medicine (15th Edition)*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 503-09, p. 503; G. Fenton and Dan L. Longo, "Cell Biology of Cancer," in *Harrison's Principles of Internal Medicine*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 509-17, p. 510.
[536] Collins and Trent, "Cancer Genetics.", p. 503; Fenton and Longo, "Cell Biology of Cancer.", p. 510.
[537] Collins and Trent, p. 503.
[538] Collins and Trent, p. 506-8.
[539] Collins and Trent, p. 503.

the cause of most cancers.[540] A third type implicated in cancer involves genes which repair DNA, as DNA can experience mutation errors in transcription during cell division.[541]

Cell growth itself is not a sign of malignancy – cell growth and division is a natural process and part of the body's ability to heal, grow, and develop. Cell growth is malignant, however, when two key properties are evidenced: Fenton and Longo note first that cell growth unregulated by external signals is characteristic of all new growth tissues (termed neoplasia, which may be benign or malignant); second, malignant neoplasia occurs when the new growth invades other tissues and metastasizes to other distant sites. Cancer, they note, is synonymous with malignant neoplasia,[542] and is "most common in tissues in tissues with rapid turnover, especially those exposed to environmental carcinogens and whose proliferation is regulated by hormones."[543]

Cell growth normally is naturally limited – Fenton and Longo note that on average each somatic cell can replicate itself about 30 times (the Hayflick limit).[544] Each replication involves a loss of some DNA from the end of the DNA chain (the loss occurs in tandem repeats of a six nucleotide sequence called telomeres (the sequence is three guanine nucleotides, two thymine nucleotides, one adenine nucleotide)). The replication of telomeres is performed by telomerase, an RNA-dependent polymerase. Germ-line cells express their own telomerase, which can, in theory, allow for unlimited replication. Normal somatic cells, however, do not produce their own telomerase, hence the natural limit on the number of potential replications for each somatic

[540] Fenton and Longo, "Cell Biology of Cancer.", p. 510.
[541] Collins and Trent, "Cancer Genetics.", p. 503.
[542] Fenton and Longo, p. 509.
[543] Fenton and Longo, p. 510.
[544] Fenton and Longo, p. 510.

cell. It has been proposed that a dysfunction in telomerase production is implicated in the process of malignant neoplasia.[545]

Cells normally are self-regulating in their life and death cycle. A variety of mechanisms exist that prevent cell growth from becoming neoplastic. A transcription factor called p53, for example, is normally not needed in cell reproduction, and as a result is shuttled out of the cell nucleus to degrade. However, when DNA damage occurs, it builds up in the cell and either halts cell cycle progression (allowing for repair) or initiates self-destruction in the cell (a process called apoptosis). There are a variety of mechanisms of p53 activity, including hypoxia, DNA damage, ribonucleotide depletion, telomere shortening, and dysregulated oncogene activity. Mutation of p53 is the most common genetic aberration in human cancer (found in greater than 50% of human cancers).[546]

Overexpression or increased signaling of tyrosine kinase-linked pathways are also linked with human cancer. Fenton and Longo note that about 30% of human cancers have a mutation in the *ras* protein.[547] In addition to *ras*, other overexpressed tyrosine kinase receptors include EGF receptors, IGF-I receptors, HER-2/neu, and mutations in the Ret receptor.[548]

There are other physiological processes involved in cancer. Neovascularization – the formation of new capillaries – is a normal process of wound repair, muscle repair, reproduction and embryonic development. These processes, however, are normally self-limiting. In pathologic angiogenesis, however, the growth of new vasculature is unregulated. When this unregulated angiogenesis continues for extended periods of time (months/years), it supports the

[545] Fenton and Longo, "Cell Biology of Cancer.", p. 510.
[546] Fenton and Longo, p. 510-11; Judah Folkman, "Angiogenesis," in *Harrison's Principles of Internal Medicine (15th Edition)*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 517-30, .p. 518.
[547] Fenton and Longo, p. 513.
[548] Fenton and Longo, p. 513; Folkman, "Angiogenesis.", .p. 518.

growth of solid tumors and leukemias, facilitates inflammatory diseases, and leads to hemorrhages and intraperitoneal bleeding, among other angiogenic disease processes.[549] Folkman notes that non-angiogenic lesions exist in a far greater percentage of the population than is diagnosed with a tumor – a small percentage of these convert to angiogenic tumors after long periods of time.[550] The shift to an angiogenic form of the tumor is denoted by increased tumor mass (to a detectable level), localized bleeding, and tumor metastasis. Four triggers of angiogenesis have been found: avascular carcinomas stimulate neovascularization in adjacent vasculature; circulating endothelial cells may localize around the tumor, stimulating angiogenesis; tumors may induce other cells to overexpress angiogenic factors like vascular endothelial growth factor (VEGF); or preexisting vessels may be accessed by the tumor.[551] Folkman notes that once tumors have switched to an angiogenic phenotype, they rarely revert to the nonangiogenic phenotype."[552]

There are a variety of psychosocial sequelae to a diagnosis of cancer; some are very specific (e.g., disfigurement in head and neck cancers or breast cancer; impotence following prostate cancer surgery), others are generalized, and applicable regardless of the site or type of cancer (e.g., depression or anxiety following diagnosis or anticipatory anxiety in the face of a potential recurrence or metastasis – what Longo refers to as the "Damocles syndrome").[553] Psychological sequelae will be covered in greater detail below. Physical pain can be a significant concern for cancer patients. Longo notes that:

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[549] Folkman, "Angiogenesis.", p. 517.
[550] Folkman, p. 518.
[551] Folkman, p. 518.
[552] Folkman, p. 518.
[553] Fitzsimmons, et al., "Differences in Perception of Quality of Life Issues Between Health Professionals and Patients with Pancreatic Cancer," *Psycho-Oncology* 8 (1999): 135-43, p. 135; Cook Gotay, Joan L. Holup, and Ian Pagano, "Ethnic Differences in Quality of Life Among Early Breast and Prostate Cancer Survivors," *Psycho-Oncology* 11 (2002): 103-13, p. 103; Longo, "Approach to the Patient with Cancer.", p. 496.

> Pain occurs with variable frequency in the cancer patient: 20 to 50% of patients present with a pain at diagnosis, 33% have pain associated with treatment, and 75% have pain with progressive disease. The pain may have several causes. In about 70% of cases, pain is caused by the tumor itself – by invasion of bone, nerves, blood vessels, or mucous membranes or obstruction of a hollow viscous or duct. In about 20% of cases, pain is related to a surgical or invasive medical procedure, to radiation injury (mucositis, enteritis, or plexus or spinal cord injury), or to chemotherapy injury (mucositis, peripheral neuropathy, phlebitis, steroid-induced aseptic necrosis of the femoral head). In 10% of cases, pain is unrelated to cancer or its treatment.[554]

As such, there are a variety of concerns secondary to the disease process that need to be managed in a patient with cancer. Understanding of the disease process, treatment options, influence of lifestyle, self-image, and risk of recurrence are but a handful of the multifaceted care cancer demands.

Epidemiology

The epidemiology of cancer varies in accordance with the type of malignancy in question. Overall, Longo notes that:

> In 2000, 1.2 million new cases of invasive cancer (619,700 men, 600,400 women) were diagnosed and 552,300 people (284,100 men, 268,100 women) died from cancer…Cancer incidence has been declining by about 2% each year since 1992. The most significant risk factor for cancer overall is age; two-thirds of all cases were in people over age 65. Cancer incidence increases as the third, fourth, or fifth power of age in different sites. For the interval between birth and age 39, 1 in 62 men and 1 in 52 women will develop cancer; for the interval between ages 40 and 59, 1 in 12 men and 1 in 11 women will develop cancer; and for the interval between ages 60 and 79, 1 in 3 men and 1 in 4 women will develop cancer. Cancer is the second leading cause of death behind heart disease…Along with the decrease in incidence has come an increase in survival for cancer patients. The 5-year survival for white patients was 39% in 1960-1963 and 61% in 1989-1995. Cancers are more often deadly in blacks; the 5-year survival was 48% for the 1989-1995 interval. Incidence and mortality vary among racial and ethnic groups. The basis for these differences is unclear.[555]

[554] Longo, "Approach to the Patient with Cancer.", p. 495.
[555] Longo, p. 491.

Uchitomi notes that lung cancer is the **"**most common cancer and the most common cause of cancer-related death in the world."[556] This claim is supported by the World Health Organization's *Global Health Statistics*, in which tracheal/bronchial/lung cancers account for 1,331,000 projected deaths globally in 2000[557] (stomach cancers were next on the list at 1,010,000 projected deaths[558], followed by liver cancer at 703,000 projected deaths[559], colorectal cancers at 592,000 projected deaths[560], and esophageal cancers at 493,000 projected deaths[561]). For a more complete picture of the projected global epidemiology of neoplastic disorders, please see *Global Health Statistics.*

Epidemiology of Depression

Psychological sequelae of cancer are significant concerns in treatment – comorbid psychological illness can fundamentally affect a patient's ability to recover or fight the illness. Katz, et al., note that depression is a common issue in depression; they note that "depression is thought to be one of the most common psychosocial sequelae of cancer and the most likely reason for referral to a mental health professional. While transient states of dysphoria are part of the expected response to a life threatening illness, prevalence rates of clinically significant depression have varied widely, with reported rates between 1-53% in published studies."[562] Overall, they note a commonly cited prevalence between 20-30% of a comorbid depressive disorder in cancer patients.[563] Sellick and Crooks cite studies noting that 47% of cancer patients met the criteria defining a psychiatric illness; of this 47% "13% of the 47% met the criteria for

[556] Yosuke Uchitomi, et al., "Depression After Successful Treatment for Nonsmall Cell Lung Carcinoma," *Cancer* 89, no. 5 (Sept. 1 2000): 1172-79, p. 1172.
[557] Murray and Lopez, *Global Health Statistics: A Compendium of Incidence, Prevalence and Mortality Estimates for Over 200 Conditions*, p. 558.
[558] Murray and Lopez, p. 546.
[559] Murray and Lopez, p. 552.
[560] Murray and Lopez, p. 549.
[561] Murray and Lopez, p. 543.
[562] Katz, et al., "Screening for Depression in Head and Neck Cancer," *Psycho-Oncology* In press (2003), (in press).
[563] Katz, et al., "Screening for Depression in Head and Neck Cancer.", (in press).

major depression and 68% had an Adjustment Disorder characterized by anxiety or depression."[564] They further note that it would, in fact, be surprising if a patient did not experience a spectrum of psychological sequelae consistent with a diagnosis of depression.[565] Ciaramella and Poli suggest that comorbid depressive disorders occur in 5-40% of cancer patients,[566] and in their own reports, they find a comorbid rate of major depression of 28%.[567] Longo notes an overall rate of comorbid depression of approximately 25%, but this rate may fluctuate in patients who are more compromised.[568] McQuellen, et al., note that "up to 30% of patients report moderate to severe levels of anxiety and depressive symptoms at their initial outpatient visit."[569]

Individual rates of depression based on different types of cancer can vary. Uchitomi, et al., found a rate of approximately 15% of comorbid major or minor depression in non-small cell lung cancer.[570] In older adults, Deimling, et al., note that approximately 13-25% of cancer survivors experience clinically significant levels of depression.[571] Kaguya, et al., found that among patients with head and neck cancers, "66.4% of the patients met criteria for a psychiatric diagnosis, and 16.8% showed psychologic distress (adjustment disorders or major depression)"

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[564] M. Sellick and Dauna L. Crooks, "Depression and Cancer: An Appraisal of the Literature for Prevalence, Detection, and Practice Guideline Development for Psychological Interventions," *Psycho-Oncology* 8 (1999): 315-33, p. 317-318.
[565] Sellick and Crooks, "Depression and Cancer: An Appraisal of the Literature for Prevalence, Detection, and Practice Guideline Development for Psychological Interventions.", p. 319.
[566] Antonella Ciaramella and Paolo Poli, "Assessment of Depression Among Cancer Patients: The Role of Pain, Cancer Type and Treatment," *Psycho-Oncology* 10 (2001): 156-65, p. 156.
[567] Ciaramella and Poli, "Assessment of Depression Among Cancer Patients: The Role of Pain, Cancer Type and Treatment.", p. 160-2.
[568] Longo, "Approach to the Patient with Cancer.", p. 494-5.
[569] Richard P. McQuellon, et al., "Reducing Distress in Cancer Patients with an Orientation Program," *Psycho-Oncology* 7 (1998): 207-17, p. 207.
[570] Uchitomi, et al., "Depression After Successful Treatment for Nonsmall Cell Lung Carcinoma.", p. 1175.
[571] Gary T. Deimling, et al., "Cancer Survivorship and Psychosocial Distress in Later Life," *Psycho-Oncology* 11 (2002): 479-94, p. 490.

based on DSM-IIIR criteria.[572] They caution, however, that their sampling may have been biased by their population focusing on somatic complaints. Golden-Kreutz & Andersen noted previous studies finding the prevalence of diagnosable depression in breast cancer to be 20-30% in general, with specific studies finding rates of 6% to 29%.[573] Their own research yielded a comorbid rate of 18%, with specific risk factors correlated with depressive symptoms (i.e., perceptions of global stress, intrusive thoughts about cancer-related traumatic stress, financial difficulties and other stressful life events, neuroticism and racial minority status).[574] Kurtz, et al., found that as many as half of all lung cancer patients "experience depressive symptomatology at a level that would qualify for clinical diagnosis,"[575] with the greatest prevalence in patients 65 and older.[576] In a study of changes in endocrine levels in patients with metastatic cancer, Cohen, et al., found that "18% of the sample were experiencing moderate to severe symptoms of depression."[577] Fitzsimmons, et al., note that the psychopathology of pancreatic cancer may also be linked to endocrine or acid-base changes.[578]

Several studies have noted that clinically significant depression is not diagnosed in cancer patients.[579] Further, there are barriers that prevent recognition of depression and depressive

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[572] Kugaya, et al., "Prevalence, Predictive Factors, and Screening for Psychologic Distress in Patients with Newly Diagnosed Head and Neck Cancer," *Cancer* 88, no. 12 (June 15 2000): 2817-23, p. 2821.
[573] M. Golden-Kreutz and Barbara L. Andersen, "Depressive Symptoms After Breast Cancer Surgery: Relationships with Global, Cancer-Related, and Life Event Stress," *Psycho-Oncology*, (in press).
[574] Kreutz and Andersen, "Depressive Symptoms After Breast Cancer Surgery: Relationships with Global, Cancer-Related, and Life Event Stress.", (in press).
[575] M.E. Kurtz, et al., "Predictors of Depressive Symptomatology of Geriatric Patients with Lung Cancer - A Longitudinal Analysis," *Psycho-Oncology* 11 (2002): 12-22, p. 12.
[576] Kurtz, et al., "Predictors of Depressive Symptomatology of Geriatric Patients with Lung Cancer - A Longitudinal Analysis.", p. 13.
[577] Lorenzo Cohen, et al., "Endocrine Levels at the Start of Treatment Are Associated With Subsequent Psychological Adjustment in Cancer Patients with Metastatic Disease," *Psychosomatic Medicine* 63 (2001): 951-58, p. 955.
[578] Fitzsimmons, et al., "Differences in Perception of Quality of Life Issues Between Health Professionals and Patients with Pancreatic Cancer.", p. 136.
[579] Uchitomi, et al., "Depression After Successful Treatment for Nonsmall Cell Lung Carcinoma.", p. 1177; Katz, et al., "Screening for Depression in Head and Neck Cancer.", (in press); Kugaya, et al., "Prevalence, Predictive Factors, and Screening for Psychologic Distress in Patients with Newly Diagnosed Head and Neck Cancer.", p.

symptomology, including a lack of time to assess depression, a lack of familiarity with assessment measures, or "ignorance or pessimism about treatment approaches."[580] Berard, et al., expand upon this list, suggesting that:

> The overlap of symptomatology, the fact that sadness and psychological distress are to an extent normal and expected reactions to a diagnosis or recurrence of disease, the lack of resources and staff prohibiting effective biopsychosocial assessment and care, and patients' unwillingness to disclose emotional problems are some of the reasons for this state of affairs.[581]

Further difficulties exist in symptom recognition and isolation. Ciaramella and Poli note that some of the somatic symptoms of cancer can be mistaken for the somatic symptoms of depression. They state:

> Anorexia, weight loss, low energy and sleep disturbance are common in all acutely ill cancer patients, but are also neurovegetative signs of depression. Several authors have proposed excluding these somatic symptoms from depression diagnosis criteria. They also found that the prevalence point of major depression dropped from 42 to 24% when all somatic symptoms were eliminated as criteria.[582]

Berard, et al., note that several authors have suggested using the HADS and BDI as screening tools for depression. The HADS removes many of the somatic symptoms from the diagnostic criteria in favor of the psychological criteria, and hence, may be able to discern symptoms that stem from an underlying psychiatric comorbidity.[583]

Risk Factors for Depression

There are a variety triggers for depression in cancer, including medical comorbidities, self-perception, and psychosocial support. Depression is not an uncommon phenomenon, and as

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2817; R.M.F. Berard, F. Boermeester, and G. Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management," *Psycho-Oncology* 7 (1998): 112-20, p. 112.

[580] Katz, et al., "Screening for Depression in Head and Neck Cancer.", (in press).

[581] Berard, Boermeester, and Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management.", p. 112.

[582] Ciaramella and Poli, "Assessment of Depression Among Cancer Patients: The Role of Pain, Cancer Type and Treatment.", p. 156-7.

[583] Berard, Boermeester, and Viljoen, p. 117.

such is a significant concern in psychooncology, especially in light of the suggested influence of depression on long-term survival.[584] Research has identified several notable risk factors.

Uchitomi found that satisfaction with one's confidants was the only variable significantly related to depression in lung cancer patients in the three months following surgery; at four months, pain and performance were also significantly related.[585] He noted that while several studies have linked social support and communication with confidants as significant in psychosocial management, there are other interpretations of this data. He argues that "patients who are depressed might by much more likely to express dissatisfaction with support."[586]

The presence and severity of depression have been linked to and may be contingent upon the type of cancer diagnosed and whether it has metastasized. Ciaramella and Poli note that there is a demonstrable difference in psychopathological sequelae in pancreatic cancer versus other types.[587] They also note that the presence of pain has been found to be a risk factor for major depression in all cancer patients.[588] They note previous research demonstrating a higher lifetime incidence of depression in low-pain versus high-pain groups, but a higher point prevalence of major depression in high-pain groups than in low-pain groups.[589] Finally, they note that metastasis is a contributory factor in the onset of depression – 50% of their patients with metastasized cancer experienced major depression, which they suggest explains why the diagnosis of depressive comorbidities increases as the disease progresses to more advanced stages.[590]

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[584] Uchitomi, et al., "Depression After Successful Treatment for Nonsmall Cell Lung Carcinoma.", p. 1178.
[585] Uchitomi, et al., p. 1175.
[586] Uchitomi, et al., p. 1177.
[587] Ciaramella and Poli, "Assessment of Depression Among Cancer Patients: The Role of Pain, Cancer Type and Treatment.", p. 157.
[588] Ciaramella and Poli, p. 157.
[589] Ciaramella and Poli, p. 157.
[590] Ciaramella and Poli, p. 162.

Deimling, et al., suggest that the strongest predictor of psychological sequelae like depression and post-traumatic stress disorder is current cancer-related symptoms. They suggest that "individuals who continue to experience sequelae of cancer are more likely to be depressed and report hyper-arousal symptoms such as impaired concentration or sleep disturbance."[591] Further, they note that the means of cancer treatment can produce specific sequelae – e.g., the toxic short-term and long-term effects of chemotherapy may contribute to psychopathological sequelae and distress.[592] They found that aging may offer a somewhat protective function against the development of depression – it is suggested that other losses or challenges in the patient's life may displace cancer as his or her preeminent concern.[593]

Street endorsed other risk factors, noting that several studies found elevated risks of depression in three areas. Specifically, he notes that "**t**he majority of these studies have suggested that negative self-beliefs, feelings of hopelessness and a lack of perceived social support are all significant in predicting depression in cancer patients."[594]

Akechi, et al., found poor performance status, employment status, and severe depression to be significant risk factors for suicidal ideation in cancer patients in univariate analysis.[595] Multivariate analysis linked poor physical functioning and severity of depression with suicidal ideation, which correlated with earlier studies linking poor physical functioning with depression and depression with suicidality.[596]

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[591] Deimling, et al., "Cancer Survivorship and Psychosocial Distress in Later Life.", p. 490.
[592] Deimling, et al., p. 490.
[593] Deimling, et al., p. 491.
[594] Helen Street, "The Psychosocial Impact of Cancer: Exploring Relationships Between Conditional Goal Setting and Depression," *Psycho-Oncology* 12 (2003): 580-89, p. 581.
[595] Akechi, et al., "Why Do Some Cancer Patients With Depression Desire an Early Death and Others Do Not?" *Psychosomatics* 42, no. 2 (March/April 2001): 141-45, p. 142.
[596] Akechi, et al., "Why Do Some Cancer Patients With Depression Desire an Early Death and Others Do Not?", p. 144.

Sellick and Crooks note a variety of characteristics, expected and unexpected, linking gender, education levels, familial history of affective disorder, stage of the illness, etc. They argue that:

> Significant correlates for depression include: female gender, lower level of education, never married, separated, widowed or divorced, employed as a homemaker, lower income earners, Hispanics and persons aged 15-24. Other correlates include age under 60; patients with a history of affective disorder, alcoholism, or poorly controlled pain, and patients on medications and/or treatments causing depression; patients with physical impairment, medical illness, disability, or advanced illness; and women with early stage disease.[597]

Unlike other researchers, they do not posit a link between the patient's available support network and comorbid affective illness.

Kaguya, et al., note that in head and neck cancers, being unmarried and living alone are predictive of psychological distress (in univariate analysis).[598] While they note that social support in newly diagnosed patients with head and neck cancers is important, they argue that a lack of social support or dissatisfaction with available levels of social support are not necessarily predictive of psychological distress.[599]

Berard, et al., found that the severity of the patient's medical illness was not a significant risk factor for depression, but stressed that their data was drawn from outpatient populations, and as such, may not be representative of hospitalized patients with advanced disease (they refer to depression found in in-patients as the 'end of the road syndrome').[600] They report that a variety of extrinsic factors can be linked with the onset of depressive symptoms, and as such, the psychopathological disease model is likely much more complex than simply the patient's

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[597] Sellick and Crooks, "Depression and Cancer: An Appraisal of the Literature for Prevalence, Detection, and Practice Guideline Development for Psychological Interventions.", p. 330
[598] Kaguya, et al., "Prevalence, Predictive Factors, and Screening for Psychologic Distress in Patients with Newly Diagnosed Head and Neck Cancer.", p. 2821-2
[599] Kaguya, et al., p. 2821-2
[600] Berard, Boermeester, and Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management.", p. 119

response to his or her physical illness – cancer creates a vulnerability to depression, but is not the sole cause.[601]

Golden-Kreutz and Andersen suggest that a combination of psychosocial stressors may increase the risk of depression in women. The trauma of the cancer, financial difficulties, and neurotic tendencies may increase the risk of developing depressive symptoms.[602] They note that prior research has suggested that psychological symptoms in cancer patients "wax and wane over time with initial adjustment disorders developing into depressive or anxiety disorders."[603]

Finally, Kurtz, et al., suggest that in elderly lung cancer patients, severity of their symptoms, limitations in social functioning, and radiation treatment were the biggest risk factors for the development of depression in the first year following diagnosis.[604] The elderly face multiple threats – as their friends and relatives age, they face a dwindling support network, and as such, are at a greater risk of developing depressive symptoms that they cannot manage alone.[605] They note that prior research has demonstrated the significance of social support for the psychological well-being of the elderly – spouses and children can be protective against depression.[606]

The picture that emerges from these findings is that there are a variety of potential risk factors for depression. Clinicians should pay careful attention to the severity of the patient's symptoms and pain, potential for disfigurement as a result of treatment, and efforts to maintain a good social support network ought to be incorporated into the patient's treatment plan.

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[601] Berard, Boermeester, and Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management.", p. 119.
[602] Kreutz and Andersen, "Depressive Symptoms After Breast Cancer Surgery: Relationships with Global, Cancer-Related, and Life Event Stress.", (in press).
[603] Kreutz and Andersen, (in press).
[604] Kurtz, et al., "Predictors of Depressive Symptomatology of Geriatric Patients with Lung Cancer - A Longitudinal Analysis.", p. 20.
[605] Kurtz, et al., p. 13.
[606] Kurtz, et al., p. 20.

Treatment

There are several modalities of cancer treatment – surgical, radiological, chemotherapeutic, and biological.[607] Surgical therapy is the most effective therapeutic intervention – approximately 40% of cancers are cured by surgical resection of the malignant tissue (and, sometimes, the tissue surrounding the malignancy).[608] Radiation therapy and chemotherapy tend to be the most familiar interventions to the general public. Biological therapy involves treatment with interferons, immunotherapy, differentiating agents, and agents designed to attack the specific biology of the cancer cell. The four therapies are often used in conjunction with one another.[609]

Treatment may not necessarily involve a complete cure. Proximity to nerves or vasculature, inaccessibility to the recommended therapy, stage of the illness, metastasis, etc. may prevent a complete cure or remission. Longo notes that cancer treatment uses a specific language to describe the disease and its progression/regression:

> A *complete response* is defined as disappearance of all evidence of disease, and a *partial response* as >50% reduction in the sum of the products of the perpendicular diameters of all measurable lesions. *Progressive disease* is defined as the appearance of any new lesions or an increase of >25% in the sum of the products of the perpendicular diameters of all measurable lesions. Tumor shrinkage or growth that does not meet any of these criteria is considered *stable disease*.[610]

It may not be possible to completely remove or eradicate a malignancy. As such, the goal of cancer treatment may not necessarily be curative – there are times when palliative care is the end desired.[611] Tumors compressing a nerve and causing pain, paresis, etc. may be shrunk or

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[607] Edward A. Sausville and Dan L. Longo, "Principles of Cancer Treatment," in *Harrison's Principles of Internal Medicine (15th Edition)*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 530-47, p. 530.
[608] Sausville and Longo, "Principles of Cancer Treatment.", p. 531.
[609] Sausville and Longo, p. 530.
[610] Longo, "Approach to the Patient with Cancer.", p. 494.
[611] Sausville and Longo, p. 530.

partially removed by treatment, but the underlying malignancy remains. The principle goal of cancer treatment, however, is eradication of the cancer.

There are side effects of cancer therapy. Surgery can cause disfiguring scars and subsequently profoundly effect the patient's self-esteem and quality if life. Radiation can compromise immunity or bring about secondary tumors. Chemotherapy is debilitating, causing a variety of somatic problems. Biological therapies can produce psychological sequelae like depression. As such, Longo notes, "the dictum *primum non nocere* is *not* the guiding principle of cancer therapy…The guiding principle of cancer treatment is *primum succerrere*, first hasten to help."[612] Treatment of cancer involves more than simply managing the physical aspects of the illness – it requires treating both the physical as well as the complex psychological sequelae of the disease.[613]

The first form of treatment under consideration is surgical management of the neoplasm. Surgery is the form of therapy most frequently employed in the management and treatment of neoplastic malignancies. Resection of the tumor through surgical intervention is not without potential long-term effects, however, and many patients are concerned about the possibility of resultant scarring or physical deformity (especially in breast or head/neck cancers).[614] Further, like all forms of treatment, there is a risk of recurrence or metastasis.

Radiation therapy is another frequently employed mechanism of control or eradication. Radiation therapy can shrink tumors or destroy them by destabilizing or damaging the DNA within the tumor cell nucleus, a technique that is more effective in oxygenated cells (hypoxic

[612] Sausville and Longo, "Principles of Cancer Treatment.", p. 530.
[613] Longo, "Approach to the Patient with Cancer.", p. 494.
[614] Michael C. Perry and Dan L. Longo, "Late Consequences of Cancer and Its Treatment," in *Harrison's Principles of Internal Medicine (15th Edition)*, ed. Eugene Braunwald, et al. (New York: McGraw-Hill Medical Publishing Division, 2001), 650-53, p. 650.

cells are more resistant).[615] There are several concerns about radiation therapy, principally the possibility of damaging healthy cells in an effort to destroy the malignancy. Sausville and Longo note that "the challenge for radiation treatment planning is to deliver the radiation to the tumor volume with as little normal tissue in the field as possible.[616] Teletherapy – a beam of radiation directed at the tumor site – is the most frequent means of radiation-based treatment.[617] Sometimes the effects of radiation are not immediately lethal – some cells only begin to die off after they try to replicate.[618] There are a variety of cancers that can be attacked with radiation therapy, and it has both curative and palliative uses. Sausville and Longo note that:

> Radiation therapy is a component of curative therapy for a number of diseases including breast cancer, Hodgkin's disease, head and neck cancer, prostate cancer, and gynecologic cancers. Radiation therapy can also palliate disease symptoms in a variety of settings; relief of bone pain from metastatic disease, control of brain metastases, reversal of cord compression and superior vena caval obstruction, shrinkage of painful masses, and opening threatened airways. In high-risk settings, radiation therapy can prevent the development of leptomeningeal disease and brain metastases in acute leukemia and lung cancer.[619]

There are, however, concerns about radiation toxicity. There are cells in the human body that are far more sensitive to the effects of radiation (like bone marrow), and cells that are resistant (like bone, heart and skeletal muscles, nerves).

There are long-term effects of radiation therapy that can cause concern. Sausville and Longo note that chronic toxicities can lead to thyroid dysfunction, problems with vision and dentition, taste and smell dysfunction, a greater risk of myocardial infarction, vascular and pulmonary problems, as well as a significant risk of secondary tumors.[620] Perry and Longo note

[615] Sausville and Longo, "Principles of Cancer Treatment.", p. 532.
[616] Sausville and Longo, p. 532.
[617] Sausville and Longo, p. 532.
[618] Sausville and Longo, p. 532.
[619] Sausville and Longo, p. 533.
[620] Sausville and Longo, p. 533.

that radiation can damage normal organ function, increase the risk of second solid tumors in radiation ports, and promote atherosclerosis.[621]

The form of cancer treatment that tends to be most familiar is chemotherapy. It is commonly used to treat metastatic cancers, and tends to be employed if surgical resection or radiation therapy has not been effective in destroying a localized tumor. Sausville and Longo note that chemotherapy can be used as part of a multifactorial primary approach to tumor management (i.e., as part of a treatment regime for a given neoplastic malignancy), and/or as "an *adjuvant* to surgery or radiation, a use that may have curative potential in breast, colon, or anorectal neoplasms."[622] Chemotherapy tends to be used either in conventional dose or high-dose regimens.[623] Conventional doses produce readily manageable (if uncomfortable or dystonic) side effects in patients. High-dose regimens can produce more therapeutic effects, but this comes at the price of an increased risk of potentially life-threatening complications.[624] Perry and Longo note that chemotherapy can damage bone marrow, create immunodeficiencies, lead to a wide variety of organ dysfunctions, or produce latent subclinical damage which can increase the risk of developing later illnesses or neoplastic malignancies.[625]

A fourth category of cancer treatment involves biological approaches to tumor management. Sausville and Longo note that "the goal of biologic therapy is to manipulate the host-tumor interaction in favor of the host."[626] They further note that several different mechanisms have been found to be potentially effective. The first area of exploration is immune function – they note that:

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[621] Perry and Longo, "Late Consequences of Cancer and Its Treatment.", p. 650.
[622] Sausville and Longo, "Principles of Cancer Treatment.", p. 533.
[623] Sausville and Longo, p. 534.
[624] Sausville and Longo, p. 534.
[625] Perry and Longo, p. 650.
[626] Sausville and Longo, p. 546.

> The very existence of a cancer in a person is testimony to the failure of the immune system to deal effectively with the cancer. Tumors have a variety of means of avoiding the immune system: (1) they are often only subtly different from their normal counterparts; (2) they are capable of downregulating their major histocompatibility complex antigens, effectively masking them from recognition by T cells; (3) they are inefficient at presenting antigens to the immune system; (4) they can cloak themselves in a protective shell of fibrin to minimize contact with surveillance mechanisms; and (5) they can produce a range of soluble molecules, including potential immune targets, that can distract the immune system from recognizing the tumor cell…Cancer treatment further suppresses host immunity.[627]

They note that current research is exploring the potential use of allogenic T cells (T cells from a donor, rather than T cells from the cancer patient [referred to as autologous]), as this has been shown to be effective in bone marrow transplants. A second intervention involves removal, manipulation, and reintroduction of autologous T cells. Manipulation of autologous T cells would allow for the creation of tumor-antigen-specific T cells, or they may be activated with polyclonal stimulators and then stimulated by other agents (like interleukin). A third approach involves attempting to use tumor vaccines to boost T cell immunity – the host T cells are primed against tumor-associated peptides.[628]

Antibodies, interferons, and interleukins are also being tapped as potential biological tools to combat cancer. Sausville and Longo note that research has found humanized antibodies to be effective against some lymphomas and epithelial cancers. They note that antibodies used in conjunction with other forms of treatment may be effective.[629] Interferons are not necessarily curative, but have been found to be effective – they "can induce partial responses in follicular lymphoma, hairy cell leukemia, chronic myeloid leukemia, melanoma, and Kaposi's sarcoma. It has been used in the adjuvant setting in stage II melanoma, multiple myeloma, and follicular

[627] Sausville and Longo, "Principles of Cancer Treatment.", p. 546.
[628] Sausville and Longo, p. 546.
[629] Sausville and Longo, p. 548.

lymphoma."[630] There are some side effects of interferon treatment, including fever, fatigue, depression, and immune diseases.[631] Interleukins "can produce tumor regressions in ~20% of patients with metastatic melanoma and renal cell cancer. About 5% of patients may experience complete remissions that are durable, unlike any other treatment for these tumors."[632] There are a number of side effects, however, including hypotension, impaired renal and hepatic function, and pulmonary problems.[633]

A common potential concern in any cancer treatment is recurrence of a malignancy or metastasis. Aside from the concomitant psychological stress this causes, there are physiological differences in the cancer that result as well. It has been found that these secondary malignancies grow at a faster rate than the primary tumors.[634]

There are long-term effects of all modalities of cancer therapies. Long-term survival rates of all patients with cancer are high, but many survivors will experience some form of medical or psychosocial problem. Perry and Longo note that:

> The 5-year survival rate of all patients diagnosed with cancer is now 59%. This year alone, nearly 700,000 survivors will be added to the 7 million already considered cured. Virtually all of these survivors will bear some mark of their diagnosis and its therapy, and many will experience long-term complications, including medical problems, psychosocial disturbances, sexual dysfunction, and inability to find employment or insurance.[635]

The medical problems that may result from cancer treatment affect most major organs or organ systems. Perry and Longo note clinically significant long-term consequences of cancer treatment in the cardiovascular, pulmonary, nervous, reproductive, musculoskeletal, and endocrine systems. Further, specific organs or areas can develop chronic dysfunction as a result of

[630] Sausville and Longo, "Principles of Cancer Treatment.", p. 548.
[631] Sausville and Longo, p. 548.
[632] Sausville and Longo, p. 548.
[633] Sausville and Longo, p. 548.
[634] Sausville and Longo, p. 530.
[635] Perry and Longo, "Late Consequences of Cancer and Its Treatment.", p. 650.

treatment, including the liver, kidneys, bladder, eyes, and mouth.[636] Last, there is a significant risk of developing second tumors and/or fatal clinical syndromes like myelodysplasia or acute myeloid leukemia. They note that the risk of developing a second tumor "is modest in the first decade after treatment but reaches 1% per year in the second decade, such that populations followed for 25 years or more have a ≥25% chance of developing a second treatment-related tumor.[637] Myelodysplasia and acute myeloid leukemia are uncommon following chemotherapy, but occur often enough to warrant attention. They note that "both forms of acute leukemia are highly refractory to treatment, and no preventive strategy has been developed."[638]

Quality of Life

A key issue in treatment of cancer is maintaining the patient's subjective quality of life. Fitzsimmons, et al., found that there is a fundamental difference between how patients and health professionals assess quality of life. Among the differences they note are descriptions of quality of life factors in generalized terms by health professionals, while patients tend to be more specific in their descriptions. Further, health professionals tended to be more mechanistic, direct and linear in their assessment of the illness on the patient's quality of life.[639] They further note that patients did not attribute quality of life changes to their symptoms; rather, what seemed to influence their assessment most was coping with a perceived threat and maintenance of control in their disease process.[640] All of this, they note, suggests that in self-assessment, patients have a more holistic approach, instead of a pure medical model approach.[641] Zittoun, et al., found that

[636] For a full description of long-term consequences, please see Perry and Longo, "Late Consequences of Cancer and Its Treatment."
[637] Perry and Longo, "Late Consequences of Cancer and Its Treatment.", p. 652.
[638] Perry and Longo, p. 652.
[639] Fitzsimmons, et al., "Differences in Perception of Quality of Life Issues Between Health Professionals and Patients with Pancreatic Cancer.", p. 138.
[640] Fitzsimmons, et al., p. 139.
[641] Fitzsimmons, et al., "Differences in Perception of Quality of Life Issues Between Health Professionals and Patients with Pancreatic Cancer.", p. 140

self-assessed overall quality of life was influenced principally by fatigue and emotional factors rather than physical morbidities.[642] Gotay, et al., note that aside from self-reports of function in Filipino patients, there were no ethnic differences in perception of quality of life in cancer – they suggest that "cancer and its associated toxic treatments likely transcends more subtle differences between individuals."[643]

CONCLUSION

The illnesses and comorbid depressive disorders described above are by no means constitutive of the entirety of the issue. As was noted previously, there are many, many more conditions that have depressive psychological sequelae. The scope of the problem, however, should be apparent – depression is a very common consequence of profound or poorly managed illness, and significant concern should be given to decisions that may be the result of the illness. Our concern as ethicists must be that the decision to forgo medical treatment is a statement from the patient's authentic self, and not simply the illness speaking for the patient.

In the next chapter, we will briefly return to the theme of cognitive heuristics. Specifically, we will examine a current controversy in the cognitive psychology literature – the debate about depressive biases versus depressive realism. These two schools of thought raise a fundamental conflict in their assumptions about depressed cognition – advocates of a depressive bias suggest that cognition is fundamentally flawed, producing distorted cognitions which perpetuate the depressive episode and lead to significant psychological morbidity. Depressive realists, on the other hand, note research which suggests that individuals who are mildly to moderately depressed may actually have more realistic thought processes – they may be capable

[642] Zittoun, Sophie Achard, and Martine Ruszniewski, "Assessment of Quality of Life During Intensive Chemotherapy or Bone Marrow Transplantation," *Psycho-Oncology* 8 (1999): 64-73, p. 72.
[643] Gotay, Holup, and Pagano, "Ethnic Differences in Quality of Life Among Early Breast and Prostate Cancer Survivors.", p. 111.

of more honest assessments of the situations in which they find themselves. As a logical consequence of the depressive realism hypothesis, it may be the case that normal cognition actually displays an optimistic bias – we may be unrealistic in our assessments of the world around us. Following this discussion of depressed cognition, we will turn to the autonomy models popular in current medical ethics. It will be argued that they expect unrealistic levels of cognitive ability from moral agents, and that they persist in asserting homuncular autonomy – there is a tendency to ignore the complex processes actually involved in cognition, specifically the backstage elements that exert significant influence.

CHAPTER FOUR: OF ICEBERGS AND AUTONOMY – COGNITIVE DISTORTIONS AND THE FALLACY OF HOMUNCULAR AUTONOMY

Previous chapters have demonstrated how cognition is a reducible concept, and that it is influenced by a variety of backstage and automatic processes. The current chapter explores the kinds of influences that are particular to depression, before examining contemporary popular and influential theories of personal autonomy. The present discussion examines two competing and influential theoretical and empirical viewpoints on depressive cognition. The cognitive theory of depression (raised to prominence by Aaron Beck) argues that individuals with a depressive disorder display pervasive cognitive distortions, twisting reality to fit into a pessimistic schema, which both maintains the existent depressive state as well as facilitates future depression. It contrasts with the theory of depressive realism, which counters Beck's claims with studies demonstrating that depressed individuals may actually make more realistic decisions – instead of being biased by their depressive disorder, they are freed from the unrealistic optimistic bias demonstrated by non-depressed individuals. This conflict and the discussion it has produced demonstrate the difficulty with which one can make assessments of cognitive ability and decision-making. This will set the stage for a critique of dominant models of autonomy in the contemporary medical ethics literature – quite simply, *the most influential autonomy models in the field today are based upon cognitive models that do not exist, and, in fact, have never existed.*

DEPRESSIVE COGNITION

How individuals process information is not simply a concern of cognitive psychology and neuroscience. Clinical psychology also has a vested interest in the topic – how we process information is just as vulnerable to inside interference and influence as it is to outside, as argued in chapters one and two. Individuals with psychological or psychiatric disturbances are vulnerable to influences at a variety of levels of reduction. At a social level, psychiatric illness

carries a social stigma – in some cultures it is revered and in others reviled. This social element affects relationships and social status, which in turn affects cognition. At a personal level, the individuals self-perception is affected, which in turn affects cognition. Whether the individual has insight into his condition is immaterial; both insight and ignorance have cognitive sequelae.[1] At the cellular level and below, biochemical processes are altered, which in turn affects cognition (e.g., serotonin deficiencies producing anxiety, dopamine deficiencies producing novelty-seeking behavior, etc.). There is no shortage of ways in which depression and other affective disorders can influence how we think.

Cognitive Distortion and Beck's Model

In Beck's model, depression produces systemic distortions in information processing and other forms of cognition. This distortion itself may be the culmination of profound and persistent discrepancies between external environmental stimuli and our corresponding internal psychological components.[2] In short, depression may result from persistently misinterpreting the situational and environmental cues and stimuli that surround us. By exploring the bases of these dysfunctional cognitions, we can address and correct them in therapy. These distortions arise from automatic thoughts – much akin to the automatic thoughts discussed in chapter two. Beck's automatic thoughts are cognitive responses to stimuli which produce affective reactions. If these automatic thoughts are mistaken, a pattern of biased cognition develops, potentially leading the agent astray. Beck noted that the automatic thoughts are very specific, are perceived as very plausible by patients, and are very resistant to change:

[1] If the individual possesses insight, he may view himself more negatively, which affects self-esteem and self-image, or may be more circumspect in reality judgments (see the section below on the phenomenon of depressive realism). If the individual does not possess insight, he may view himself as persecuted (in the case of thought disorders or psychotic depression), or have unchallenged, unrealistic assessments of the future, the world around him, or himself. These are but a few possibilities when it comes to influences on cognition.

[2] Aaron T. Beck, *Cognitive Therapy and the Emotional Disorders* (New York: Meridian, 1976), 25.

> As already indicated, the more disturbed a patient was, the more salient were the automatic thoughts. As the patient improved, the automatic thoughts were less obvious; if his condition worsened, the thoughts became more apparent again. These automatic thoughts reported by numerous patients had a number of characteristics in common. They generally were not vague and unformulated, but were specific and *discrete*. They occurred in a kind of shorthand; that is, only the essential words in a sentence seemed to occur – as in a telegraphic style. Moreover, these thoughts did not arise as a result of deliberation, reasoning, or reflection about an event or topic. There was no logical sequence of steps such as in goal-oriented thinking or problem-solving. The thoughts 'just happened,' as if by reflex. They seemed to be relatively *autonomous* in that the patient made no effort to initiate them and, especially in the more disturbed cases, they were difficult to 'turn off.' In view of their involuntary quality they could just as well have been labeled 'autonomous thoughts' as automatic thoughts. In addition, the patient tended to regard these automatic thoughts as *plausible* or reasonable, although they may have seemed far-fetched to somebody else. The patients accepted their validity without question and without testing out their reality or logic. Of course, many of these thoughts were realistic. But the patient often tended to believe the unrealistic thoughts even though he had decided during previous discussions that they were invalid.[3]

The automatic thoughts in question tend to be negative self-evaluations – the individual tends to be self-deprecating (e.g., in skill, ability, personality, or other characteristics), tends to externalize attributions of success (e.g., believing that he 'got lucky', since he could not have succeeded on his own), and tends to internalize attributions of failure. These are elements of a 'cognitive triad' (see below). These self-evaluations (or self-reproaches) produce feelings of guilt or sadness, which over time "shade into pathological states such as depression in which self-reproaches and self-criticisms are paramount."[4]

Beck notes that individuals form rules that guide actions, interpretations, expectancies, and self-instructions. These rules are applied in a variety of situations, and reinforce the distortions that arise from persistently negative self-cognition. This rule book "contains coding systems used to determine the meanings of stimuli and events."[5] These rules are idiosyncratic –

[3] Beck, *Cognitive Therapy and the Emotional Disorders*, 36.
[4] Beck, 40.
[5] Beck, 42-3.

they are specific to the individual in question, and account for a variety of personal interpretations of a shared event (i.e., why different people take different meanings from the same experience). This idiosyncrasy, when combined with aberrant or dysfunctional 'rules', produces inappropriate or excessive emotional responses – affective disorders.[6] As such, when the cognitive mechanisms of 'normals' and individuals with a psychopathology are examined (i.e., when they are asked to evaluate their external reality), significant differences become apparent. This is not to say that 'normal' cognition cannot produce error – Beck notes that no one "respond(s) consistently well to all challenges. We have specific vulnerabilities, 'fault lines' along which stresses accumulate and may set off tremors or eruptions – behavior commonly labeled 'over-reacting.' Under such conditions unrealistic appraisals override realistic appraisals, and we may realize that our reactions are largely irrational."[7] As a result, it is possible for us to challenge and overcome unrealistic thoughts by examining their bases and the automatic elements which gave rise to them.[8]

Beck's analysis suggests a fundamental challenge to personal autonomy in patients with acute emotional disturbances (as autonomy is classically understood).[9] Specifically, these patients demonstrate an inability to appreciate specific situational stimuli – the patient becomes fixated on certain aspects of his situation over others, exhibiting a kind of 'tunnel vision'.[10] In the case of depressive cognition, it results in focusing on the negative aspects of one's life, and a

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[6] Beck, *Cognitive Therapy and the Emotional Disorders*, 52.
[7] Beck, 76.
[8] This parallels the claims made in the psychological model proposed in chapter two – because our cognition depends significantly on backstage elements, we frequently do not challenge the conclusions we reach or the actions we perform. Beck's argument bolsters the philosophical claim that genuine 'free will' comes from divorcing the automatic cognitive responses we have to situations from our ultimate actions (i.e., challenging the thought process that leads us from environmental stimulus to behavioral or decisional outcome).
[9] I.e., classical autonomy as understood as the deontological, purely rational agent; this will be explored more fully in the second half of this chapter when the concept of autonomy is discussed.
[10] Beck, 79.

difficulty, if not inability, to think of one's life being any different than it is at that moment – a sensation of being trapped:

> The thought content of depressed patients centers on a significant loss. The patient perceives that he has lost something he considers essential to his happiness or tranquility; he anticipates negative outcomes from any important undertaking; and he regards himself as deficient in the attributes necessary for achieving important goals. This theme may be formulated in terms of the cognitive triad: a negative conception of the self, a negative interpretation of life experiences, and a nihilistic view of the future. The sense of irreversible loss and negative expectation leads to the typical emotions associated with depression: sadness, disappointment, and apathy. Furthermore, as the sense of being trapped in an unpleasant situation or of being enmeshed in insoluble problems increases, spontaneous constructive motivation dissipates. The patient, moreover, feels impelled to escape from the apparently intolerable condition via suicide.[11]

This results in a variety of cognitive distortions – aside from the immediate distortion Beck proposes, it is easy to see how other sources of error (e.g., availability, anchoring, affective forecasting, etc. from chapter two) can influence the patient's thought process. The decisions that result tend to exhibit undue generalizations from event to event, catestrophizing events (perceiving minor failures or setbacks as significantly more negative than they actually are), and making arbitrary inferences (e.g,. drawing conclusions where the evidence is lacking or contradictory to one's conclusion), among other distortions.[12] These sources of error present significant challenges to philosophical models arguing that individual autonomy ought never be trumped by a concern for the patient's welfare – they demonstrate that this autonomy is self-destructive, and not self-affirming, a topic that will be addressed in the second half of this chapter. In fact, Beck specifically notes that an affective disorder prevents individuals from making objective judgments. Individuals with affective disorders cannot "disentangle the personal meaning of an event from its objective characteristics" – a characteristic of 'normal'

[11] Beck, *Cognitive Therapy and the Emotional Disorders*, 84.
[12] Richard G. MacGillivray and Pierre Baron, "The Influence of Cognitive Processing Style on Cognitive Distortion in Clinical Depression," *Social Behavior and Personality* 22, no. 2 (1994): 146.

cognition.[13] To repeat a previous point, these meanings are idiosyncratic, and as a result, subjectively significant losses may not seem significant to others.[14]

There are recurring triggers for depressive cognition. Beck notes that vulnerability factors, like meanings, are idiosyncratic, but that the literature has found themes common throughout, including "the disruption of a relationship with a person to whom the patient is attached; failure to attain an important goal; loss of a job; financial reverses; unexpected physical disability; and loss of social status or reputation."[15] Serious medical illness clearly can affect each of these triggers; as a result, the argument of the last chapter concerning the underdiagnosis of depressive disorders should not be surprising. Once a patient experiences a trigger for depression, we begin to see the hallmarks of affective forecasting:

> The predictions of depressed patients tend to be overgeneralized and extreme. Since the patients regard the future as an extension of the present, they expect a deprivation or defeat to continue permanently. If a patient feels miserable now, it means we hill always feel miserable. The absolute, global pessimism is expressed in statements such as 'things won't ever work out for me', 'life is meaningless…It's never going to be any different.' The depressed patient judges that, since he cannot achieve a major goal now, he never will. He cannot see the possibility of substituting other rewarding goals. Moreover, if a problem appears insoluble now, he assumes he will never be able to find a way of working it out or somehow bypassing it.[16]

As a result, we see how depression can fundamentally alter an individual's thought process, twisting and distorting his perception of reality; skewing it from realistic to unrealistic thought.

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[13] Beck, *Cognitive Therapy and the Emotional Disorders*, 91.

[14] As a quick example, while on clinical rotations, I was involved in a case consultation for a patient who wished to forgo dialysis because of how he believed it would affect his social life (he stated that he believed being dependent upon dialysis would limit his mobility, and therefore would limit how he would be able to socialize with his friends and family). This case has since caused me significant concern – when we interviewed him, it was clear that he appreciated the consequences of his decision, and that he was clear on his motivations for forgoing treatment. In light of these two factors, as an ethics consult team we recommended that his wishes be upheld, and died several days later. I cannot help questioning, however, whether the right choice was made for this patient, especially in light of the kinds of automatic cognitive processes that have been demonstrated in the past several decades. The hospital protocol required only an assessment by the psychiatric consult liaison prior to the ethics consult – the liaison assessed the patient for a psychiatric comorbidity, but did not assess or challenge the patient's overall thought process, which may have been the more benevolent course of action.

[15] Beck, 108.

[16] Beck, 117.

Decisions based upon this biased cognitive processing immediately become suspect – as clinicians, we must be aware of this phenomenon and we must be willing to challenge it. As has been said before, if a patient is going to forego treatment, we must make sure that she does so for the right reason, and not because of avolitional, automatic processes. When a source of consistent error is identified, any clinical decision of such significance as forgoing treatment must be examined in light of it.

Supporting Evidence

Support for Beck's model of cognition is significant, and a full treatment of the supporting research would require significantly more space than is available in the present work.[17] At present, we will focus on a few studies over the past two decades that have corroborated Beck's theory, through direct exploration of cognition during depression as well as psychological sequelae in other conditions (e.g., depression and anxiety, depression and pain, etc.).

Moretti and Shaw[18] provide support for Beck's postulated negative biases in self-referencing information processing in individuals experiencing a depressive disorder. They suggest that there are demonstrable differences in how depressed versus non-depressed individuals view the likelihood of personal failure or inadequacy and how they perceive external versus internal sources of positive and negative events.[19] These are all essential elements of Beck's proposed 'cognitive triad' – views of the self (self-perception and adequacy), views of

[17] There are significantly more studies that have supported Beck's model since it was proposed than I could plausibly cover here. Beck's model, and his corresponding therapeutic intervention model (cognitive-behavioral therapy) have been proposed as a paradigmatic model of empirical therapeutic intervention.
[18] Marlene M. Moretti and Brian F. Shaw, "Automatic and Dysfunctional Cognitive Processes in Depression," in *Unintended Thought*, ed. James S. Uleman and John A. Bargh (New York: Guilford Press, 1989), 383-421.
[19] Moretti and Shaw, "Automatic and Dysfunctional Cognitive Processes in Depression," 397-8.

the world (internal vs. external locus of control), and views of the future (probability of success/failure).

Mitchell and Campbell[20] explored cognitions resulting from depressive and anxiety states, and noted that individuals with depressive disorders tended to generalize inappropriately from situation to situation. They note "cognitions reflecting both helplessness and hopelessness are characteristic of depression whereas cognitions reflecting helplessness alone are more characteristic of anxiety. If the measure of generalization across situations is taken to be a measure of hopelessness then this suggestion is supported by the present results."[21] This provides support both for Beck's argument concerning generalization; further, the hopelessness model provides support for Beck's cognitive triad (view of the future) as well as the affective forecasting noted in chapter two.

Smith, et al.,[22] explored cognitive sequelae of depression in chronic pain.[23] When they contrasted the results of chronic pain patients with depressed patients, they found that:

> [D]epressed chronic pain subjects were characterized by high levels of cognitive distortion in pain-related situations and less pronounced distortion in nonpain situations. In contrast, depressed nonpain subjects displayed equally high levels of distortion in both types of situations. This suggests some situational specificity to the cognitive characteristics of depressed chronic patients that is not apparent in typical depressed persons. The tendency of depressed nonpain patients to generalize their distorted thinking to a hypothetical situation (i.e., if they had a pain problem) is consistent with the cognitive model of depression.[24]

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[20] S. Mitchell and E. A. Campbell, "Cognitions Associated with Anxiety and Depression," *Personality and Individual Differences* 9, no. 4 (1988): 837-8.
[21] Mitchell and Campbell, "Cognitions Associated with Anxiety and Depression," 838.
[22] Timothy W. Smith, Jennifer L. O'Keeffe, and Alan J. Christensen, "Cognitive Distortion and Depression in Chronic Pain: Association with Diagnosed Disorders," *Journal of Consulting and Clinical Psychology* 62, no. 1 (1994): 195-98.
[23] This has immediate relevance not only to Beck's model, but also to patients with cancer, as discussed in chapter three. Pain is a significant concern in managing the illness, and can present a significant conundrum in treatment, as severe pain may only respond to powerful pain killers (e.g., morphine), which have concomitant physical concerns (e.g., cardiac and respiratory suppression).
[24] Smith, O'Keeffe, and Christensen, "Cognitive Distortion and Depression in Chronic Pain: Association with Diagnosed Disorders," 197.

The study provides further evidence of cognitive distortion, as well as problems of generalization between situations, upholding Beck's proposed model of depression (and other cognitive models). In addition, there is further evidence of affective forecasting and the problems it creates.

Henriques and Leitenberg[25] explored several models of depression, and found support for some elements of Beck's model. They note that some of Beck's assumptions about the stability of traits have been questioned by some empirical studies:

> Further, a number of studies have found that, contrary to Beck's model, dysfunctional attitudes do not appear to be stable or trait-like, but seem to wax and wane with depressed mood, suggesting that dysfunctional attitudes might result from depressed mood rather than vice-versa. A possible reason for this ostensibly contradictory result is that dysfunctional attitudes remain latent or inaccessible until an individual is confronted with a negative circumstance that activates the beliefs.[26]

They note that Beck's proposed model is essentially a diasthesis-stress model, in which latent vulnerabilities are only activated by exposure to environmental stressors, which would explain the apparent incongruity. Their study further found that negative social feedback predicted alteration in social self-esteem and changes in mood, following Beck's proposed model (although some evidence supported other theories in the etiology of depression [e.g., negative-to-positive cognitive error ratios as a measure of distortion]).[27] They conclude by noting that Beck's proposed cognitive errors (overgeneralizations, abstractions, catastrophizations, etc.) may have a critical role in the etiology of depression, and therefore warrant significant further research.

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[25] Gregg Henriques and Harold Leitenberg, "An Experimental Analysis of the Role of Cognitive Errors in the Development of Depressed Mood Following Negative Social Feedback," *Cognitive Therapy and Research* 26, no. 2 (April 2002): 245-60.
[26] Henriques and Leitenberg, "An Experimental Analysis of the Role of Cognitive Errors in the Development of Depressed Mood Following Negative Social Feedback," 246.
[27] Henriques and Leitenberg, 257.

Challenges to Beck's Theory

There is, however, some disagreement regarding the validity of Beck's model. For example, although Moretti and Shaw supported Beck's theory regarding the content and valencing of self-referencing information, they did not support his characterization of these thoughts as automatic, a key aspect of Beck's model.[28] In fact, their review of the literature noted three key trends in the literature.[29] First, as of the time of the writing of their article, few studies had explored whether the self-denigrating content of such cognitions actually were automatic – the studies cited as support tended to focus on the valence of the cognition, and not its speed or source. Second, they noted methodological problems with other studies purporting to demonstrate automaticity – researchers had a tendency to inappropriately interpret response latencies as directly indicative of the degree of automaticity without considering confounding or nuisance variables.[30] Finally, they note that there are other important aspects of depressive cognition that warrant further exploration, e.g., whether the automatic processes are susceptible to inhibition or control by the individual. These are all serious concerns, and may confound Beck's model. Nevertheless, even though they may not necessarily offer proof, they do note that the available data *are* consistent with Beck's theory, and they urge further research exploring "the contexts that are most likely to give rise to dysfunctional processing, the role of affect in dysfunctional automatic processing, and the extent to which dysfunctional processes can be interrupted and altered."[31]

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[28] Moretti and Shaw, "Automatic and Dysfunctional Cognitive Processes in Depression," 398.
[29] Moretti and Shaw, 406.
[30] Confounding variables are other explanations for the target behavior that were not either not predicted or were not accounted for in the experiment (they 'confound' the experiment: they make it unclear whether the observations were due to the manipulated variable or due to some unknown variable). Nuisance variables are potentially causative factors that have been identified, and which then become further independent variables in the experiment (i.e., they are a nuisance because they are a causal factor that cannot easily be removed from the experimental conditions).
[31] Moretti and Shaw, 412.

MacGillivray and Baron[32] studied cognition in depressed women which yielded findings contrary to what one would expect under Beck's model. Specifically, three key differences were noted.[33] First, the errors committed by the depressed women might actually have been due to a longstanding cognitive style, instead of being idiosyncratic to a depressive state. The women studied may have *always* thought in a particular manner, and did not demonstrate significantly different thoughts following the onset of a depressive state – any errors that may have been produced may not have been caused by their depressive state. Since Beck's model is predicated on demonstrable differences between 'normal' and dysfunctional cognition, this is a serious and fundamental challenge. Second, they did not note an interaction between quality of life events and the cognitive style of the individual. As Beck proposes a diasthesis-stress model, the lack of an interaction undercuts the idea that situational variables are critical components in the etiology of depression. MacGillivray and Baron suggest that cognitive styles are more pervasive than situation-specific. Third, differences between the women studied did not produce variations or differences in the degree of depression – if stressors are interpreted idiosyncratically, one would expect differences in symptom severity, which did not occur.

Beck's theory is not static – it has been modified since it was first proposed. For instance, following the depressive realism studies (see below), Beck would revise his theory to suggest that individuals with more severe forms of depression would demonstrate cognitive distortions, while dysphoric and nondepressed participants would demonstrate unbiased thought. Kapci and Cramer, however, noted that inconsistencies would still remain, as both of these

[32] MacGillivray and Baron, "The Influence of Cognitive Processing Style on Cognitive Distortion in Clinical Depression."
[33] MacGillivray and Baron, "The Influence of Cognitive Processing Style on Cognitive Distortion in Clinical Depression." 154-5.

groups demonstrated their own types of distortions and biases.[34] The objections covered here are not the full extent of the objections raised to Beck's model, but are indicative of the kinds of challenges, both methodological and conceptual that have been raised. By far, however, the biggest challenge to Beck's theory of cognitive distortion is the phenomenon of depressive realism, to which we now turn.

The Theory of Depressive Realism

Alloy and Abramson

The most frequently cited article in the depressive realism literature is Alloy and Abramson's discussion of four models of depressive cognition. They note that a fundamental paradox has emerged from recent studies – the commonly accepted model of depressed cognition was that of cognitive distortion, but that a trend in research had emerged in which depressed individuals actually demonstrated more accurate or realistic perceptions and inferences than nondepressed people.[35] They note that Beck and other cognitive theorists have proposed systematic maladaptive cognitive schemata in depressed individuals, which led them in selectively abstracting negative elements in their environment, overgeneralizing other elements, and making arbitrary inferences, all of which are the results of rigid schemata automatically applied to particular situations.[36] These schemata apply to analysis of the self, the world, and the future (Beck's cognitive triad – see above); Beck's analysis further argues that cognitive biases and distortions are not found in normal, nondepressed individuals.[37]

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[34] Emine G Kapci and Duncan Cramer, "The Accuracy of Dysphoric and Nondepressed Groups' Predictions of Life Events," *Journal of Psychology* 132, no. 6 (November 1998): 667.
[35] L.B. Alloy and L.Y. Abramson, "Depressive Realism: Four Theoretical Perspectives," in *Cognitive Processes in Depression*, ed. L.B. Alloy (New York: Guilford Press, 1988), 223.
[36] Alloy and Abramson, "Depressive Realism: Four Theoretical Perspectives," 225.
[37] This is ironic, in that systematic cognitive errors and biases were seen to be part of everyday, "normal" cognition in chapter two of the present work.

Alloy and Abramson note that depression is likely heterogeneous – as has been noted in Chapter three, there are, in fact, a family of depressive disorders, with distinct etiologies. Because of this diversity, they suggest that the arguments made by cognitive theorists may be applicable only to specific diagnoses (i.e., depressions with concomitant negative cognitions, instead of other forms of the illness). They note that individuals with inferential biases are not necessarily inaccurate – the objective circumstances can be congruent or incongruent with the conclusions they reach.[38] This leads, they argue, to difficulty in understanding rationality and irrationality – they propose defining rationality as 'realism over the long run',[39] in that the cognitive biases that a person utilizes may serve them well in the long run.[40] If such a 'long run' perspective is adopted, then it would make sense for the person to adopt them in the present situation, even if the bias is contradicted by the available information and leads to immediate errors – it might be 'rational' to make mistakes.[41]

The experiments which bolstered Alloy and Abramson's claims dealt with the perception of control in experimental conditions – students were asked to judge the degree of control their responses had over the illumination of a light in contingent and non-contingent response-outcome scenarios. If Beck's assumptions about depressive cognition were accurate, it would be

[38] This objection, I think, is significant, in that Beck's approach *assumes* a level of distortion, potentially inappropriately. For instance, a typical example of Beck's cognitive therapy is to imagine oneself walking down a street and observing a pair of individuals approximately fifty feet away. These individuals turn to look at you, turn back to each other, and laugh. The depressed patient infers from the scenario that the individuals in question were laughing about him or her, which tends to produce feelings of anger. The therapist then examines the thought process that gave rise to the emotion, suggesting that there are other ways of interpreting the situation (e.g., the pair registered the presence of the patient, but were discussing a funny program they had seen, which made them laugh). This is designed to alter the thought process and help alleviate the affective consequences of that situation. The problem, however, is that it is possible that the individuals in question *were* talking about the patient – in this scenario, the patient is accurate in his or her assessment, and the therapist has introduced a distortion!

[39] Alloy and Abramson, "Depressive Realism: Four Theoretical Perspectives," 227.

[40] A comparable argument is made by Gigerenzer and others regarding the evolutionary advantages of fast and frugal heuristics (see chapter two).

[41] This point will require clarification, as it leads to a counterintuitive ethical analysis. I would argue that despite the supposed 'rationality' of allowing individuals to apply long-term biases to end-of-life decisions, which would seem to be the logical consequence were Alloy and Abramson's analysis applied to these situations, it would be justifiable to challenge the bias. This point will be developed more fully later in this chapter.

expected that the depressed participants would demonstrate a bias or inaccuracy in assessing the contingency. However, the experiment yielded results which directly contradicted Beck's argument:

> It was only nondepressed students who systematically erred in judging their control. Nondepressives exhibited an 'illusion of control' and overestimated their control over uncontrollable outcomes that occurred with high frequency or that were associated with success. In addition, nondepressives showed an 'illusion of no control' and underestimated their impact on controllable outcomes associated with failure. Nondepressives also underestimated their control over controllable outcomes when the passive response of not pressing was associated with greater success.[42]

The non-depressed participants overestimated their control for successes and underestimated their control for failure; these offer evidence that instead of a depressive bias (or 'pessimistic bias' in Beck's analysis), non-depressed individuals actually exhibit an optimistic bias, an illusory and self-promoting image of themselves consistently applied in action and cognition. Alloy and Abramson interpret their results as demonstrating that depressed individual's judgments may not be distorted when they have had the chance to perceive the contingencies of their situation. They note that past research has supported this analysis – as depressed individuals were exposed to non-contingency situations, their perception of control became more realistic, while non-depressed people maintained their perception of control.[43] Generalizing from their own studies and others, they conclude that "depressed individuals appear to be less susceptible to 'illusions of success' than nondepressed individuals for themselves (and perhaps in private) but more susceptible to 'illusions of success' for others.[44]

Other areas of study in depressive realism concern ambiguous and unambiguous personality feedback. Again, if the cognitive model of Beck were accurate, then the

[42] Alloy and Abramson, "Depressive Realism: Four Theoretical Perspectives," 228.
[43] Alloy and Abramson, 230.
[44] Alloy and Abramson, 233.

interpretations offered by the depressed research participants should demonstrate perceptual biases that render their interpretation of the data inaccurate. The experiments yielded compelling results along a continuum – both depressed and non-depressed participants demonstrated some optimistic bias; however, the depressed groups consistently demonstrated less optimistic bias than the non-depressed groups. They note that, generally, "the more positive an individual's self-schema, the more positive the degree of bias he or she showed in ambiguous-feedback perception."[45]

The difference between depressed and non-depressed individuals' cognitive approaches and schemata is explicable when one explores the content of each. Alloy and Abramson suggest that the relative absence of optimistic bias in the cognition of depressed individuals may be due to a balance between negative and positive content, in contrast to disproportionate ("differentiated") positive content in non-depressed individuals. Analysis of self-concepts supports this suggestion, as there are empirical data demonstrating that non-depressed people have a "strong, positive self-schema", while mild and moderately depressed people have schemata containing "mixed and rather balanced positive and negative content."[46] Support for the different schemata can be found in studies of the effects of self-directed attention (studies of self-focus). Studies have demonstrated that self-focused attention increases the accuracy of self-reporting and self-referent judgments, and that depressed individuals may demonstrate more self-focus than non-depressed individuals.[47]

All of these studies demonstrate the depressive realism; while Alloy and Abramson note a variety of perspectives on the data presented, they argue for what they term the "naïve"

[45] Alloy and Abramson, "Depressive Realism: Four Theoretical Perspectives," 236.
[46] Alloy and Abramson, 250.
[47] Alloy and Abramson, 251.

perspective ('naïve' in the sense that it is one which takes the empirical data at face value). This has three main consequences for our understanding of depressive cognition:

> If the naïve perspective is correct, then three intriguing implications may follow. First, the perspective would suggest that Beck's cognitive model of depression, with its emphasis on negative depressive distortions, is wrong and that at least one psychopathological group is less rather than more susceptible than normals to cognitive irrationality and distortion. Second, this perspective raises the possibility that a realistic and unbiased perception of oneself and one's relation to the world contributes to the cause or maintenance of depression. Finally, the naïve perspective suggests that cognitive therapy for depression, a demonstrably effective program for treating depression, may work *not* by enhancing the realism of depressives' cognitions, as is currently assumed, but by training depressives to construct for themselves the kinds of optimistic biases and distortions typically exhibited by nondepressed people.[48]

There is a necessary caveat to this discussion – Alloy and Abramson note that depressive realism is a phenomenon found in only some degrees of depression (mild and moderate); severe depressions do exhibit the maladaptive and unrealistic distortions Beck proposes.

All of this leads Alloy and Abramson to argue that the experience of depression is the dysfunction of normal optimistic biases and distortions. "Healthy personal illusions" may be a hallmark of everyday cognition – this is to say that our cognitive schemata are normally self-biasing, producing feelings of competency, worth, and ability greater than what we actually possess. They stress that this facet of cognition warrants further research; while cognitive psychology has made inroads into optimistic biases, there are still important aspects of cognition to be addressed, like the boundaries between depressive realism and depressive distortion.[49]

[48] Alloy and Abramson, "Depressive Realism: Four Theoretical Perspectives," 253.
[49] Alloy and Abramson, 257.

Studies providing supporting evidence

Support for the depressive realism hypothesis comes from a variety of sources. Greenberg, et al.,[50] elaborate upon the self-schema concept. They note that schemata separate, filter, and compartmentalize the myriad stimuli we constantly experience. In doing so, schemata function as a means of "cognitive economy"; they direct our attention to particular aspects of the situation, instead of attempting to take in the situation in its entirety – as such, elements inconsistent with the schema is ignored, while others are adapted to make them consistent. These processes introduce elements of bias and distortion.[51] Studies of schemata are prevalent in cognitive, social, and personality psychology, and the mechanisms by which they perform analyses and introduce errors have been given particular attention:

> In particular, personality studies have supported the existence of a well-organized self-schema that influences a person's endorsement of trait adjectives as personally descriptive, increases the efficiency or speed of processing stimuli that match the self-schema content, and enhances the recall of schema-consistent information while at the same time producing erroneous recall and recognition of self-schema-congruent material that was never presented."[52]

They note that studies have suggested different contents for self-schema among depressed and non-depressed participants, in line with Alloy and Abramson's above analysis. However, they note that there are differences in the findings regarding depressed participants' schemata; some studies note explicitly negative schema content, others note unstable or mixed negative/positive schema content. If the schemata are of the mixed variety, the cognitions they produce will shift as the balance between negative and positive content shifts.[53]

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[50] Michael S. Greenberg, Carmelo V. Vazquez, and Lauren B. Alloy, "Depression Versus Anxiety: Differences in Self- and Other-Schemata," in *Cognitive Processes in Depression*, ed. Lauren B. Alloy (New York: Guilford Press, 1988), 109-42.
[51] Greenberg, Vazquez, and Alloy, "Depression Versus Anxiety: Differences in Self- and Other-Schemata," 114.
[52] Greenberg, Vazquez, and Alloy, 115.
[53] Greenberg, Vazquez, and Alloy, 116.

Their own study yielded several relevant findings.[54] First, the 'normal' subjects were the only group that consistently favored positive over negative adjectives in self-description, and tended to make self-judgments which endorsed nearly all positive adjectives and rejected nearly all negative adjectives. Second, non-depressed participants demonstrated faster responses (interpreted as greater processing efficiency) for positive than for negative self-references, and demonstrated comparable speed in their rejection of negative self-references. Third, the non-depressed participants were the only group not to demonstrate enhanced recall for certain depression- and anxiety-related adjectives. Depressed participants' schemata demonstrated consistently mixed or balanced positive and negative content. They did not demonstrate any particular preference for either positive or negative depression- or anxiety-related adjectives, nor did they have the kind of extreme results found in the non-depressed participants. They demonstrated comparable recall times for both positive and negative self-describing adjectives, and demonstrated comparable recall for both positive and negative depression-related adjectives. These findings, they suggest, offer support for a non-depressed cognitive schema that exhibits unrealistic optimism, and suggest that there is no corresponding bias in depressed cognitive schemata.[55]

A second pillar of support is found in Rehm's analysis of the inferences made by depressed individuals,[56] noting that several characteristics emerge. First, a recurrent theme in the literature stresses that the life experiences of depressed people differ significantly than nondepressed people. Depressed people are more likely to have had fewer positive and more

[54] Greenberg, Vazquez, and Alloy, "Depression Versus Anxiety: Differences in Self- and Other-Schemata," 130-2.
[55] Greenberg, Vazquez, and Alloy, 132.
[56] Lynn P. Rehm, "Self-Management and Cognitive Processes in Depression," in *Cognitive Processes in Depression*, ed. Lauren B. Alloy (New York: Guilford Press, 1988), 143-76.

frequent negative life events,[57] which can lead to and reinforce a particular schema for interpreting the world around them.[58] This affected cognitive schema does not distort their perception of the outside world (i.e., paralleling the depressive realism hypothesis) – depressed individuals are more accurate in reporting specific life events. Depression does lead to a different perspective on how they interpret information, however – Rehm notes that ambiguity and abstraction in self-referential information tend to be interpreted more negatively:

> Inferences, interpretations, and judgments should be viewed as internal processing variables rather than as input variables. Inference in judgmental processes has received a great deal of attention, but different theories have postulated different types of inferences as the core factors in determining depression. Although the evidence is somewhat mixed, there is a trend suggesting that depressed individuals make negative attributions about failures, and perhaps about successes. Depressed individuals are more negative in their estimates of their own ability to produce desired responses (self-efficacy). They do not, however, seem to be unrealistic in objectively making predictions or offering expectancies about their performance. This seems quite consistent with the idea that depressed persons are accurate in their perceptions. The only exceptions to accurate expectancy would be in those areas in which depressed persons may, in reality, perform more poorly (e.g., in social skills). It is quite clear that depressed persons evaluate themselves negatively, and it appears that a discrepancy between expectancy and standards is the determining factor.[59]

As noted in chapter two, affective valence is an essential element of memory and cognition. Rehm notes that a lifetime of negatively valenced memories and experiences can produce a particular cognitive schema that affects future cognition. Information that was neutrally valenced would not necessarily be organized by this schema in the same manner as negatively-valenced information, and as a result, it might not be accessed as readily. In fact, Rehm suggests that individuals may actually find it difficult to access neutral information when they are in a depressed state.[60] Rehm suggests that this cognitive modeling schema has therapy implications

[57] Rehm, "Self-Management and Cognitive Processes in Depression," 167.
[58] Rehm, 169.
[59] Rehm, 168.
[60] Rehm, 169.

as well, and that depressive cognition could be challenged in an effort to access positive cognition.

Miller and Moretti[61] suggest that differences in information processing also lead to differences in causal attribution. Part of the difficulty of the issue of depressive realism versus distortion in cognition stems from difficulties in assessing what constitutes a normative model of cognition – questions about accuracy in perception may be more appropriate when rephrased in terms of which type of cognition is more rational. Miller and Moretti stress that the different experiences of depressed and non-depressed individuals must be understood, assessed, and considered if we are to make sense of their self-representations.[62] They note that the available data, however, suggest that depressed individuals tend to be less self-serving in their causal attributions (e.g., statements of control).[63] In light of the optimistic bias that has emerged in the depressive realism research, being less self-serving may reflect a tendency to avoid unwarranted optimism in self-assessment and judgments of efficacy (causality). Miller and Moretti propose that differences in information processing may be indicative of a deeper difference in seeking explanations:

> It is possible that the most significant difference between depressives and nondepressives lies in the presence or absence of the tendency to ask 'Why?' questions and to seek explanations for the events in their lives. We know that there are individual differences in the inclination to ponder causal questions, and this may be one of the differences between depressives and nondepressives. Indeed, recent studies have demonstrated that depressives differ from nondepressives in their use of attributional information.[64]

Perhaps nondepressed individuals simply do not seek explanations of causal factors (accurately) perceived to be outside of their control.

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[61] Dale T. Miller and Marlene M. Moretti, "The Causal Attributions of Depressives: Self-Serving or Self-Disserving?" in *Cognitive Processes in Depression*, ed. Lauren B. Alloy (New York: Guilford Press, 1988), 266-88.
[62] Miller and Moretti, "The Causal Attributions of Depressives: Self-Serving or Self-Disserving?" 277.
[63] Miller and Moretti, 281.
[64] Miller and Moretti, 282.

Evans and Hollon[65] suggest that the differences in information processing between depressed and non-depressed individuals parallel the cognitive heuristics proposed by Tversky and Kahneman (see chapter two) – specifically, the fact that the heuristics operate in a non-normative function, which can produce errors or biases. They note quite plainly that the information processing approaches in non-depressed individuals "appear frequently to operate in nonnormative ways."[66] This is especially problematic, in that non-depressed individuals readily generate new explanations for the information they receive, but are quite reluctant to revise these explanations.[67] Simply put, the positions created by optimistic biases tend to be very resistant to change. This is a phenomenon comparable to having pet theories – we force new information to fit the theory, instead of the other way around. Evans and Hollon suggest that Beck's depressive distortion and the phenomenon of depressive realism are examples of cognitive heuristics:

> *The distortions in information processing described by Beck as operating in depressives are actually representatives of the ubiquitous class of events labeled 'heuristics' by Tversky and Kahneman.* In discriminating between the accuracy of beliefs and the normativeness of information processing, we would argue that both depressives and nondepressives may be inaccurate in many of their beliefs, although typically in opposite ways; at the same time, both may share the same general nonnormative heuristics. We know of no empirical evidence that speaks either for or against this premise, but we think the parallels between heuristics in nondepressives and 'distortions' in depressives are so striking as to merit study.[68]

They are so confident of this that they suggest that neither the depressive distortion nor the depressive realism phenomena will be tenable in the long-run. Instead, they suggest that the particular schema by which information is acquired and processed by depressed and nondepressed individuals will be found to be simply opposing and predictable heuristics (and

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[65] Mark D. Evans and Steven D. Hollon, "Patterns of Personal and Causal Inference: Implications for the Cognitive Therapy of Depression," in *Cognitive Processes in Depression*, ed. Lauren B. Alloy (New York: Guilford Press, 1988), 344-77.
[66] Evans and Hollon, "Patterns of Personal and Causal Inference: Implications for the Cognitive Therapy of Depression," 350.
[67] Evans and Hollon, 351.
[68] Evans and Hollon, 352.

that both of which can lead to inaccuracy). In the cases of mild depression, individuals may make more accurate decisions simply because they use the heuristics of both depressed and non-depressed individuals. As a final caveat, they note that they "cannot, however, rule out the possibility that at least mild depressives are universally accurate, while nondepressives are optimistically inaccurate. Individuals who evidence low levels of depression may do so as a result of viewing themselves and their environments more critically, and hence accurately."[69] While this amounts to less enthusiastic support, it is still support nonetheless.

Rosenfarb and Burker[70] found differences in sensitivity to changing contingencies between depressed and nondepressed people. They suggest that depressed individuals may be less sensitive to externally applied rules when those rules become inaccurate.[71] This, in turn, allows them to be more accurate in their judgments since they are no longer bound to rules that lead to mistakes.

Koenig, et al.,[72] explored the optimistic bias phenomenon in greater detail, but found mixed results favoring both the depressive distortion and depressive realism models. They found that depressed individuals were less accurate in both self-evaluations as well as self-versus-other evaluations, which they note supports Beck's proposed model of depressive distortion. However, they found that there was no evidence that the depressed individuals' self-inaccuracy was due to systematic distortions of self-relevant evaluations; instead, the differences emerged because the depressed individuals were less derogatory towards others than the non-depressed participants. They hypothesize that this tendency to be derogatory towards others serves to

[69] Evans and Hollon, "Patterns of Personal and Causal Inference: Implications for the Cognitive Therapy of Depression," 356.
[70] Irwin S. Rosenfarb and Eileen J. Burker, "Effects of Changing Contingencies on the Behavior of Depressed and Nondepressed Individuals," *Journal of Abnormal Psychology* 102, no. 4 (November 1993): 642-46.
[71] Rosenfarb and Burker, "Effects of Changing Contingencies on the Behavior of Depressed and Nondepressed Individuals," 646.
[72] Linda J. Koenig, Ann B. Ragin, and Martin Harrow, "Accuracy and Bias in Depressive's Judgments for Self and Other," *Cognitive Therapy and Research* 19, no. 5 (1995): 505-17.

maintain a self-enhancement/self-esteem function (part of the self-serving, optimistic bias).[73] They note that their findings appear to conflict with Alloy and Abramson, but they then propose that this discrepancy may be due to having examined more depressed subjects (i.e., subjects who were more than mild to moderately depressed). They suggest that there are enough supporting studies to call into question some of Beck's assumptions regarding depressive cognition, specifically the assumption that depressive cognition is unrealistic.[74]

Johnson and DiLorenzo[75] found that both depressed and non-depressed individuals demonstrate accuracy and error in cognition – specifically they are more accurate in interpreting schema-congruent information. While they note that this offers support for schema-influenced processing, they note that it is unclear exactly how self- and other-schemata produce the biases in question.

McKendree-Smith and Scogin[76] explore the difference between milder forms of depression and more severe forms, underscoring the differences in processing between the two (and between both and non-depressed individuals). In analyzing previous experiments, they note that:

> In 20 of 23 studies involving ambiguous information or events, nondepressed participants showed optimistic biases. However, they showed these biases in only 11 of 18 studies using unambiguous information. Depressed participants showed pessimistic distortions in only six of 23 studies providing information but in eight of 18 studies using unambiguous information. These results suggest that depressed people do not negatively bias ambiguous information. However, the evidence is not conclusive.[77]

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[73] Koenig, Ragin, and Harrow, "Accuracy and Bias in Depressive's Judgments for Self and Other," 513.
[74] Koenig, Ragin, and Harrow, 515.
[75] Thomas J. Johnson and Thomas M. DiLorenzo, "Social Information Processing Biases in Depressed and Nondepressed College Students," *Journal of Social Behavior and Personality* 13, no. 3 (September 1998): 517-30.
[76] Nancy McKendree-Smith and Forrest Scogin, "Depressive Realism: Effects of Depression Severity and Interpretation Time," *Journal of Clinical Psychology* 56, no. 12 (2000): 1601-08.
[77] McKendree-Smith and Scogin, "Depressive Realism: Effects of Depression Severity and Interpretation Time," 1602-3.

Clearly, further and more refined study is necessary to get an accurate picture of depressed versus non-depressed cognition and distortion.

The literature suggests that distortion occurs in both depressed and non-depressed populations, but it is possible that it occurs at different times. They note that depressed participants tended not to show bias when discussing immediate perceptions (unlike the non-depressed participants); however, they *did* display bias when recalling information later or thinking about it further[78] – time may be the variable in transforming unbiased accuracy to biased inaccuracy.[79] They note that their study supports the hypothesis that depressive realism is moderated by the severity of the patient's depression. Specifically, individuals with mild forms of depression were relatively accurate in their assessments, non-depressed participants tend to exhibit positive biases, and moderately/severely depressed participants tend to exhibit negative biases.[80] They suggest, however, that further research is warranted.

Challenges to Depressive Realism

Depressive realism has proven to be a very controversial issue, and like Beck's theory, finds itself challenged at fundamental levels. As with the discussion of Beck, instead of providing a survey of the entire body of literature, it will suffice to focus on a smaller group of studies over the past fifteen years critiquing Alloy and Abramson's position.

[78] This potentially makes the issue of the choice to forgo medical treatment even murkier. If the heuristics and biases perspective is accurate, then decisions are spur of the moment and potentially more accurate than choices stemming from longer rumination. However, we encourage patients to think about their choices, a process which leads them to rumination and recall (and hence, greater distortion), which is already subject to sources of error as demonstrated by the availability heuristic. Taking some time to think about the choice to forgo treatment may exacerbate the depressive disorder itself, as well as potentially increasing the likelihood of an error in cognition occurring!

[79] McKendree-Smith and Scogin, "Depressive Realism: Effects of Depression Severity and Interpretation Time," 1603.

[80] McKendree-Smith and Scogin, 1606-7.

Campbell and Fehr[81] explored perceptions of self-concept in individuals of varying degrees of self-esteem. They also explored the role of "negative affectivity" in judgments of observer feedback. They noted that, contrary to what one would expect from the depressive realism model, individuals with high negative affectivity do have more pessimistic views of themselves and the world, but that one ought to be skeptical as to whether this provides a more objectively accurate perspective.[82] They note that they have only tested judgments of conveyed impressions, but suggest that their results might be extended to other areas of cognition and judgment, but only after empirical testing. They reject the characterization of depressed individuals as 'sadder, but wiser':

> Many current texts in psychology portray people high in NA as 'sadder, but wiser.' On the basis of the research evidence to date, this characterization seems clearly inappropriate with respect to knowing how others view the self. These individuals are certainly sadder; they hold more negative beliefs about themselves and the impressions they convey to others. This general pessimism, however, provides no special access to wisdom because others' impressions of a given individual exhibit considerable variability over people, time, and roles (e.g., partners vs. observers). When reality itself is not constant, any stereotypical view of that reality sometimes will be right, but also sometimes will be wrong.[83]

As such, any bias or distortion that is systematically applied to judgments will occasionally be accurate, in the same manner that a broken watch will still tell the right time twice a day. They note that the research on depressive realism tends to ignore the relative adjustment of low versus high negative affectivity – despite occasional sources of error in individuals with low negative affectivity, they are generally better adjusted, healthier, and adapt much more readily than high negative affectivity.

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[81] Jennifer D. Campbell and Beverley Fehr, "Self-Esteem and Perceptions of Conveyed Impressions: Is Negative Affectivity Associated with Greater Realism?" *Journal of Personality and Social Psychology* 58, no. 1 (January 1990): 122-33.
[82] Campbell and Fehr, "Self-Esteem and Perceptions of Conveyed Impressions: Is Negative Affectivity Associated with Greater Realism?" 130.
[83] Campbell and Fehr, 131.

Ackermann and DeRubeis[84] surveyed the literature on depressive realism in a variety of contexts and noted that the findings are mixed. Specifically, the accuracy of depressed and dysphoric individuals appears to be contingent upon the kind of task examined:

> Upon reviewing the depressive realism literature, two things become evident. First, although many of the findings are consistent with the hypothesis, almost as many are inconsistent with the hypothesis. Second, the findings appear to vary systematically as a function of the type of task used in the studies. Whereas in the contingency judgment and self-other studies the dysphoric subjects tend to be more accurate or evenhanded than the nondepressed subjects, in the recall of evaluative feedback studies the nondepressed subjects were more accurate than their depressed and dysphoric counterparts in every study but two.[85]

This appears to contradict the assumptions of Alloy and Abramson's theory – again, there are times when the resultant cognitive schema are in fact more accurate, and times when they produce further errors. Extending this analysis, Ackermann and DeRubeis suggest that both depressed and nondepressed individuals employ cognitive schemata, which will both have corresponding accurate and inaccurate situations. Anticipating later studies[86] (and offering some support for depressive realism), Ackermann and DeRubeis note that the studies demonstrating accuracy frequently used dysphoric, instead of depressed individuals, and suggest that accuracy may result from the more mild and moderate forms of depression, instead of severe cases,[87] but note that other explanations are also possible (e.g., motivational factors may be present or absent).

[84] Ruby Ackermann and Robert J. DeRubeis, "Is Depressive Realism Real?" *Clinical Psychology Review* 11 (1991): 565-84.
[85] Ackermann and DeRubeis, "Is Depressive Realism Real?" 579.
[86] For instance, J.A. Hancock, A.P.R. Moffoot, and R.E. O'Carroll, ""Depressive Realism" Assessed Via Confidence in Decision-Making," *Cognitive Neuropsychiatry* 1, no. 3 (1996): 213-20.
[87] Ackermann and DeRubeis, "Is Depressive Realism Real?" 580.

Dunning and Story[88] examined depressed participants' views of the future, and found that contrary to the depressive realism hypothesis, depressed individuals were more unrealistic in their assessment of the likelihood of future events. Reviewing the literature critiquing the depressive realism hypothesis, they note two key criticisms.[89] First, there is concern about the normative standards employed by previous experiments – it was assumed to be possible to classify one set or pattern of responses as accurate or normative, and that therefore others were dysfunctional. This seems quite unlikely, as cognition is idiosyncratic.[90] However, it does seem clear that there are interpretations of the world that are completely at odds with the actual phenomena.[91] The second criticism concerns the ecological validity of the depressive realism phenomenon – the studies conducted were laboratory experiments, which may not necessarily translate into everyday behaviors. If a phenomenon cannot be reproduced outside of carefully controlled situations, a fundamental question must be raised as to whether that phenomenon is a valid representation of normal behavior.

The conceptual criticisms aside, Dunning and Story note that the results of their study yielded a fundamental challenge to depressive realism: depressed individuals were actually less realistic about their anticipations of future events than non-depressed individuals. This strikes at the core of the depressive realism hypothesis, which assumes greater realism in depression.

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[88] David Dunning and Amber L. Story, "Depression, Realism, and the Overconfidence Effect: Are the Sadder Wiser When Predicting Future Actions and Events?" *Journal of Personality and Social Psychology* 61, no. 4 (October 1991): 521-32.

[89] Dunning and Story, "Depression, Realism, and the Overconfidence Effect: Are the Sadder Wiser When Predicting Future Actions and Events?" 522.

[90] Idiosyncratic in more than the sense proposed by Beck. How we think is a unique reflection of a variety of learned and inherited mechanisms – as indicated in chapters one and two, there are myriad genetic and environmental influences on how we process information. Further, the experiences we have had in life offer further influences which are generally unique to us – the likelihood of having the *exact* same experience as someone else (i.e., employing *exactly* the same cognitive structures with *exactly* the same relevant memories and *exactly* the same interpretation) is quite low.

[91] In making this claim, I think back to a patient I worked with on the behavioral health unit who maintained psychotic beliefs about her family being imposters, another who attempted to eat a sandwich while holding it six inches from his mouth, etc. These beliefs may be *subjectively* accurate (i.e., in line with the individuals' beliefs at that time), but are *objectively* inaccurate, in that they are disconnected from reality.

While they note that their study found some differences in the confidence of the perception between the two groups, they stress that non-depressed individuals were more realistic:

> The results of both studies converged to a single and surprising answer: Depressed individuals were less realistic about their futures. In both studies, the level of accuracy they achieved was lower than that attained by their nondepressed counterparts. In addition, across the two studies, they proved to be more overconfident in the predictions they rendered. In sum, in our mildly depressed student sample, we did not find any evidence of the realism observed in past social psychological work. If anything, we found the exact opposite. We should note, however, that although nondepressed subjects made more realistic judgments than depressed participants, this is not to say that the nondepressed were realistic and the depressed were not. Both groups displayed unrealistic confidence in the likelihood that their predictions would prove accurate.[92]

As a result, they suggest that depressive realism may reflect a tendency in certain situations, rather than a uniform phenomenon in depressive cognition.

Dobson and Pusch[93] note that while depressive realism has been replicated in experiments, it is becoming clear that boundary conditions are being identified.[94] Noting that Abramson and Alloy tested college students, Dobson and Pusch focused on the cognition of depressed versus nondepressed women in judgments of contingency. They note that depressive realism would suggest that the depressed women would be more accurate in assessing contingency; this hypothesis, however, was not upheld – the depressed subjects overestimated their control significantly. These results, they argue, suggests that a phenomenon observed in college students may not be generalizable to other populations.[95] In fact, they note that there are reasons to doubt the ecological validity of other experiments supporting depressive realism (as noted by Dunning and Story) – the results from experiments attempting to overcome the

[92] Dunning and Story, "Depression, Realism, and the Overconfidence Effect: Are the Sadder Wiser When Predicting Future Actions and Events?" 529.
[93] Keith S. Dobson and Dennis Pusch, "A Test of the Depressive Realism Hypothesis in Clinically Depressed Subjects," *Cognitive Therapy and Research* 19, no. 2 (April 1995): 179-94.
[94] Dobson and Pusch, "A Test of the Depressive Realism Hypothesis in Clinically Depressed Subjects," 180.
[95] Dobson and Pusch, 191.

ecological validity criticism do not offer much support for the phenomenon. They note that "[r]esults from these studies have tended to be somewhat contradictory, leading to the suspicion that while depressive realism may be relatively easy to identify in a laboratory setting, as researchers move into domains that have greater personal relevance to subjects the phenomenon becomes increasingly elusive."[96]

Albright and Henderson[97] explored social comparisons in cognition between depressed and nondepressed individuals. They noted that dysphoric individuals had a tendency to rate themselves unfavorably in comparison with others (i.e., promote others and put themselves down), while non-dysphoric individuals tended to rate themselves favorably (i.e., they had a tendency to put others down and be self-promoting). However, while both groups demonstrated persistent schemata in information processing, Albright and Henderson argue that it would be inaccurate to claim that depression produces the more realistic cognition between the two:

> Finally, it is important to note that both groups demonstrated distortion; thus, interpretations of depressive realism which suggest that depression represents an absence of distortion may be incorrect. One hypothesis is that realism may occur only for mildly depressed or dysphoric subjects, whose self-schemata may consist of both positive and negative content, but not for individuals who are more severely depressed, who should demonstrate consistently negative distortions. However, because distortion in the magnitude of social comparison differences occurred for the mildly depressed subjects in our sample, we propose that negative distortion, not realism, is a fundamental component of depression, and that the magnitude of distortion may increase as the severity of depression increases.[98]

Again, there appears to be empirical evidence that the phenomena of 'depressive realism' may be limited in scope, may be only be applicable in particular circumstances, or may be a misunderstanding of the appropriate depression model.

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[96] Dobson and Pusch, "A Test of the Depressive Realism Hypothesis in Clinically Depressed Subjects," 192.
[97] Jeanne S. Albright and Margit C. Henderson, "How Real is Depressive Realism? A Question of Scales and Standards," *Cognitive Therapy and Research* 19, no. 5 (October 1995): 589-609.
[98] Albright and Henderson, "How Real is Depressive Realism? A Question of Scales and Standards," 607.

Wood, et al.,[99] echo the concern that depressive realism may be a limited phenomenon – they noted significant differences between dysphoric and depressed participants. As Alloy and Abramson relied upon *dysphoric* college students, they argue that it may be inappropriate to attempt to extend the principle to *depressed* individuals, as there are notable differences:

> Although dysphoric undergraduates are certainly more plentiful and easier to recruit into research studies, we conclude that they may not provide valid models from which to extrapolate to clinically depressed patients. An alternative viewpoint is that the key factor in studying depressive phenomena is depression severity. The mean BDI score of the dysphoric subjects (14.3) was less than half that of the clinically depressed subjects 33.0). Correlational analyses revealed particularly strong associations between severity and self-confidence, which correct, only in Experiment 1, which included severely depressed patients. It may be that if we had studied more severely dysphoric undergraduates who had higher BDI scores, we may have replicated the findings obtained with patients suffering from major depression. However, we maintain that dysphoric subjects with BDI scores in the range of 12-15 do not provide a valid analogue of major depression. It is ironic that this is exactly the range of BDI scores of the dysphoric subjects used in the seminal study of Alloy and Abramson, which claimed to have established the phenomenon of 'depressive realism.'[100]

As such, these concerns about the generalizability of the depressive realism findings contain a fundamental critique of the depressive realism phenomenon – if there is something to the theory, it might make more sense to be rechristened 'depressive dysphoria.'

Pusch, et al.,[101] reaffirm the contradictory results of studies in the depressive realism literature – they note that as the control of ecological validity in a given experiment increases, the degree of support for the depressive realism hypothesis decreases.[102] In their own study, they noted that all groups under study exhibited some negative bias in their reality assessments. This

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[99] J. Wood, A.P.R. Moffoot, and Ronan E. O'Carroll, ""Depressive Realism" Revisited: Depressed Patients Are Realistic When They Are Wrong but Are Unrealistic When They Are Right," *Cognitive Neuropsychiatry* 3, no. 2 (May 1998): 119-26.

[100] Wood, Moffoot, and O'Carroll, ""Depressive Realism" Revisited: Depressed Patients Are Realistic When They Are Wrong but Are Unrealistic When They Are Right," 125-6.

[101] Dennis Pusch , et al., "The Relationships Between Sociotropic and Autonomous Personality Styles and Depressive Realism in Dysphoric and Nondysphoric University Students," *Canadian Journal of Behavioral Science* 30, no. 4 (1998): 253-65.

[102] Pusch , et al., "The Relationships Between Sociotropic and Autonomous Personality Styles and Depressive Realism in Dysphoric and Nondysphoric University Students," 254.

contradicts research suggesting that non-depressed individuals demonstrate a consistent optimistic or self-enhancing bias.[103] They suggest further that earlier experiments may have failed to account for personality styles in constructing experimental methodologies, which may be an important variable in accounting for participant's responses, cognitive processing and judgments; i.e., these earlier experiments may have been confounded. They suggest that future research ought to do more than simply explore differences in mood state – specifically, they suggest that researchers take into account mood state, personality style, and the nature of the experimental task, as these variables interact and may explain the variance in the observed behaviors. They found that when dysphoric individuals were asked to evaluate personally non-salient scenarios, they demonstrated cognition free from distortion; errors only crept in when they became personally involved in the scenarios being evaluated.

Kapci and Cramer[104] also noted that dysphoric and non-dysphoric research participants demonstrate different kinds of bias in cognition and judgment. Testing the predictions and experiences of positive and negative experiences, they found that "[t]he dysphoric participants were more accurate than the nondepressed participants in predicting which positive events they would not experience. An opposite pattern, however, emerged for negative events. The nondepressed participants proved to be more accurate than the dysphoric participants in predicting negative life events."[105] This provides further evidence for the explanation presented earlier that both depressed and non-depressed individuals display biases and distortions in their thinking. Kapci and Cramer suggest that in the case of dysphoric individuals, the pessimistic

[103] Pusch , et al., "The Relationships Between Sociotropic and Autonomous Personality Styles and Depressive Realism in Dysphoric and Nondysphoric University Students," 262.
[104] Kapci and Cramer, "The Accuracy of Dysphoric and Nondepressed Groups' Predictions of Life Events."
[105] Kapci and Cramer, 665.

expectation of the future may maintain the depressive state[106] – this may actually create a kind of self-fulfilling version of affective forecasting (e.g., "I will feel bad at some future date because I will worry about feeling bad until then.").

A final critique is offered by Stone, et al.,[107] who reaffirmed the idea that different mood states carry corresponding biases. They suggest that it is impossible to divorce depressive realism from a general negativity[108] – that is, depression carries with it behavioral and personality characteristics that fundamentally affect cognitive processing. This general negativity presents a significant concern when evaluating cognition, as it may not necessarily be clear what behaviors and decisions stem from a particular cognitive process versus a pervasive attitude. Further, Stone, et al., entertain the possibility that the negative biases may cancel out the positive biases, producing "accuracy without any true insight."[109] If this were to be the case, the judgments made by dysphoric individuals may be more accurate, but the person in question may have no real understanding of the decision or its bases. They reject this possibility, however, suggesting that a pervasive negativity may offer a better explanation: "Although it would be possible to posit that the depressed participants were realistic in their individual item judgments but not in their aggregate judgments, a more parsimonious explanation is that the depressed participants displayed a general negativity, which was manifested as reduced overconfidence in one situation and as underconfidence in the second."[110] Depressive realism, therefore, may actually represent specific situations in which inappropriate overconfidence is reduced – an accidental property, rather than anything indicative of systematic greater realism.

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[106] Kapci and Cramer, "The Accuracy of Dysphoric and Nondepressed Groups' Predictions of Life Events," 668.
[107] Eric Stone, Carrie L. Dodrill, and Natasha Johnson, "Depressive Cognition: A Test of Depressive Realism Versus Negativity Using General Knowledge Questions," *Journal of Psychology* 135, no. 6 (November 2001): 583-602.
[108] Stone, Dodrill, and Johnson, "Depressive Cognition: A Test of Depressive Realism Versus Negativity Using General Knowledge Questions," 584.
[109] Stone, Dodrill, and Johnson, 587.
[110] Stone, Dodrill, and Johnson, 598.

Which Is Right?

None of the above is meant to argue for or against either Beck's model or the depressive realism model. It should be quite clear that there is significant disagreement as to whether depression produces systematic errors in cognition, systematic realism, or situation-specific accuracy. In fact, the above discussion presents an interesting quandary. If the depressive realism hypothesis is true, then dysphoric patients are more realistic about their prognosis, and hence the decisions made when mild to moderately depressed will be a more (objectively) accurate expression of autonomy, in which case we would want to correct cognitive heuristical errors generated that are *not* affective in nature, which requires *selective* challenges to *specific* opinions. If the depressive distortion hypothesis is true, then patients are operating out of a flawed cognitive process, in which case we would want to challenge both the cognitive and the affective heuristics to make the patient's choice as accurate as possible. However, if in this process we restore them to a baseline function, we *reintroduce optimistic bias*, which is an irrational process. However, restoration to this state of optimistic bias carries with it a reduced chance of morbidity and mortality, which then may change the actual prognosis and outcome. In essence, depending on the perspective we take on the literature, either we continue down a path that fosters depression, which normally would potentially render the patient incompetent to make a choice under current standards of autonomy, or we foster a positive cognitive illusion, which creates irrational thought processes, which normally would potentially render the patient incompetent to make a choice under current standards of autonomy.

What is germane for the purposes of the larger argument is that the presence of depression in a patient is not necessarily automatically a reason to deem a patient unable to make his or her own medical decisions. However, it should offer an impetus for greater dialogue and

exploration of the choice to forgo treatment – not merely the final decision, or the experiences upon which the decision is based, but also upon the cognitive process involved. If it becomes apparent that a distortion is influencing the patient's choice, we would be remiss not to attempt to correct it – this may not necessarily change the outcome, but at least it will be for reasons not predicated on inaccuracy, misunderstanding, or distorted thought.

We turn now to a discussion of autonomy, a central principle of medical ethics. We will first discuss dominant or highly influential theoretical perspectives on patient autonomy, and will then turn to models that take into account arguments from psychology and psychiatry, as well as critiques of the traditional model from other philosophical perspectives. These perspectives offer critical insights into how we *actually* view the world. Current autonomy models don't account for the concepts stressed in chapters one, two, and in the above discussion. Instead, they stress a homuncular autonomy that is divorced from heuristical and bounded rational thought – these models are predicated upon the most basic reference to cognition, the very tip of the cognitive and decisional iceberg. This presents a fundamental problem – as they are formulated and have been developed, current autonomy models have created an idealized rational agent *that doesn't actually exist, and never has*.

AUTONOMY

The debate surrounding autonomy has demonstrated several interesting points. Paramount in this debate are the assumptions regarding human cognition – many historical and contemporary models are predicated upon a 'classical model' of reason, in which we, as rational agents, debate in a risk/benefit, maximin (maximum gain, minimum loss) manner. Lakoff and Johnson[111] note five recurring assumptions of the classical model: it is assumed to be literal,

[111] George Lakoff and Mark Johnson, *Philosophy in the Flesh: The Embodied Mind and Its Challenge to Western Thought* (New York: Basic Books, 1999).

logical (in the sense of formal logic), conscious, transcendent (disembodied), and dispassionate.[112] All of this assumptions, however, are wrong – our cognition is actually metaphorical, not literal, dependent upon framing, metonymy, and prototype-based inferences, not logical, for the most part unconscious, fundamentally embodied, and fundamentally connected to and influenced by emotion.[113] As such, the idealized conception of rationality and reason simply is not supported by the empirical evidence. Lakoff and Johnson phrase it more succinctly: "The traditional view of rationality, together with Kant's idea of autonomy, gave rise to the view of human beings as autonomous rational actors, with complete freedom of the will and a transcendent rationality that allows them to think anything at all and to freely choose their purposes and beliefs. This view is false."[114] There are implicit limitations upon the ways in which we can think, both in terms of content as well as methodology – the product of evolution has yielded heuristical, not algorithmic, cognitive processes. To make this kind of thought meaningful in our everyday lives and major moral decisions, it becomes evident that we must first explore and understand how we think – we must abandon philosophies that fiat homuncular models of cognition. There is an inherent difficulty, however, in that the objections faced are not empirical – as the empirical evidence of cognitive science and neuroscience repeatedly demonstrate the concepts mentioned by Lakoff and Johnson – but ideological. It is difficult to abandon deeply held beliefs about radicalized autonomy; despite the implicit danger of such an ideology:

> Cognitive science has something of enormous importance to contribute to human freedom: the ability to learn what our unconscious conceptual systems are like and how our cognitive unconscious functions. If we do not realize that most of our thought is unconscious and that we think metaphorically, we will indeed be slaves to the cognitive unconscious. Paradoxically, the assumption that we have a

[112] Lakoff and Johnson, *Philosophy in the Flesh: The Embodied Mind and Its Challenge to Western Thought*, 513.
[113] Lakoff and Johnson, 514.
[114] Lakoff and Johnson, 536.

> radically autonomous rationality as traditionally conceived *actually limits* our rational autonomy. It condemns us to cognitive slavery – to an unaware and uncritical dependence on our unconscious metaphors. To maximize what conceptual freedom we can have, we must be able to see through and move beyond philosophies that deny the existence of an embodied cognitive unconscious that governs most of our mental lives."[115]

Simply put, if we want to be genuinely autonomous, to genuinely demonstrate a semblance of free will in decision-making, we must become much more aware of the role of the cognitive unconscious and other influences in routine, everyday cognition. By extension, a meaningful sense of autonomy in medical ethics must also incorporate this understanding – sticking to unempirical viewpoints, in addition to being bad science by forcing fact to fit ideology, is inherently dangerous, and threatens to constrict autonomy further by denying that the shackles are even there.

What further evidence exists for the requisite nature of cognitive models of autonomy? A variety of researchers and theorists over the past two decades have begun to stress the important of heuristics and biases, along with other cognitive constructs, in maintenance of 'normal' thought.[116] Well before Lakoff and Johnson, Miller and Moretti noted that the evidence increasingly supported cognitive models promoting bounded rationality – the idea that rationality is limited in scope and situationally dependent, even in 'normal' cognition.[117] Evans and Hollon support this conclusion, noting that "Existing beliefs appear to distort the processing of new information and to bias the retrieval of prior information from memory; information processing itself may rely more on intuitive heuristics that only haphazardly produce accurate

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[115] Lakoff and Johnson, *Philosophy in the Flesh: The Embodied Mind and Its Challenge to Western Thought,* 537-8..
[116] There is, of course, significant debate over the definition of what constitutes 'normal' thought, especially in light of the idiosyncrasies noted in cognition. A full accounting of the 'normalcy' debate is well-beyond the purview of this work; the errors and distortions noted in this work are common enough, I believe, to exempt them from the 'normal/abnormal' debate, although others have disagreed (principally raising the objection that they believe no normative standards exist).
[117] Miller and Moretti, "The Causal Attributions of Depressives: Self-Serving or Self-Disserving?" 266.

inferences."[118] Moretti and Shaw note that many of the assumptions we make about the world around us go unchallenged, regardless of their accuracy, and as such, we ought not to assume that our own cognitive feedback mechanisms will catch the automatic and habitual errors we are making.[119] Matthews notes that illness undermines personal autonomy – the underlying structures of cognition are altered by the experience of illness, producing decisions that may stem from the disease process, instead of the patient's authentic wishes.[120] Medical intervention then becomes necessary, to restore individuals capacity to speak for themselves. Parascandola, et al., note that clinicians should make themselves aware of common errors and cognitive biases, and to be aware of them in themselves as well, as they fundamentally affect treatment decisions.[121] Draper and Sorell lament that very little attention is paid to the kinds of decisions that patients *ought* to make – they note that it appears at times as if some models of autonomy create infallible patients, and that bad decisions are the responsibility of the physician, not the patient.[122] Clearly, then, there is significant need to explore influences on cognition not traditionally covered in theories of medical ethics.

Critiques of Autonomy

In addition to critics calling for exploration of the cognitive elements of autonomy, the principle has sustained attacks from a variety of perspectives, from feminist viewpoints,[123]

[118] Evans and Hollon, "Patterns of Personal and Causal Inference: Implications for the Cognitive Therapy of Depression," 348.
[119] Moretti and Shaw, "Automatic and Dysfunctional Cognitive Processes in Depression," 387-8.
[120] Eric Matthews, "Autonomy and the Psychiatric Patient," *Journal of Applied Philosophy* 17, no. 1 (2000): 67.
[121] Mark Parascandola, Jennifer Hawkins, and Marion Danis, "Patient Autonomy and the Challenge of Clinical Uncertainty," *Kennedy Institute of Ethics Journal* 12, no. 3 (2002): 257.
[122] Heather Draper and Tom Sorell, "Patients' Responsibilities in Medical Ethics," *Bioethics* 16, no. 4 (2002): 338.
[123] Jennifer Parks, "A Contextualized Approach to Patient Autonomy Within the Therapeutic Relationship," *Journal of Medical Humanities* 19, no. 4 (1998): 299-311; Anne Donchin, "Understanding Autonomy Relationally: Toward a Reconfiguration of Bioethical Principles," *Journal of Medicine and Philosophy* 26, no. 4 (2001): 365-86.

sociological viewpoints,[124] and other philosophical objections.[125] The recurrent conflict between autonomy and beneficence – briefly, a concern for the patients well-being, but a principle explored in more detail later – has raised the specter of paternalist thought. While ethicists have attempted to centralize patient autonomy, which has been rather successful in the past few decades, some are questioning whether this centralization of autonomy is a wise decision. Gawande, for instance, notes that giving autonomy *trump* power, instead of *pluralistic* importance:

> Where many ethicists go wrong is in promoting patient autonomy as a kind of ultimate value in medicine rather than recognizing it as one value among others. Schneider found that what patients want most from doctors isn't autonomy per se; it's competence and kindness. Now, kindness will often involve respecting patients' autonomy, assuring that they have control over vital decisions. But it may also mean taking on burdensome decisions when patients don't want to make them, or guiding patients in the right direction when they do. Even when patients do want to make their own decisions, there are times when the compassionate thing to do is to press hard: to steer them to accept an operation or treatment that they fear, or forgo one that they'd pinned their hopes on. Many ethicists find this line of reasoning disturbing, and medicine will continue to struggle with how patients and doctors ought to make decisions. But, as the field grows ever more complex and technological, the real task isn't to banish paternalism; the real task is to preserve kindness."[126]

Clearly the conflict regarding autonomy requires an examination of popular autonomy models – the concept appears to fall victim to its own assumptions about the individual, both in respect to his cognitive ability, as well as his psychosocial environment. In order to clarify the differences

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[124] Bruce Jennings, "Autonomy and Difference: The Travails of Liberalism in Bioethics," in *Bioethics and Society: Constructing the Ethical Enterprise*, ed. Raymond DeVries and Janardan Subedi (Prentice Hall, 1998), 258-69; Donald W. Light and Glenn McGee, "On the Social Embeddedness of Bioethics," in *Bioethics and Society: Constructing the Ethical Enterprise*, ed. Raymond DeVries and Janardan Subedi (Prentice Hall, 1998), 1-15; Paul Root Wolpe, "The Triumph of Autonomy in American Bioethics: A Sociological View," in *Bioethics and Society: Constructing the Ethical Enterprise*, ed. Raymond DeVries and Janardan Subedi (Prentice Hall, 1998), 38-59.
[125] Robert E. Lane, "Moral Blame and Causal Explanation," *Journal of Applied Philosophy* 17, no. 1 (2000): 45-58; Beate Roessler, "Problems with Autonomy," *Hypatia* 17, no. 4 (2002): 143-62; Richard W. Homan, "Autonomy Reconfigured: Incorporating the Role of the Unconscious," *Perspectives in Biology and Medicine* 46, no. 1 (Winter 2003): 96-108; Alfred I. Tauber, "Sick Autonomy," *Perspectives in Biology and Medicine* 46, no. 4 (2003): 484-95.
[126] Atul Gawande, *Complications: A Surgeon's Notes on an Imperfect Science* (New York: Picador, 2002), 223-4.

between the homuncular and cognitive autonomy models, important theories of each will be explored. We will examine theories in which cognitive elements are not given priority first.

Homuncular Autonomy Models

There are three principle models that will be examined in this section – those of Veatch, Faden and Beauchamp, and Beauchamp and Childress. A running theme in these models is an overt claim of rationality on the part of the patient in question. Some of these models make direct appeals to Kantian rational agents, a paradigmatic representation of the classical sense of rationality. Others make covert appeals to the model, linking cognitive bias and distortion to specific pathologies or outside influences. The recurring problem, then, is that the models propose personal autonomy without recognizing that the cognition upon which the autonomy is predicated is much less volitional than they make it out to be. They fiat a kind of homuncular autonomy – an idealized rational agent whose mind is akin to a little man selectively choosing what will influence his decision-making; a little man whose only apparent sources of weakness are disease, addiction, infancy, or dementia. As is evident from chapters one and two, this homuncular autonomy does not exist, nor has it ever existed. Our cognition involves the formation of myriad associations and interpretations well before our thought enters our conscious awareness – there is no "little man" to be found.

Veatch

One of the first influential theories of medical ethics was proposed by Robert Veatch in 1981.[127] Veatch establishes a relationship between deontological and consequentialist principles, which includes promise keeping, beneficence and autonomy. The fundamental question in his model, however, is whether personal autonomy – derived from liberty – ought to be an absolute,

[127] Robert M. Veatch, *A Theory of Medical Ethics* (New York: Basic Books, Inc., 1981).

or whether it ought to be potentially restricted by other concerns..[128] Veatch further divides autonomy into 'liberty rights' and 'entitlement rights'; the former prevent others from infringing upon the individuals ability to act, while the latter require others to act in some manner to allow us to act in turn (i.e., others must perform some action in order for us to be able to exercise our rights). Veatch categorizes the right to refuse medical treatment, the right to control one's body, and the right to consent to treatment under liberty rights.[129] The principle of autonomy carries with it innate moral worth, and as such, is classified as a deontological norm – it's good and worth is not derived or dependent upon any consequent good it may produce.[130] Veatch then proposes a rather counterintuitive claim – although professionals engaged in covenantal relationships (e.g., doctor and patient) have a prima facie duty to provide necessary information to their patients in the process of informed consent, they have no right to force information upon them. In fact, Veatch claims that the "same principle of autonomy that generates a right to be informed gives humans the freedom to act in a less than responsible manner. Imposing information on a person would violate the individual's autonomy just as withholding information would."[131] This claim will be problematic in light of the analysis presented thus far – Veatch is making an autonomy claim trump informed decision-making, and essentially allowing the irrational to trump the rational. This will be explored more fully following the presentation of his argument.[132]

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[128] Veatch, *A Theory of Medical Ethics*, 194.
[129] Veatch, 194-5.
[130] Veatch, 197.
[131] Veatch, 206.
[132] As an aside, I have significant objections to making autonomy an absolute, especially after spending several years wrestling with the ethical issues involved in psychiatric care. I have worked with patients who have been involuntarily committed who, following hospital stays of both short and lengthy durations, have been appreciative of the care they have received, and, in fact, say that it was better for them to have been committed. These patients carried a variety of diagnoses, ranging from severe depressions to schizophrenias. Had their autonomy been absolute, there would have been no philosophical justification for their commitment; in fact, caring for them would

Implicit in the covenantal model is the ability to remove oneself from the professional relationship. Veatch argues that entering into the care of a physician is not entering into a covenantal relationship for the rest of their lives for either physician or patient. Instead, both possess the ability to sever the relationship once it becomes clear that the course of treatment proposed or desired violates either the physician's or the patient's value systems or conscience.[133] For example, a patient would be free to sever the covenant were the physician to insist upon a course of treatment that violated the patient's religious views (e.g., transfusing blood products in the case of a Jehovah's Witness), or if the patient were to insist on a course of treatment that the physician believes to be completly unwarranted or against the standards of medical practice (e.g., a patient insisting on hemodialysis for a case of gout).[134]

The principle of autonomy carries trump value at all stages of life. Veatch explicitly argues that the right to refuse treatment applies equally throughout the stages of one's health – one cannot, for instance, arbitrarily treat against a patient's wishes simply because he or she is terminally ill:

> In this regard the fact that the patient is terminally ill would appear to count for little. Of course, we, as a society, might adopt the position that the principle of autonomy applies less rigorously once one is terminally ill. Unless we adopt such a stance, though, the right of self-determination applies equally in these circumstances. If there is any obligation to prolong life at all (independent of considerations of benefit and harm or a duty to avoid killing), it cannot authorize professionals to violate the autonomy of the terminally ill. The society cannot let a minority of physicians who believe that they have a duty to prolong life override the principle of autonomy in such situations.[135]

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have been, under this model, unethical – a statement Veatch himself verified when I discussed this with him at a conference at Georgetown University.

[133] Veatch, *A Theory of Medical Ethics*, 207-8.

[134] This obviously only concerns non-emergent situations or situations in which the patient is unable to express her wishes – state and federal law recognize exceptions to informed consent in a variety of situations (e.g., emergent situations, incapacitation, public health crises, etc.). See, for instance, Barry R. Furrow , et al., *Health Law*, Second Edition. (St. Paul: West Group, 2000), 334.

[135] Veatch, *A Theory of Medical Ethics*, 208.

This clearly represents a significant point of contention between Veatch and my argument – it has been the purpose of this dissertation to demonstrate in no uncertain terms that common medical conditions, including terminal illnesses, can profoundly affect how an individual processes information and makes decisions. It would be simply negligent to fiat autonomy as a trump in the case when an individual's cognition has been fundamentally compromised. This clearly does not happen in every patient, however, which is a topic to be explored in the final chapter of the present work. Veatch does recognize that moral decisions do not exist in a social vacuum – patients have other relationships that constitute moral communities, which must be factored into decision-making.[136] As such, he does recognize that autonomy is a larger concept, but he chooses to allow it only to grow outward (moral communities), rather than inward (reductive cognition and automaticity).

Veatch considers whether consequentialist principles might be better explanations for our moral sense. He notes that a variety of social perspectives – religious, Western secular, socialist, etc. – have all found the utilitarian perspective insufficient as an absolute (highest) norm in explaining our moral judgments.[137] All have modified it in some manner. The same critique holds true for the principle of promise-keeping; in fact, Veatch concludes that "a single, overarching principle as a solution to the dilemma of conflicting principles seems most implausible and probably would not be much help even if it could be found."[138] Instead of focusing on a single principle, Veatch explores the possibility that a pluralistic system might be more tenable – it might be possible for a *class* of principles to take precedence in cases of

[136] This is a central tenet of care ethics (also referred to as feminist ethics), in which the moral valuation of a decision changes in light of the relationships it affects. The closest analogy I can offer is that of a spider-web – any decision of moral significance does not simply affect one strand. Rather, the effects can be seen in light of how the overall structure of the web changes. Cutting one strand affects the shape and stability of the entire web, fundamentally altering other relationships.
[137] Veatch, *A Theory of Medical Ethics* 297-8.
[138] Veatch, 298.

conflict. There is precedence for this approach, as other systems of ethics have employed similar considerations (e.g., virtue ethics' pluralistic approach). This leads him to consider whether non-consequentialist principles as a class should be given priority over consequentialist principles.

Veatch ultimately concludes that the entire class of deontological norms must be satisfied before we explore consequentialist norms. This is to say, we can only begin to explore the consequences of our actions *after* we have ensured that our *prima facie* duties have been met:

> Perhaps, though, traditional professional physician ethics have the priority just reversed – perhaps the nonconsequentialist principles should be given first priority. Certainly this would change the character of medical ethical decisions made by lay people and professionals, but the decisions could be made. The nonconsequentialist principles could, in the normal case, be satisfied and still give leeway to pursuit of decisions under the principle of beneficence, or benefiting the patient. In fact, if the nonconsequentialist principles are to have any power in a medical ethical system, it may be that they together have to be given a lexical priority over the principle of beneficence. Otherwise consideration of consequences can always swamp the other moral considerations. As we saw in considering the ethics of human experimentation – especially with the Nazi experiments – if enough benefit is put into the calculus other moral considerations will always be overpowered. That is why it makes sense to insist that the other principles (autonomy, with its requirement of consent, keeping of contracts, avoiding killing, telling the truth, and promoting justice) must be satisfied as prior necessary conditions before consequences of the research can be put on the agenda. No amount of good consequences can overpower the inherent moral requirements of the nonconsequentialist principles.[139]

As such, we can never trump autonomy concerns by noting that good consequences will result. On the face of it, this makes sense. A persistent concern in utilitarian ethics (and other forms of consequentialism) is that of the "utility monster", in which actions against which many have a visceral reaction (e.g., genocide and slavery) become the moral course of action because of the good consequences they may produce – in short, it is a fundamental objection to the idea that the end justifies the means. Veatch categorizes a variety of principles as non-consequentialist,

[139] Veatch, *A Theory of Medical Ethics*, 299-300.

including promise keeping, autonomy, honesty, avoiding killing, and justice.[140] These all carry similar moral weight, and as such, become a potential source of conflict – a persistent critique of deontological systems of ethics is that it becomes very difficult to reconcile conflicting absolute principles. Veatch suggests that when exploring different courses of action, one ought to consider what course of action will produce the fewest violated non-consequentialist principles. If two options produce an equal minimum of violations, one could then proceed onto consequentialist considerations.[141] Veatch notes that this conflict resolution process may not be satisfying, but may be the only tenable approach, unless one were to attempt again to find the heretofore elusive, single, overarching principle.[142]

As indicated earlier, there are several potential areas of contention with Veatch's account of autonomy, but I will focus on two at the moment: Veatch's apparent cognitive model, and fundamental problems with other deontological principles (i.e., promise keeping). Both involve significant theoretical and empirical concerns.

The first immediate concern is that Veatch builds his conception of autonomy and rationality upon an unrealistic cognitive model. He offers very few potential exceptions to this model – he only makes occasional concessions that illness may compromise a patient's competence, and focuses more on exceptions resulting from a patient's lack of information.[143] As has been demonstrated in chapter one, the structure of cognition and consciousness is reducible to several levels of organization, and as such, is susceptible to influence at a variety of levels. We can alter thought by changing the environment the individual finds herself in. We

[140] Veatch, *A Theory of Medical Ethics*, 303.
[141] It seems almost ironic that Veatch resorts to a negative utilitarian calculus (minimizing the violation instead of maximizing the good) in resolving conflicting deontological norms.
[142] Veatch, 304.
[143] I will give Veatch the benefit of the doubt regarding backstage cognition and automaticity, in that this research did not really come into its prime until several years after Veatch published his work, as well as regarding neuroimaging, which only reached its prime in 1985 with the advent of magnetic resonance imaging.

can alter thought by changing the physical structure of her brain. We can alter thought by changing the sensitive biochemistry of her neurochemical pathways. Each of these is a significant cause for concern, as each of them is affected by hospitalization, especially when the hospitalization is due to a serious or terminal illness. Even the therapies we use can alter thought processes – by their very nature, medications alter biochemistry, which can affect cognition, a process some have referred to a 'pharmacological Calvinism' after the determinist theologian.[144] We have further seen that the cognitive phenomena of which we are aware are only the tip of the iceberg – it is quite possible that our 'consciousness' is only an epiphenomenon of our deeper and underlying mechanisms. In chapter two, we have seen that psychological processes are also dependent upon deeper, backstage elements and automatic processing. The way humans have evolved is not that of a Cartesian information processor – we do not algorithmically take in and weigh every bit of information that is presented to us. Instead, we adopt a variety of cognitive heuristics which allow us to quickly judge and assess situations, and thereby act in an environmentally adaptive manner. We have already seen that this produces both good and bad decisions; in fact, the nature of our cognition allows us to make disastrous decisions which we can, ostensibly, learn from. Further, we have seen that emotion plays a significant role in cognition, not only providing valences for memory, but also triggering these emotional memories in new situations. All of these considerations demonstrate that 'rational' cognition is susceptible to significantly greater errors than simply not having all the information – there is no guarantee that the patient will understand the information presented, and, without challenging the patient's decision-making process, there is no manner of assessing *how* persons come to the conclusion they reach. The cognitive model proposed by Veatch – and by extension, to other philosophical models positing autonomy as an absolute – is unrealistic in its assessment of human cognition.

[144] G. Klerman, "Psychotropic Hedonism Vs. Pharmacological Calvinism," *Hastings Center Report* 2 (1972): 1-3.

Even if one wished to preserve the ability of patients to be mistaken in the choices they make, I would suggest that this presents a case of the letter of the law destroying its spirit. Many philosophical, psychological, and theological models have suggested that mistakes allow for personal growth, by being able to appreciate and learn from the consequences of our actions (e.g., empiricist epistemology, Irenaean theodicy, associationist learning models, etc.). How is a patient to learn from a mistaken choice to forgo medical treatment, if this is potentially the last choice he or she will make in life? The choice to forgo medical treatment ought to be a, if not *the*, choice in which we would seek to eliminate as much error as humanly possible, since, ostensibly, this is a choice we might not be able to reconsider.

A second fundamental concern is the principles included in the deontological category, specifically promise-keeping. My concern about this principle revolves around times when it may be inappropriate to do so. In light of Veatch's argument, I find it quite likely that he and I will disagree regarding the exceptions to this *prima facie* duty. As an example, I offer advanced directives. In a covenantal relationship, one can apply Veatch's principle of promise-keeping to respecting treatment preferences expressed in an advanced directive.[145] Many ethicists have espoused the merit of documenting one's health care preferences, as it is widely believed to be an accurate presentation of one's values, and will help in making personal-value-congruent treatment decisions were one to become incapacitated. Once the legal standards have been met verifying and validating the advanced directive and the treatment team has the documentation on the patient's chart, one would hope that they would then simply respect the preferences contained within. Here, however, the issue gets murky. First, it is not so clear that the legal standards have been met, as these vary from state to state. Does one have an obligation to 'keep

[145] One could actually apply promise-keeping or autonomy to this situation. It will be evident, however, that both become problematic when analyzed in light of contemporary psychological research.

the promise' of a potentially legally non-binding document? Even if the legal standards have indeed been met, there are deeper and more problematic issues. Fagerlin , et al., have challenged both of the above assumptions of the medicophilosophical community – there is no guarantee that an advanced directive will accurately present one's values, and there is no guarantee that the document will help make value-congruent treatment decisions.[146] Through metanalysis of the medical and psychological literature, they note that it became evident that individual preferences change over time, sometimes quite drastically, and that sometimes uninvolved third parties can make more value-congruent choices than the proxies named in the advance directive. These both raise fundamental challenges – if preferences change over time, how certain are we that the document in front of us is an expression of contemporaneous wishes? Further, in light of the errors introduced by affective forecasting (how we believe we will feel about future events), how can we be sure that the patient was able to really understand in the past their present situation? If promise-keeping is a deontological norm, and therefore precedent to beneficence, do we have an obligation to uphold promises that might no longer represent what the patient wants?

Neither of these fundamental concerns are easy to resolve, but a step in the right direction is adopting a model of decisional ethics which appreciates the complexity of human thought, a topic we will return to later.

Faden and Beauchamp

A second influential model is that proposed by Ruth Faden and Tom Beauchamp.[147] Like Veatch, they stress the primacy of autonomy in medical ethics, and define it in terms of individual rights. They argue that the right to make autonomous choices and perform

[146] Angela Fagerlin , et al., "The Use of Advance Directives in End-of-Life Decision Making: Problems and Possibilities," *American Behavioral Scientist* 46 (2002): 268-83.
[147] Ruth R. Faden and Tom L. Beauchamp, *A History and Theory of Informed Consent* (New York: Oxford University Press, 1986).

autonomous actions correlates to the duty to refrain from interfering in the choices and actions of others.[148] Others, therefore, have a corresponding duty not to infringe upon our rights. They note that 'autonomy' serves as an umbrella term, by which we mean disparate concepts like "privacy, voluntariness, self-mastery, choosing freely, the freedom to choose, choosing one's own moral position, and accepting responsibility for one's choices."[149] In moral philosophy specifically, it carries the connotation of self-governance and self-direction, along with a freedom from undue influence from other external or internal sources (i.e., control by others or from personal limitations). They diverge from the literature, however, by focusing on the action performed, instead of the actor – their concern is that it is possible for otherwise autonomous individuals to make non-autonomous choices, due to a variety of influences.[150]

There are a variety of means by which autonomous individuals can render non-autonomous decisions; Faden and Beauchamp immediately note manipulation on the part of clinical staff as especially pernicious (e.g., withholding relevant information, not recognizing refusal of treatment, etc.). Respect for autonomy, they argue, is recognizing that individuals are entitled to their own views, values, and beliefs.[151] Failure to respect that autonomy is to raise the specter of paternalism – a phrase referring to the philosophical outlook that the clinician must look out for the welfare of his children, and therefore has the ability to overrule a recalcitrant patient as one would a misbehaving child. This is a significant concern, as medical ethics has strived to equalize the physician-patient relationship as much as possible.[152] They note that

[148] Faden and Beauchamp, *A History and Theory of Informed Consent*, 7.
[149] Faden and Beauchamp, 7.
[150] Faden and Beauchamp, 8.
[151] Faden and Beauchamp, 8.
[152] This is a necessarily hedged statement – while there will certainly be exceptions, most clinicians know more about the etiology and prognosis of the patient's condition, and as such, there will likely be a knowledge gap between clinician and patient, and therefore, it is likely that the clinician will have to correct some errors in the patient's understanding (so at least *some* paternalistic elements will remain in the relationship). However, the decision model currently preferred stresses that both the clinician and the patient be treated as peers, meaning that

placing authority in the patient's hands has been a popular recourse in the medical and research communities, for reasons as diverse as promoting beneficence, preventing morbidity and mortality, and preventing undue risks – extrinsic values, instead of implicit valuation of patient autonomy.[153] Paralleling Veatch, Faden and Beauchamp argue for a pluralistic approach to medical ethical principles (e.g., respect for autonomy, beneficence, and justice are all important considerations). Contrary to Veatch's model (among others), however, they argue that respect for autonomy is not lexically prior to other principles – there are conditions in which beneficence and justice can outweigh respect for autonomy:

> Numerous authors in biomedical and research ethics believe that if a person is acting autonomously and is the bearer of an autonomy right, then his or her choices morally ought *never* to be overridden by considerations of beneficence or proper care. This is not our assumption. Although the burden of moral proof will generally be on those who seek to intervene in another's choice, as the need to protect persons from harm becomes more compelling, thereby increasing the 'weight' of moral considerations of beneficence in the circumstances, it becomes more likely that these considerations will validly override demands to respect autonomy. Similarly, because some autonomy rights are less significant than others, the demands to protect those rights are less weighty in the face of conflicting demands.[154]

As such, we begin to see a balancing principle emerging in Faden and Beauchamp's model; unlike Veatch, it is essentially consequentialist in nature.

Faden and Beauchamp's concept of an autonomous agent does not employ a significant number of strict and rigid criteria. They contrast themselves with other models, which might require the autonomous person to be "consistent, independent, in command, resistant to control by authorities, and the source of his or her basic values and beliefs. The person's life as a whole

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the physician does not have automatic trump power over the patient's preferences. This produces a more collegial relationship and group decision-making as a result.

[153] Faden and Beauchamp, *A History and Theory of Informed Consent*, 14.

[154] Faden and Beauchamp, 19.

expresses self-directedness."[155] Such strict criteria create a nearly impossible standard to meet – in practical terms, very few individuals display these characteristics. Faden and Beauchamp suggest that a theory of personal autonomy does not require such extreme criteria; instead, they offer a model that involves three basic criteria: "We analyze autonomous actions as follows: X acts autonomously only if X acts 1. intentionally, 2. with understanding, and 3. without controlling influences."[156] They note that these criteria might still be wanting, but they believe that they suffice for an everyday understanding of autonomy. With the exception of intentionality, these criteria exist along a continuum – it is possible for the individual to possess greater or lesser degrees of understanding or control; intentionality is an all-or-nothing condition. As such, autonomy itself exists along a continuum, as measured by varying understanding and non-control.

The variables interact, but do not have collective trump power – this is to say, one cannot label an action autonomous if one of the three criteria is not met. No degree of intentionality and understanding can render an action autonomous if it is coerced, nor can intentionality and complete non-control render an action autonomous if it is not understood. In developing the idea of 'autonomy by degrees', Faden and Beauchamp establish thresholds for autonomous actions – points along the continuum above which all actions are considered autonomous, and below which they are considered non-autonomous.[157] However, as was mentioned earlier, intentionality does not exist along a continuum – as an all-or-nothing condition, it is necessary to establish criteria to discern whether or not an action is intentional. Drawing upon literature in philosophy and psychology, they argue that intentionality requires that the moral agent "integrate

[155] Faden and Beauchamp, *A History and Theory of Informed Consent*, 236.
[156] Faden and Beauchamp, 238.
[157] Faden and Beauchamp, 240.

his cognitions into a blueprint for action."[158] Essentially, for an agent to act intentionally, he must have a definite plan and act to follow through on this plan. This removes accidental actions, unformulated plans, and habituated behaviors from consideration when discussing intentionality.[159] Faden and Beauchamp do recognize that their suggestion that intention is not a matter of degree may seem questionable to some, and they note that one might consider it a matter of degree based on two possible grounds: 'mindfulness of willing' and 'correspondence with an action plan.'[160] The first of these notes that the degree of intentionality changes dependent upon how emphatically one wills the action. Less intentional actions are more automatic, with little cognitive awareness.[161] Ultimately, however, Faden and Beauchamp reject a continuum of intentionality, in favor of the all-or-none standard.

Their second critieria of autonomous action – understanding – presents unique challenges, as they note that 'understanding' as a construct in philosophical and psychological literature has garnered very little discussion. They argue that psychology has endeavored to explain the mechanism by which people "understand or comprehend human communications, with…emphasis on cognitive and neurophysiological human processes."[162] In essence, they suggest that the literature has sought an exploration of the mechanism of understanding, and has failed to examine what the term actually means. In order to clarify the issue, they define 'understanding' in propositional terms – the agent must understand that the action fits into a specific descriptive category and carries specific consequences. In essence, they want to be sure

[158] Faden and Beauchamp, *A History and Theory of Informed Consent*, 242.
[159] It is at this point that my first critique of Faden and Beauchamp's model appears. As has been argued in chapters one and two, backstage cognitive processes and automaticity are a significant part of cognition – in essence, our minds become habituated to respond in certain ways. If habitual action undermines their intentionality criteria, then the majority of everyday actions are not autonomous. I will return to this critique later.
[160] Faden and Beauchamp, 247.
[161] Again, strictly speaking, one could raise the objection that since cognitive science has demonstrated that the bulk of cognition is outside cognitive awareness, very few actions are actually 'intentional' in this sense.
[162] Faden and Beauchamp, 249.

that the agent has justified beliefs about what it is that he is doing.[163] If the moral agent is able to describe the intended action and its consequences, then that agent demonstrates understanding in their sense of the term. They refine their definition, however, in that there is a difference between *ideal* versus partial understanding:

> A person has a *full or complete* understanding of an action if there is a fully *adequate* apprehension of all the *relevant* propositions or statements (those that contribute in any way to obtaining an appreciation of the situation) that correctly describe (1) the nature of the action, and (2) the foreseeable consequences and possible outcomes that might follow as a result of performing and not performing the action. To the extent that this ideal is less than satisfied, an action is based on less than *full* understanding, and thus is less than a fully autonomous action.[164]

This explains the necessity of placing understanding along a continuum – it is intuitively apparent that one can have greater or lesser understanding of one's actions. Further, Faden and Beauchamp argue that having false beliefs about the proposed action fundamentally undermines understanding, opening the door to non-autonomous actions (even in situations in which the individual is 'responsible' for these false beliefs).[165] There is, however, difficulty in determining whether and which beliefs diminish understanding.

The third criterion centers on coercion and controlling influences. Faden and Beauchamp argue vigorously for the separation of the ideas of willful action, voluntary action, and controlled/non-controlled action.[166] While some sources conflate these concepts, Faden and Beauchamp note that it is possible for actions to fit into one category without necessarily fitting into another. For instance, while one might be controlled, it does not therefore logically follow that the agent does not intend the outcome or action.[167] In a like manner, actions can be

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[163] Faden and Beauchamp, *A History and Theory of Informed Consent,* 250-1.
[164] Faden and Beauchamp, 252.
[165] Faden and Beauchamp, 253.
[166] Faden and Beauchamp, 256.
[167] I normally endeavor to avoid thought experiments, but it seems appropriate here to include a quick example differentiating between volitional and avolitional control. In the course of his experiments, Penfield found that he could cause muscle spasms in his patients by stimulating particular regions of the motor cortex (located on the

completely free from coercion, and yet be non-intentional (e.g., accidents, muscle spasms, etc.). Further, Faden and Beauchamp distinguish *influencing* an action versus *controlling* an action. It is entirely possible to be influenced by others without being controlled by them. An attempt at influence is an attempt to persuade, not to control. Persuasion and influence do not exist on a continuum – Faden and Beauchamp understand them as standing at opposite ends of a spectrum: "Coercion and persuasion are not continuum concepts: Coercion is always controlling, but not by degrees; persuasion is never controlling and involves no degree of noncontrol. Manipulation, by contrast, *is* controlling or noncontrolling and admits of degrees."[168] As a result, an agent is nonautonomous when he is being manipulated (e.g., when relevant information is withheld).

Having established their three criteria for autonomous action, Faden and Beauchamp turn their discussion to other concepts that have been proposed in the autonomy discussion, taking time to address the concept of authenticity. In a nutshell, authenticity argues that actions must represent the reflected-upon values and life plans of the patient to truly qualify as autonomous. It is raised here because Faden and Beauchamp recognize that individuals can act in a manner that meets all three proposed criteria, and yet fail to act autonomously (e.g., acting due to addictions or other psychiatric conditions).[169] Ultimately they reject authenticity as a necessary condition for autonomy, principally because while it does raise germane concerns about ownership of actions, it places an undue and unnecessary burden upon the moral agent. As a matter of practicality, the great bulk of the decisions that we make that would normally fit the

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posterior region of the frontal lobe). If one were to manipulate the motor neurons of a patient's arm, thereby causing him to strike a loved one (whom he does not want to strike), it is clear that the patient is being controlled avolitionally – we are forcing him to perform an action he does not want to do. If, on the other hand, we were to replace his loved one with a bitter rival, we might find that the patient gleefully participates in our manipulation, instead of wanting to avoid it. Clearly we have volitional control – we are controlling his arm, but we are not going against his wishes in doing so.

[168] Faden and Beauchamp, *A History and Theory of Informed Consent*, 258.

[169] Faden and Beauchamp, 263.

definition of autonomy would be rendered non-autonomous.[170] For instance, when we conduct our day-to-day affairs, it is the exception, rather than the rule, that we weigh each and every decision in light of who we were, who we are, and who we intend to be.[171] Further, they argue that as a result, authenticity claims would fundamentally narrow the *range of decisions to be respected as autonomous*. They stress that this is not to say that only autonomous decisions are to be treated with respect, but that authenticity would narrow the range of decisions enjoying a principled defense *due* to their being autonomous. As such, they stress that one cannot arbitrarily refuse to honor an autonomous decision simply because there is reason to believe that it is not authentic.[172] At this point, it is necessary to note a caveat – thus far, it has been assumed that authenticity refers to a consistent reflective process on the part of the agent. Faden and Beauchamp note that it is also possible to conceptualize authenticity in terms of 'nonrepudiation' of one's values – i.e., the agent is not required to reflect actively upon his or her values in decision-making; authenticity can be satisfied so long as the decision does not repudiate previously held beliefs or principles (i.e., the action is 'in-character'). While this seems to be a promising reformulation, ultimately it faces empirical challenge and is of questionable merit in defining autonomous actions, and as such, it is rejected.[173]

As with Veatch's model, serious concerns can be raised about the 'rational' assumptions proposed in Faden and Beauchamp's model. While they note that psychiatric illness can be a

[170] Faden and Beauchamp, *A History and Theory of Informed Consent*, 264.
[171] More than one philosopher has made similar comments about a variety of issues connected with agency, autonomy, and authenticity. For instance, I generally find I am less concerned with whether a particular loaf of bread meshes with my personal web of values and beliefs about free will versus determined action than I am with whether or not it is on sale. A recurrent critique of philosophical arguments about agency is that they are overly focused on abstract criteria that do not gel well with day-to-day life, a position with which I am very sympathetic. However, I do and will argue that for decisions of great importance, such as the choice to forgo medical treatment, high standards – including explorations of authenticity – are warranted.
[172] Faden and Beauchamp, 266.
[173] Faden and Beauchamp, 268.

influential factor, they do not address backstage elements of cognition.[174] Further, they *do* note that psychology can offer insight into cognitive traits and abilities (which they label as inadequate when analyzed in terms of capacity to consent to treatment)[175] As has been demonstrated, however, psychology has provided significant insights into how we actually process information; when augmented with the analyses offered by cognitive science, we have a significantly greater understanding of what it means to be a *cognitive* agent, which allows us to make more accurate claims about what it means to be an *autonomous* agent. If my decisions are the result of persistent biases in information interpretation that do not fit neatly into one of the exceptions covered by Faden and Beauchamp's model (e.g., affective forecasting or the availability heuristic), it would seem to make less sense to automatically defer to my 'autonomous' choice. The crux of this objection is that research has demonstrated that there are a variety of potential internal influences which can render decisions fundamentally flawed but which would still fit their proposed definition of 'autonomy'; I would suggest that we do our patients a disservice by simply acquiescing to flawed, correctable, and terminal decisions.

Second, they fundamentally reject shared decision-making models, favoring a model in which a patient consents to treatment as a sole moral agent.[176] This is troublesome in that it seems to place an undue amount of confidence on the part of the patient. This is to say, instead of leveling the decision-making playing field, it seems that they are simply reversing the hierarchy, placing the patient above the physician. Concerns about power-relationships aside, there is significant concern about whether a patient genuinely will appreciate the medical facts of the situation, and whether the patient will be able to make objectively accurate judgments based

[174] I am willing to allow some leeway in criticizing Faden and Beauchamp, as Tversky and Kahneman's studies had only been recently published. However, the criticisms of the model still hold true – if one were to propose a similar theory today, it would be susceptible to the same criticisms I will propose here.
[175] Faden and Beauchamp, *A History and Theory of Informed Consent,* 291-2.
[176] Faden and Beauchamp, 279.

upon a potentially limited understanding. The benefits of a shared decision-making model is that it allows for the greatest possible interaction between medical understanding of the physician and the psychosocial realities of the patient. Like the physician's privileged access to medical training and experience, the patient also possesses specialized knowledge – her life experience and the meanings derived from them. Shared decision-making allows for mutual ownership and responsibility in outcomes.

Third, there is significant concern about their rejection of authenticity as a criterion for autonomous action. While it is true that a great many decisions do not meet this authenticity concern, there is no reason to abandon it. It may be more reasonable to use it as a threshold criterion – as the severity of the consequences of the decision rises, it approaches a threshold point at which authenticity may become a necessary criterion. Day-to-day decisions would not be affected – whether we buy white or wheat bread is not a matter of great moral importance. However, when our decision-making shifts to life-altering or life-ending consequences, it does not make sense to abandon the underlying goals and plans by which we have lived our lives.

Beauchamp and Childress

By far the most widely known theory of medical ethics is that proposed by Tom Beauchamp and James Childress.[177] Melding consequentialist with deontological arguments, the two proposed a theory exploring and balancing four key principles: justice, beneficence, nonmaleficence, and autonomy. A recurring issue in medical ethics is the occasional conflict between two or more of these principles. While interpretations of the principles vary – e.g., whether beneficence ought to be understood as the medical health and welfare of one's patient,

[177] Tom L. Beauchamp and James F. Childress, *Principles of Biomedical Ethics*, Fifth (New York: Oxford University Press, 2001).

or whether the principle includes an appreciation of their agency[178] – many clinical case consultations can be framed and reconciled in light of common understandings.

Autonomy in Beauchamp and Childress's model is not given priority over beneficence, non-maleficence, or justice.[179] Rather, each is of significant, but not paramount, concern – which principle is to be upheld in a given case is discerned through precedent analysis, clarification of the relevant issues and conflicts, and balancing conflicting concerns. Beauchamp and Childress note that autonomy is an umbrella term, and has taken on a variety of meanings. Central to their argument, however, is the minimal conception of personal autonomy as "self-rule that is free from both controlling interference by others and from limitations, such as inadequate understanding, that prevent meaningful choice."[180] This self-rule allows the individual to choose and follow a life-plan. However, even agents autonomous per this proposed definition sometimes fail to act autonomously, for a variety of reasons, including "illness or depression, or because of ignorance, coercion, or other conditions that restrict their options."[181] Mirroring Faden and Beauchamp, they understand autonomous actions to be defined by intentionality, understanding, and an absence of control, the first of which is absolute, while the latter two exist on a continuum; similarly, they establish thresholds for understanding and freedom from constraint in autonomous actions, instead of absolute standards, in an effort to explain autonomy in everyday life.

Respect for autonomy is a key governing principle; it is not sufficient to posit that a patient is autonomous – rather, we have an obligation to respect the rights of others to hold their

[178] For instance, I would and have argued that beneficence ought not to be interpreted as encompassing authenticity principles and respect for patient decision-making, which I believe are central to the concept of autonomy, instead. I argue that if such a distinction is not made, then there is no real need for a separate principle of beneficence.
[179] Beauchamp and Childress, *Principles of Biomedical Ethics*, 57.
[180] Beauchamp and Childress, 58.
[181] Beauchamp and Childress, 58.

own views and act accordingly. Respect for autonomy, therefore, conveys a duty for "respectful action, not merely a respectful attitude."[182] Respectful actions include facilitating autonomous actions, assisting in building the capacity to act autonomously, and acting to prevent conditions that undermine autonomous actions. As such, respect for autonomy can be phrased in terms of both positive and negative obligations: we have a duty to refrain from constraining the autonomous actions of others, and we have a duty to facilitate autonomous actions for others (who then reciprocally have corresponding obligations to us). This sense of autonomy is not an absolute obligation; instead, it is a *prima facie* obligation – we recognize that there are implicit limitations:

> Our obligations to respect autonomy do not extend to persons who cannot act in a sufficiently autonomous manner (and cannot be rendered autonomous) because they are immature, incapacitated, ignorant, coerced, or exploited. Infants, irrationally suicidal individuals, and drug-dependent persons are examples. Those who vigorously defend rights of autonomy in biomedical ethics, as do the present authors, do not deny that many forms of intervention are justified if persons are substantially nonautonomous and cannot be rendered autonomous for specific decisions.[183]

Aside from these gross impairments, respect for autonomy is clouded by the knowledge that beliefs and values change over time. As discussed above in the critique of Veatch's model, there is a substantial problem raised when we face individuals who have changed their mind. Which autonomous choices ought one to respect when the current choices conflict with previously codified choices? Akin to Faden and Beauchamp, Beauchamp and Childress address questions of authenticity and the congruence of the patient's choices with her character. They draw similar conclusions – authenticity is not a necessary criterion of autonomy – but do caution that actions deviating from one's character "can raise caution flags that warn others to seek explanations and

[182] Beauchamp and Childress, *Principles of Biomedical Ethics*, 63.
[183] Beauchamp and Childress, 65.

probe more deeply into whether the actions are autonomous."[184] While it makes intuitive sense that actions out of character ought to raise warning flags, it is not so clear that authenticity ought to be abandoned as an autonomy principle.

The question of autonomy necessarily involves the question of competence to give consent (or to refuse it). Beauchamp and Childress stress that 'competence' ought not to be understood in *global* terms – instead, a more accurate understanding of the term recognizes that different actions have different criteria of competence (e.g., the competence criteria to set up a gel electrophoresis is quite different than the competence criteria to give a philosophy lecture, which in turn differs from the competence criteria to make a soufflé). As such, one ought not be judged incompetent in every aspect of her life, merely regarding specific tasks. Further, competence can wax and wane over time – as such, it is necessary to recognize that competence is neither a global nor a static concept. As such, if competence cannot be readily assessed immediately, it would make sense to assess it over a period of time.[185] In the case of the competence criteria for medical decisions, Beauchamp and Childress propose that "Patients or subjects are competent to make a decision if they have the capacity to understand the material information, to make a judgment about the information in light of their values, to intend a certain outcome, and to communicate freely their wishes to care givers or investigators."[186] As such, they note that the criteria for autonomy and for competence are quite similar, and that similar thresholds can be established.

There is not a single, overarching threshold for competence – instead, these will vary with the decision at hand. The threshold increases with the complexity or difficulty of the task, but not necessarily with the riskiness of the outcome:

[184] Beauchamp and Childress, *Principles of Biomedical Ethics*, 68.
[185] Beauchamp and Childress, 70.
[186] Beauchamp and Childress, 71.

> It is correct to say that the threshold level of competence to decide will rise as the complexity or difficulty of a task increases (deciding about spinal fusion, say, as contrasted with deciding whether to take a minor tranquilizer). However, the level of competence to decide does not rise as the risk of an outcome increases. It is confusing to blend a decision's complexity or difficulty with the risk at stake. No basis exists for believing that risky decisions require more ability at decision-making than less risky decisions. To the contrary, a solid basis exists for believing that many non-risky decisions require more ability at decision-making than many risky decisions…We can avoid these problems by recognizing that the level of *evidence* for determining competence should vary according to risk, while competence itself varies only along a scale of difficulty in decision-making.[187]

This may be problematic, however. While Beauchamp and Childress are correct in tying competence standards to the difficulty of the task at hand, it is not clear to this author why one ought to divorce risk from competence standards as cognitive psychology has demonstrated that risk perception fundamentally influences health decisions.[188]

There are essential pieces of information that must be conveyed to the patient to facilitate autonomous action. Standard disclosures (e.g., in the process of informed consent) include such basic elements as the patient's diagnosis, prognosis, proposed intervention, alternatives, risks and benefits, and recommendations.[189] Patients must voice their understanding of these elements, and must be clear about the terms of their agreement as well – absent these requisites, there is no guarantee that an autonomous decision has been made.[190] At this point, however, there is concern about the patient's ability to process all of the information provided. Beauchamp and Childress note that some critics have suggested that patients cannot comprehend or appreciate

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[187] Beauchamp and Childress, *Principles of Biomedical Ethics*, 76.

[188] See, for instance, Neil D. Weinstein, "Exploring the Links Between Risk Perceptions and Preventive Health Behavior," in *Social Psychological Foundations of Health and Illness*, ed. J. Suls and K.A. Wallston (Malden: Blackwell Publishing, 2003), 22-53. This is not to say that it is entirely clear *how* risk perception influences behavior – psychological models cannot currently account for 100% of the variance in health behaviors, but risk perception has been shown to be germane in health behaviors. Weinstein notes, for instance, that a systematic optimistic bias in health outcomes was found in a variety of studies (participants consistently rated themselves as significantly less likely to experience a negative health outcome than the average person, which is statistically nonsensical, as the majority of any population cannot be at lesser risk than the average person).

[189] Beauchamp and Childress, 89.

[190] Some states have additional requirements – for instance, some states require clinicians do disclose their personal success rate with the intervention proposed, referred to colloquially as their 'batting average.' See Furrow , et al., *Health Law*.

the information provided, a claim which they suggest is an overgeneralization: "From the fact that actions are never *fully* informed, voluntary, or autonomous, it does not follow that they are never *adequately* informed, voluntary, or autonomous."[191] As such, patients do not necessarily need to become physicians themselves in order to be competent to decide the course of their treatment. Beauchamp and Childress do note, however, that how the information is framed can unduly influence the patient, producing decisional errors.[192] Clearly one must avoid framing effects in relaying information in order to allow the patient to reach an autonomous decision – intentionally framing the information to elicit a specific response is simple manipulation instead of ethical disclosure.[193]

The voluntary aspect of autonomy concerns undue influence by both internal and external factors. As Faden and Beauchamp noted, voluntary actions are distinct from autonomous actions – it does not suffice to use the same definition or criteria for both. They note that the voluntary aspect of an action can be undermined by disease, psychiatric disorders, and drug addiction.[194] The list of possible influences, however, is not exhausted by these three. Rather, they note that influence can occur as a result of elements as diverse as loved ones, threats and overt coercion, education, manipulations, and emotional appeals. However, simply because an influential factor is present, there is no reason to assume that the patient's decision has been altered – in fact, Beauchamp and Childress argue that clinicians have a moral responsibility to attempt to persuade the patient to pursue medically necessary treatments.[195] Such efforts to persuade may be

[191] Beauchamp and Childress, *Principles of Biomedical Ethics*, 89.
[192] Beauchamp and Childress, 90.
[193] It is quite ironic, however, in that they cite Tversky and Kahneman's study on framing effects, but do not mention other germane articles by the same author(s) dealing with systematic errors in processing, e.g., Donald A. Redelmeier, Paul Rozin, and Daniel Kahneman, "Understanding Patients' Decision: Cognitive and Emotional Perspectives," *Journal of the American Medical Association* 270, no. 1 (July 7 1993): 72-76, or the literature mentioned in detail in chapter two of this work.
[194] Beauchamp and Childress, 93.
[195] Beauchamp and Childress, 95.

necessary to ensure that the patient both has and understands the medical information necessary to inform her choice.

As noted above, the principles proposed by Beauchamp and Childress can conflict. The most frequent conflicts occur between the principles of beneficence and autonomy. The principle of beneficence has a long history in medicine – Beauchamp and Childress note that it extends back at least to Hippocrates' edict in his *Epidemics*: "As to disease, make a habit of two things – *to help, or at least to do no harm*."[196] Patient welfare is the proviso of this principle – beneficence establishes the duty of the physician to ensure that the patient receives the treatment he needs. By raising this concept to the level of a medical norm, the stage is set for conflict – the physician may want to pursue a course of treatment that the patient does not. Historically, clinicians simply overruled their patients – the concept of patient autonomy is a relatively recent phenomena in the practice of medicine. As mentioned previously, this attitude became known as paternalism. Beauchamp and Childress note that paternalism itself is normatively neutral – individuals acts of paternalism can be either justified or unjustified.[197]

Paternalism can be divided into two types of action. 'Weak paternalism' involves interventions only in cases when the patient in question is engaging in a patently nonautonomous manner. Severely demented patients, newborns, patients experiencing delirium, etc., are examples of individuals with clearly compromised or absent autonomy. These patients cannot process information, cannot make free choices, etc. – autonomy is substantially absent in these cases, and the 'choices' made may not be binding for clinicians.[198] 'Strong paternalism', in contrast, occurs when a clinician simply refuses to honor or overrides an autonomous patient's

[196] Beauchamp and Childress, *Principles of Biomedical Ethics*, 176.
[197] Beauchamp and Childress, 178.
[198] Again, as a necessary caveat, this does not refer to all decisions. There is no reason not to respect choices in areas that do not require significant cognitive abilities (e.g., in menu or clothing selection, or other less morally pressing matters).

request or wishes, or manipulates the information provided to her, preventing her from making an autonomous choice. This type of paternalism is more difficult to justify – there is no evidence that autonomy is substantially absent as it was in the weak paternalism cases. Beauchamp and Childress note that the justification of paternalism exists on a sliding scale. As the personal risks a patient takes on increase and the benefits of the patient's wishes decrease, the scale shifts away from upholding autonomy to upholding beneficence. They note that "preventing minor harms or providing minor benefits while deeply disrespecting autonomy lacks plausible justification; but actions that prevent major harms or provide major benefits while only trivially disrespecting autonomy have a highly plausible paternalistic rationale."[199] As such, there *are* times when strong paternalism is justified, but these are evaluated on a case-by-case basis, instead of establishing an overarching paternalistic policy.

As with the previous two theorists, significant objections can be raised. First, the similarities in the arguments of Faden and Beauchamp with those of Beauchamp and Childress warrant similar objections. Beauchamp and Childress suffer the same critiques concerning an overly 'rational' patient model – they make assumptions about what will influence the patient without incorporating known sources of error.[200] Likewise, concerns about divorcing authenticity from autonomy are applicable to this model. There is no need to restate the entirety of the arguments presented, however.

A second fundamental concern is their argument that divorces risky outcomes from evaluations of competence. As noted earlier, the literature on cognitive psychology has noted

[199] Beauchamp and Childress, *Principles of Biomedical Ethics*, 185.
[200] Beauchamp and Childress are open to criticism for not addressing the heuristics and biases noted by Tversky and Kahneman, as the cognitive psychology literature (as well as that of its critics) has been available for several decades. The omission causes me concern in light of their reference to framing effects, but their lack of mentioning durability biases, affective forecasting, availability, anchoring, or the other significant sources of processing errors mentioned in chapter two, which form the bulk of Tversky and Kahneman's work.

that risk perception does indeed influence health care decisions. Concerns about optimistic biases in health decisions aside, it is disconcerting that an area of research that has consistently demonstrated influence would be not be given more weight in the course of their argument.

Conclusion

The preceding analysis is not meant to fundamentally scuttle the theories discussed. They have individual strengths and weaknesses that ought to inform subsequent models. It makes eminent sense to establish prima facie duties, for instance, and to value a collaborative relationship between physician and patient. It makes eminent sense to recognize that ethics is pluralistic, and that it is unlikely that any single principle ought to carry universal and absolute weight. It makes sense to draw upon a variety of philosophical outlooks in offering justification for action, or in discerning the appropriate moral methodology for a given ethical conflict.

However, it does not make sense to predicate an ethical theory on a model of human thought that does not exist. Fiating cognitive abilities amounts to requiring us not to be human when exploring ethical dilemmas or making treatment decisions. It makes no sense to believe that we exercise control over avolitional backstage processes, or to ignore demonstrable sources of error in decision-making, especially when the choices to be made are potentially the most meaningful and most irrevocable of decisions. It makes no sense to suggest that identifiable sources of error ought not to be eliminated as much as possible, to ensure that the choice made is a genuine reflection of the patient's desires, and is not simply the disease process speaking for them. The models that follow attempt to elicit these sources of error, while reaching fundamentally different conclusions.

Cognitive Autonomy Models

In contrast to the homuncular models, the cognitive models endeavor to explore the backstage and automatic elements of patients making health decisions. Four principle models are examined, and the strengths and shortcomings of each are noted. A recurring theme in these critiques is that cognition is fundamentally influenced by a variety of factors not considered in the homuncular models. As such, by their very nature, they present models of autonomy that have much more empirical and ecological validity – they are autonomy models of actual human beings, rather than of idealized cognitive agents.

Redelmeier, et al.

The first cognitive model to be considered is that of Redelmeier, et al.,[201] Contrary to the homuncular models discussed earlier, Redelmeier, et al., note that the 'ideal' decision maker – characterized by the agent who gathers all available information, calculates the risks and benefits of every option, and then selects the optimal choice – simply does not exist.[202] Instead, actual decision-makers employ cognitive heuristics to simplify situations and find palatable solutions. Further, actual decision-makers are influenced by a variety of sources, including external and internal stimuli, as well as how information is presented to them. Framing effects – discussed earlier in relation to Beauchamp and Childress – are quite powerful:

> Peoples' interpretation of most events depends on both the nature of the experience and the manner in which the situation is presented or 'framed.' For example, a foul odor near a sewer may be disgusting, whereas the same aroma emanating from a cheese counter can be enticing. Psychological research has shown that people are often sensitive to the presentation of problems and that they fail to realize the extent to which their preferences can be altered by an inconsequential change in formulation.[203]

[201] Redelmeier, Rozin, and Kahneman, "Understanding Patients' Decision: Cognitive and Emotional Perspectives."
[202] Redelmeier, Rozin, and Kahneman, 72.
[203] Redelmeier, Rozin, and Kahneman, 73.

Minor shifts in decision context, option order, defaults, or semantics can radically alter perception and subsequent processing, and yet these are not necessarily changes of which we are aware.[204] Further, individuals can demonstrate a phenomenon called 'hindsight bias' – when individuals learn of the outcome of a given action, this knowledge affects their assessments of the likelihood of that outcome occurring.[205] This is to say that individuals tend to ignore contradictory evidence, focus only on corroborating evidence, and overestimate the probability of the outcome. This is a significant concern in medical liability cases, for instance – arguments that a clinician "should have seen this coming" demonstrate hindsight bias. In the context of medical treatment, this can affect individuals perception of their current situation (e.g., 'it was inevitable that I would get cancer'), and can feed into other sources of cognitive error (e.g., affective forecasting and the availability heuristic).

Redelmeier, et al., note that many research studies fail to take into account salient features of the patient experience when exploring outcomes and efficacy. There are emotional aspects of being a patient, for instance, which are reflected in one's sense of well-being and validation. Patients, as a result, often seek medical care for sympathy and reassurance.[206] This presents a difficulty for research, however, in that these emotional valences and experiences are difficult to quantify in the same way as one could quantify physical or mental disability. Difficulty in measurement, however, does not translate into irrelevancy.

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[204] This really is a remarkable phenomenon. Environmental cues, for instance, have been demonstrated to be a confounding variable in research, and as such, are controlled as much as possible. Presentation order has been shown to demonstrate that individuals have a tendency to choose the last option presented to them – even if the items presented are identical – and that they will offer fabricated justifications to explain why that particular option was different than the others. The presence of defaults has also been demonstrated to affect cognition – studies have demonstrated that many individuals have a tendency simply to accept default options when presented with a choice. Finally, word choice affects salience – it has been demonstrated that individuals view information differently when it is seen as self-relevant; this perception, however, can be affected by whether the individual properly understands the terminology (e.g., there will be a difference in responses between asking someone if they are diaphoretic versus asking them if they are sweating).

[205] Redelmeier, Rozin, and Kahneman, "Understanding Patients' Decision: Cognitive and Emotional Perspectives," 73.

[206] Redelmeier, Rozin, and Kahneman, 74.

This emotional content complicates medicolegal issues as well. Redelmeier, et al., note that the process of informed consent requires the clinician to disclose the risks, benefits, and outcomes of particular interventions. Ostensibly the patient then decides which option best suits his needs and values, but this concept does not take into account the plasticity of human emotion – his needs and values may not be the same once the intervention has been selected and performed. They note that "psychologists have shown that people are prone to err when making decisions about long-term consequences because they fail to anticipate how their preferences will change over time."[207] This is not limited to medical settings – studies have demonstrated that attempts to forecast how one will feel produce errors in such diverse conditions as being fired from one's job to winning the lottery. We have a tendency to believe erroneously that the joy or sorrow we are experiencing now will continue unabated for the foreseeable future. As a result, Redelmeier, et al., suggest that the informed consent process include an appreciation of changes over time, and that patients might benefit from including "statistics and interviews of people who underwent each therapeutic alternative months of years previously."[208] As a corollary to their suggestion, it would seem that in the case of forgoing treatment, comparable information might be included, if available.[209]

A special case is presented for patients who are experiencing a recurrence of their illness – some conditions are long-standing with periods of remission (cancer, for instance, or multiple sclerosis). Initially, one might be more inclined to accede to their wishes, as they have already experienced the positive and negative effects of the given intervention. However, even this first

[207] Redelmeier, Rozin, and Kahneman, "Understanding Patients' Decision: Cognitive and Emotional Perspectives," 74.
[208] Redelmeier, Rozin, and Kahneman, 74.
[209] Clearly this may present a problem, as individuals electing to forgo treatment might not necessarily be in any shape to provide said information. Other methods of providing this information might include patient testimonials (written or video), contact with surviving family members, etc. While there are difficulties in securing this information, it is not impossible in any sense of the term.

hand experience is not necessarily accurate. Redelmeier , et al., note that memories can also be inaccurate and subject to error.[210] As such, we ought not simply defer to patients' prior experience – they may have a distorted sense of the experience:

> When people make a medical decision between alternatives that they have already experienced (e.g., a second round of radiation therapy), they compare their mental representations of each of the alternatives and, presumably, choose the alternative that they remember as less unpleasant. But memories are inaccurate and subject to error. One major distortion is that duration may not be as well represented as peak intensity. Thus, a few days of intense acute pain may be remembered as more unpleasant than many weeks of moderate chronic pain, in the same way that a brief media soundbite may be more memorable than a longer detailed report."[211]

In light of all of these concerns, Redelmeier , et al., caution that the process of medical decision-making must involve clinicians providing guidance about medical information, but also about common cognitive errors.[212]

This model provides a more accurate picture of actual cognitive processing in decision-making, but it is hardly a complete ethical theory. Rather, the article serves as an effort to translate the heuristic and biases literature into clinical settings, and to make clinicians aware of the issues that they will have to face. More developed theories of autonomy are found in the arguments and models presented next.

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[210] This is not a new claim – Hume, for instance, noted this phenomenon in his epistemology: our (simple and complex sense) impressions cannot be mistaken, but our recollections of those complex sense impressions are fallible. It is quite easy for us to misremember events, locations, and experiences, amplifying certain characteristics and suppressing others. As such, personal recollection and experience are not necessarily infallible guides for action.

[211] Redelmeier, Rozin, and Kahneman, "Understanding Patients' Decision: Cognitive and Emotional Perspectives," 74-5.

[212] This is not to claim that clinicians are in a privileged position and do not employ the same kinds of errors; for instance, see David M. Eddy, "Probabilistic Reasoning in Clinical Medicine: Problems and Opportunities," in *Judgment Under Uncertainty: Heuristics and Biases*, ed. Daniel Kahneman, Paul Slovic and Amos Tversky (New York: Cambridge University Press, 1982), 249-67; Neal V. Dawson and Hal R. Arkes, "Systematic Errors in Medical Decision Making: Judgment Limitations," *Journal of General Internal Medicine* 2 (1987): 183-87.

Grisso and Appelbaum

Like Beauchamp and Childress, Thomas Grisso and Paul S. Appelbaum[213] stress that the concepts of autonomy and of competence to consent to (or refuse) treatment are related. They argue that competence to consent necessarily involves four criteria.[214] First, it is necessary that the moral agent be able to express a choice – this is not tied to any particular medium of communication (e.g., the patient does not need to be able to speak to do so), but rather, the patient must possess the ability to make his or her choices known. Second, the patient must be able to understand the information germane to the health care decision. If the patient cannot understand the information at hand, there is no way to act upon it or to voice a preference for one intervention over another. Third, the patient must appreciate the significance of the information and the expected outcomes. If there is no way for the patient to gauge risk or to weigh outcomes, there is no way for the patient to take ownership of the decision – there is a fundamental disconnect between the decision and the outcome. Fourth, the patient must be able to reason with the germane information in a manner that allows him or her to logically weigh treatment options. If a patient cannot reason and deliberate about the decision, there is no manner by which he or she can make a genuinely autonomous choice – it is akin to being asked to write a paper without having any writing implement – some organization may be possible, but clearly the ultimate goal will not be able to be realized. These four criteria are not to be understood as being 'all-or-none' principles – that is to say, each of these criteria exists on a continuum; patients manifest different abilities for each at different times. As such, like Beauchamp and Childress, Grisso and Appelbaum argue that competence is not to be understood globally, but is

[213] Thomas Grisso and Paul S. Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals* (New York: Oxford University Press, 1998).
[214] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 31.

task specific. Ethical judgments must be cognizant of each of these criteria, but "in practice, not all of them uniformly will be 'required.'[215] Further, Grisso and Appelbaum reject appeals to competence criteria based popular wisdom – i.e., they reject competence criteria tied to whether most people would consider the judgment wise or correct. As such, respect for autonomy in their model requires us to respect patients' decisions despite apparent eccentricity or inadvisability (although cases of gross deficiency to make a choice do not enjoy similar protection).[216] These criteria individually are necessary, but not sufficient, for autonomy – a marked inability to meet one of these criteria would render the autonomy of the decision suspect, but being able to meet one of these criteria is not sufficient evidence to render the autonomy of the decision beyond reproach.[217]

The most referenced criterion is that of *Understanding* – Grisso and Appelbaum note that courts often rely upon this in decisions about competence.[218] The concept, however, is quite tricky – the underlying mechanisms and processes of the 'Understanding' construct are not well known or easily defined:

> A person's accurate assimilation involves a complex series of events. First the information must be received as presented, a process that is influenced not only by sensory integrity, but also by perceptual functions such as attention and selective awareness. Whatever is received then undergoes cognitive processing and is encoded in a manner consistent with the person's existing fund of information and concepts, which in turn influences how, and how well, the message is recorded and stored in memory.[219]

This complex series of events is not the only mechanism by which cognition is influenced. There are a host of medical disorders, medications, and other injuries that can profoundly affect

[215] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 33.
[216] Grisso and Appelbaum, 34.
[217] Grisso and Appelbaum, 35.
[218] Grisso and Appelbaum, 38.
[219] Grisso and Appelbaum, 38.

cognition. The ease with which disruption occurs facilitates examination and assessment – if a lack of understanding seems evident, there is reason to suspect disrupted underlying cognitive mechanisms. This is not, however, a clearly defined case of cognitive deficiency – patients may *appear* to misunderstand information when the actual underlying mechanism is *miscommunication*.[220]

Grisso and Appelbaum note that *Appreciation* as a competence standard refers to whether patients appreciate that they have a disorder and acknowledge the consequences of that disorder and its treatments.[221] This use of the term parallels other authorities who refer to an absence of this appreciation and acknowledgement as demonstration of holding objectively false beliefs, explicable in terms of definite cognitive distortions. A caveat is introduced, however, in that this lack of appreciation or acknowledgement must be due to more than disagreement with the diagnosis. They note that several conditions are necessary to demonstrate that a distortion is present, rather than simple disagreement. First, the underlying beliefs the patient holds must be substantially irrational or unrealistic.[222] For instance, it is not unrealistic to doubt a diagnosis when conflicting information is presented, or there is evidence of clinical disagreement.[223] It is quite another issue, however, to doubt a diagnosis because one believes that he has superhuman

[220] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 41.
[221] Grisso and Appelbaum, 42-3..
[222] Grisso and Appelbaum, 45.
[223] As a quick example, a recent patient experienced a painful swelling on her foot and lower leg following a ballet rehearsal. The first clinician to examine her in the Emergency Department ruled out torn ligaments or tendons, noting that while the swelling had abated, a rash-like discoloration remained. Operating on the premise that it was either a reaction to a bacterial or viral infection of the fascia, he contacted infectious diseases and admitted the patient for what would amount to a ten-day stay. The rash did not respond to the treatments provided, and, in fact, the antibiotics administered provoked a further reaction on the patients hands and arms. The patient and her family became quite skeptical about the diagnosis, despite the insistence by the clinician that it was an infectious disease. Eventually an orthopedist – a friend of the family – visited, and immediately declared that the mysterious 'rash' was simply a bruise that resulted from torn ligaments; the hospital orthopedist concurred, and the patient was discharged later that day. Clearly the patient's and family's disagreement with the diagnosis was not unreasonable or irrational. Questions about the rationality of the patient's and family's beliefs would have been more appropriately raised had she claimed that she was immune to all diseases and infections.

powers. The second criterion is that the belief must be the consequence of impaired cognition or affect.[224] This is necessary in light of the objections of established religions to specific aspects of otherwise routine treatment – there is a fundamental difference between impaired cognition ("I have superhuman abilities") versus expressions of systemic values or belief sets (e.g., Jehovah's Witnesses prohibitions on using blood products). Some of these systemic beliefs sets may be considered by the clinician to be eccentric, but that does not mean that they can be ignored. The third criterion is that the belief must be relevant to the patient's treatment decision.[225] If the patient is exhibiting distorted cognition that does not reflect on the treatment decision at hand, it is not germane to an assessment of Appreciation. If a patient maintains the belief that gravity does not apply to him, but manifests no treatment-relevant cognitive distortions, there is no compelling reason to doubt his ability to appreciate other information.[226]

As mentioned in chapter three, comorbid depression is a significant concern, in that it directly affects morbidity and mortality, and it is frequently underdiagnosed. There is a common reaction in medicine that patients are expected to react negatively to bad health news – in fact, many consider it a sign of pathology if bad news does not engender some manner of depressive reaction. However, this can have a profound impact on the course of treatment – clinicians can quite easily endorse decisions of questionable competence:

> It is easy for caregivers to perceive patients' expressions of discouragement or hopelessness as reasonable reactions to patients' illnesses, when they may actually reflect their depressed moods. This 'false empathy' can lead clinicians to accept decisions to forego medical treatment that are, in fact, of questionable competence. Since it is often impossible to know the extent to which depression

[224] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 47.
[225] Grisso and Appelbaum, 48.
[226] For instance, over the past few years I have worked with patients with schizophrenia of a variety of severities and degrees of subsequent cognitive impairments, including auditory and visual hallucinations, perceived conspiracies and threats, irrational degrees of grandiosity, and with varying degrees of insight into their conditions. This has not prevented them from being able to engage and process information in many other areas of their lives, nor has their illness prevented many of them from being able to appreciate their clinical situation and course of treatment.

> is affecting patients' appreciation, evaluators need to have a high index of suspicion. The best course may be to defer a decision about patients' capacities, and about the proper course of treatment, while antidepressant medication is instituted on an empirical basis.[227]

As has been stressed before, it may be preferable to err on the side of caution when there is evidence of cognitive distortion. Not all cases will be clear cut, and require significant sensitivity to the biopsychosocial elements of the disease and its pathophysiology.

The *Reasoning* criterion requires that patients be able to engage in logical cognitive processes using the information they understand and appreciate.[228] As noted above, there is significant concern that one may be given information but not be able to use it. Cases of anterograde amnesia, for instance, present challenges to processing because of the speed with which information is forgotten. Alzheimer's dementia and cerebrovascular accidents near memory structures carry similar risks – they prevent individuals from being able to work with new information presented to them. As such, clinicians assessing competence in patients with conditions similar to these ought to be aware of potential influences. Grisso and Appelbaum caution, however, that this criterion ought not be used to deny individuals their right to autonomy simply because they employ non-normative approaches to information processing.[229] They note that most, if not all, individuals fail to meet idealized standards of decision-making in everyday situations, and that these deficits may become more apparent in times of crisis. As such, they stress that Reasoning deficits should focus on cases in which "a patient's mental abilities are so

[227] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 51.
[228] Grisso and Appelbaum, 52.
[229] Grisso and Appelbaum, 54.

impaired by illness or disability that even basic functioning with regard to these considerations is seriously and negatively influenced."[230]

Grisso and Appelbaum stress that certain cases merit greater attention than others – significant changes in mental functioning (generally with behavioral correlates) should serve as warning signals that cognition has been altered.[231] While refusal of treatment or evaluation may be atypical for a particular patient, that alone does not suffice to demonstrate that cognitive changes have occurred, but it should serve as a warning sign, for several reasons:

> First, physicians usually try to recommend treatment that will minimize the impact of the patient's disorder. Declining to follow that recommendation raises the risks of an adverse outcome for the patient. Although patients may sometimes know more than their physicians do about what is best for them, often they do not. Their decisions may reflect misinformation, irrational biases, or the influence of other persons without extensive medical knowledge. In most circumstances, rejecting the treatment recommended by a physician is a riskier decision than agreeing to the proposal. If one of the goals of the competence requirement is to protect patients from bad outcomes, this would seem like a situation in which particular attention to patients' capacities might well be warranted…The second justification for centering attention on refusals of treatment is the empirical reality that many patients who refuse treatment do so because of diminished decision-making capacities. To admit this is by no means to agree that every patient who differs with his or her physician is incompetent. But as we have seen in the case examples in the preceding chapters, intellectual impairment, attentional difficulties, anxiety denial, delusions, dementia and other manifestations of psychopathology and impaired cognition can all lead patients to reject recommendations for treatment.[232]

They note that patients with organic impairments are especially prone to decisional incapacity (e.g., dementias, deliriums, etc.). They further note that while depression has been a frequently studied group, the results have varied (as demonstrated in the first half of this chapter). They suggest that the differences in the research findings may reflect different degrees of depression,

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[230] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 55.

[231] By this they mean patients behaving in manners contrary to their normal presentation and personality (e.g., fastidious patients who have become slovenly, gregarious patients who are withdrawn and asocial, etc.). They note that elderly patients are particularly at risk for manifesting these types of changes.

[232] Grisso and Appelbaum, 64.

with correspondingly different degrees of impairment.[233] Further, influencing factors are additive – comorbid psychopathologies can exacerbate cognitive distortions and disabilities, which are further exacerbated by medical illness and pharmacological interventions, with polypharmacy being especially problematic (and, among elderly patients, all too common). While age itself does not *necessarily* reduce competence, it does increase susceptibility to decisional impairment.[234]

The metaphor proposed by Grisso and Appelbaum is a scale whose cups are labelled 'autonomy' and 'protection'.[235] The fulcrum is off center, allowing autonomy a natural advantage (representing social preference for personal autonomy). In the context of a patient either providing or refusing consent to a particular treatment, assessment of information is added to each side, with evidence supporting competence filling the 'autonomy' cup, and evidence undermining competence filling the 'protection' cup. Clearly in this model it requires more evidence to countermand the patient's autonomy than it does to countermand the duty to protect him or her. It is very uncommon for a patient to completely lose her capacity for Understanding, Appreciation, or Reasoning – as these are continuum concepts, it is more likely that the patient's abilities will simply experience a reduced capacity. As such, clinician's need to be cognizant of the degree of impairment when balancing the metaphorical scale.[236] The consequence of maintaining this balancing metaphor is a sliding standard of competence dependent upon risk-gain ratio analysis of the intervention in question:

> In effect, a low probable gain-high risk decision, which adds substantial weight to the cup representing protection, will require a much greater weight of ability in the opposing cup to keep the scale tipped towards autonomy. In contrast, if the

[233] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 71.
[234] Grisso and Appelbaum, 75.
[235] Grisso and Appelbaum, 130-1.
[236] Grisso and Appelbaum, 133.

> patient's preference is for a high probable gain-low risk treatment, a lower degree of ability is acceptable; less weight in the autonomy cup will be required for the patient to be perceived as competent.[237]

The fulcrum of the scale is also subject to adjustment – Grisso and Appelbaum allow the clinician to move the fulcrum dependent upon the treatment preferences of the patient. For instance, if the patient elects a procedure that has a less desirable risk-gain ratio than the intervention proposed by the clinician, the fulcrum may be adjusted slightly, requiring more evidence of competence than would normally be required. The patient, however, would need to be duly informed that greater decisional capacity must be demonstrated before the preferred treatment is initiated.

There are significant strengths in this model – for instance, its awareness of the complex interactions of illness and cognition, its understanding that normal judgment can be biased by a variety of sources not normally accounted for in other autonomy models, etc. There are some concerns, however, in that it does not acknowledge that clinicians themselves can demonstrate cognitive biases. Studies have demonstrated that clinicians tend to focus on one particular diagnosis, ignoring others (displaying the anchoring heuristic), for instance.[238] The very same cognitive heuristics that plague patient decision-makers are found in the clinical staff treating them; as such, awareness of cognitive biases and distortions is not a one-way process. The model proposed by Grisso and Appelbaum would be strengthened by a more dialogical

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[237] Grisso and Appelbaum, *Assessing Competence to Consent to Treatment: A Guide for Physicians and Other Health Professionals*, 139.

[238] I recall a passionate discussion I had with one psychiatrist who insisted that a patient was a chronic paranoid schizophrenic, simply because he had carried that diagnosis for several years. The difficulty, however, was that the differential was wider than this particular diagnosis – specifically, he showed considerable evidence of a frontal lobe syndrome. Specifically, he chronically abused crack cocaine (which in long-term abusers produces feelings of paranoia, as well as auditory, visual, and tactile hallucinations), per his family history he had had a traumatic brain injury prior to the onset of his symptoms, his personality was very childlike, irresponsible, and sexually preoccupied, and his affect was not flattened (flat affect is characteristic of chronic paranoid schizophrenia).

approach, in which the distortions and biases of both physician and patient are exposed and challenged.

Katz

The psychodynamics of the physician-patient is a key element of the autonomy model proposed by Jay Katz.[239] Katz notes that there are many definitions of autonomy, but chooses to focus on what he refers to as 'psychological autonomy' – the capacity of persons to exercise the right to self-determination, which includes their ability to reflect on the choices they have made.[240] He further notes that current conceptions of autonomy make a significant number of psychological assumptions which go unexplored in the literature. Contemporary medical ethics is dominated by abstractions – specifically, abstract norms that generalize conduct in a manner that is inappropriate when considering how human agents actually behave. Ethicists have a tendency to rely upon the theories of Kant and Mill, among other philosophers, to relate the abstract formal norms to material situations. These abstractions contain implicit models of the human psyche which are not developed or clarified, which is unfortunate, in that "[a] careful scrutiny of many philosophical, moral, political or legal principles reveals all kinds of hidden, albeit woefully mutilated, assumptions about human nature."[241]

Paradigmatic in medical ethics are the assumptions made by Immanuel Kant – his idealized moral agent is a being of pure rationality; in the ideal agent, moral decision making will not be influenced by whims, emotions, or personal inclinations. Katz notes that current philosophers have championed this model – but the problem lies in that the model itself is untenable.[242] Kant himself noted that he was making a distinction between an *idealized* moral

[239] Katz, *The Silent World of Doctor and Patient* (Baltimore: The Johns Hopkins University Press, 2002).
[240] Katz, *The Silent World of Doctor and Patient,* 105-6.
[241] Katz, 108.
[242] Katz, 109.

agent, which he distinguishes from *actual* moral agents – it was a *theoretical* model, not a *practical* model. Kant's model recognizes only one aspect of human behavior – the capacity for rational thought – but ignores many other aspects of our behavior, which is contingent upon other processes, some of which are completely irrational. Because we can be influenced by so many different aspects of our rational and irrational nature, Katz notes that Kant's model is simply impractical, and therefore is irrelevant in practical situations:

> Human beings are subject to the influence of reason and unreason, with the relative strength of either being affected by many innate, developmental, and situational factors. Moreover, capacities for reason are impaired whenever human beings are in pain, in love, in mourning, or in the throes of biological, environmental, or social crises. Kant's theoretical conception of the nature of human beings is too neglectful of the complex interrelations between reason, emotions, and the external world; it is therefore of little relevance to practical situations.[243]

As a result of this irrelevancy, Katz adopts an autonomy radically different than Kant's ideal – psychological autonomy. Katz's clarifies his definition of the concept, noting that as an *ideal* definition, "psychological autonomy refers to the capacity of persons to reflect, choose, and act with an awareness of the internal and external influences and reasons that they would wish to accept."[244] Katz stresses that this is an ideal – the sheer volume of internal and external influences makes it impossible for a moral agent to ever be *fully* aware of them all.[245] Self-reflection and dialogic interaction with others can help to draw out unconscious influences, returning them to the control of the agent.

Katz notes that past discussion of psychological capacities of moral agents has tended to reflect psychopathology instead of underlying motives, i.e., questions of incompetence. Katz

[243] Katz, *The Silent World of Doctor and Patient*, 109-10.
[244] Katz, 111.
[245] In discussing internal influences, Katz is arguing from a Freudian perspective on conscious and unconscious processes, instead of the sense of the conscious, unconscious, and preconscious cognition developed in chapter two. The two are very different – the unconscious, for instance, is the domain of libidinal urges, mediated by the ego and superego in Freudian thought, while unconscious processes like heuristics and biases, information integration, and automaticity are what is meant by the term in my argument.

supports those who conclude that only the choices of clearly incompetent patients should be rejected – he argues that it quite different to recognize the sources influencing a patient and interfering with the patient's choice when one believes that they have made the 'wrong choice.'[246] There are implicit dangers in raising psychological objections to patient autonomy – he notes that exceptions to autonomy can be too readily 'found' and that the purview of psychological objections are too far-reaching and too difficult to control. This represents a significant break between Katz's model and my own – while I can appreciate his concern regarding the ease with which questions and challenges to autonomy can be raised, it would seem that the circumstances and the choices to be made would dictate the standard of psychological evidence necessary to maintain patient autonomy (as per Grisso and Appelbaum's model). I will return to this objection below.

At this point, Katz develops the sense of the unconscious employed in his model. Employing a psychodynamic approach, he breaks from other models which suggest that unconscious elements are to be identified, evaluated, and potentially discarded. Specifically he notes the central role of the unconscious in normal decision-making – the psychodynamic perspective seeks to *understand and account for* unconscious influences, rather than *identifying and eliminating* them, as well as identifying potential conflicts between conscious and unconscious motivations.[247] Further, the conscious/unconscious split is not the only germane factor – cognitive modelling of autonomy must take into account the rational/irrational split, as our decision-making process incorporates both. It is extraordinarily rare to find actions that stem from only one motivational source, and the rational/irrational mixture are idiosyncratic, and vary with the individual's situation. In Katz's model, 'rational' and 'irrational' reflect "capacities for

[246] Katz, *The Silent World of Doctor and Patient*, 113.
[247] Katz, 115.

adaptation to the external world, that is, persons' conscious and unconscious efforts to reconcile their internal mental processes with the external possibilities and limitations of the world in which they live. They denote persons' abilities to take reality into account and to give some account of the conflicts between their inner and outer worlds to themselves and others."[248] As a result, ideal decision-making will be a dialogic process, in which the idiosyncrasies of both the patient and the clinician can be explored, leading to a greater understanding of the motivations and thought processes of both. This dialogue is not likely to reveal all unconscious motives, but it can reveal more than might be accessible solely through introspection and reflection.[249]

This model has immediate consequences for individual autonomy and liberty. Katz notes that it immediately undermines two concepts in the autonomy debate – radicalized patient autonomy, and standards of perfect understanding in the clinician:

> If I am correct, then individual freedom should be equated neither with simply permitting patients to do what they initially desire nor with requiring them simply to make complete sense to their physicians. Instead, and above all, respect for freedom would demand respectful conversation. True freedom entails constant struggle and anguish with oneself and with others. This is the lesson of psychoanalysis and its theories about human conduct and interactions.[250]

By being aware of the limits of human thought, both conscious and unconscious, rational and irrational, clinicians and patients can achieve a greater understanding and awareness of their own thoughts and motivations, and allow them to recognize how their perspectives and experience have influenced them directly and indirectly. This, in turn, gives rise to greater freedom in decision-making – the more motivational factors we are conscious of, the more control we exercise in the decision-making process. This will never produce absolute control, however, and

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[248] Katz, *The Silent World of Doctor and Patient*, 117.
[249] This is comparable to the adage that 'two heads are better than one.' Individual perception tends not to be self-challenged; the presence of another individual capable of evaluating both the situation as well as the other individuals perception.
[250] Katz, 121.

as such, there is always an influence of unconscious and irrational factors in human thought. As such, the first, necessary step in self-determination is self-reflection and reflection with others.[251] This reflection may not produce agreement with the physician and patient, but it can clear up misunderstandings and misperceptions. Katz still opens the door to physicians being able to interfere in patient decisions (and hence to weak paternalism in Beauchamp and Childress's sense of the term), but he stresses that neither party is asked to submit to the other, and that conversation and shared decision-making prevent significant harms:

> In conversation with one another, patients may uncover mistaken notions about their diseases and their treatment that they have held for a long time or have recently acquired through misunderstanding the import of their doctors' recommendations. Physicians may uncover the fact that their unconscious preferences and biases compelled patients to yield to their recommendations even though consciously they had intended otherwise. Without conversation, individual self-determination can become compromised by condemning physicians and patients to the isolation of solitary decision making, which can only contribute to abandoning patients prematurely to an ill-considered fate.[252]

If our aim is to facilitate autonomous decision-making, a recurring theme in myriad theories of medical ethics, it seems that conversation and mutual exploration of motives and thought processes are necessary foundational criteria. But what should be done if the patient insists on medical decisions fundamentally at odds with the opinion of the clinician? Katz argues that if we adopt the psychological autonomy model he proposes, clinicians will be required at times to accede to 'foolish choices' – as a matter of principle of respect, the clinician does not possess the ability to simply overrule any decision which he feels to be ill-advised[253] – I will address this aspect of Katz's model below.

[251] Katz, *The Silent World of Doctor and Patient*, 124.
[252] Katz, 128.
[253] Katz, 154.

Katz's allows for clinicians to disobey a patient's choice only when two conditions have been met.[254] First, the consequences of the decision must pose significant risks to the patient's immediate physical condition. Katz clarifies this by limiting it to cases in which the patient's illness has interventions which have a good chance of preventing death or persistent serious injury, and when such outcomes are likely in a relatively short period of time. The second condition requires that the patient's cognitive processes are so seriously impaired that neither the clinician nor the patient can understand each other. If there is no apparent means of overcoming the communication barrier, then it is reasonable to proceed in the patient's best medical interest. These are very limited conditions, to be sure, but Katz argues that one ought to err on the side of autonomy. This does not create absolute patient autonomy, however, as Katz is cognizant of challenges which might arise as a result:

> If all final authority is vested in patients, the danger is great that in situations of either a total refusal to give an account of one's reasons or an unwillingness to explore one's possible confusion – when the need for conversation is the greatest – doctors will wittingly and unwittingly give up on conversation and patients prematurely because they have been stripped of all power to stop even patients' most inexplicable self-destructive course. To protect doctors and, in turn, patients from such pernicious consequences supports the creation of a *rare* exception to the rule that doctors otherwise must obey: In case of disagreement, doctors and patients should either go their separate ways, or agree to provide and to receive care within the limits imposed by the patient.[255]

Hence, significant authority remains with the patient, but not total authority – respect is a principle that is not unidirectional. Many theories of medical ethics note that clinicians are not automatons – they have moral values and beliefs, just like the patient. One cannot expect a clinician to ignore her own important principles in medical decision-making.

There are significant strengths in the model proposed by Katz. It is clear that recognition of the complex cognitive processes underlying decision-making is emphasized in this model. As

[254] Katz, *The Silent World of Doctor and Patient,* 157-8.
[255] Katz, 163.

a corollary, recognition that both patients and clinicians carry with them their own sets of rationalities and irrationalities is an important step in shared decision-making. This model explicitly requires the identification and exploration of unconscious cognitive factors for both (or all) parties involved in decision-making, in an effort to increase understanding. This allows for critical insight that might be unavailable were one to attempt simple self-exploration and self-reflection. The emphasis on a dialogic process as a requisite first step towards self-determination clearly demonstrates the need for the patient to understand himself before he can make informed decisions. It is quite clear that we cannot make meaningful decisions if we are unclear as to what it is that we want. We can certainly make choices, but it is evident that they may not actually reflect our values or beliefs – in short, they will lack the 'self' criterion of self-determination.

However, there are some concerns about Katz's model as well. First, it is unclear that one ought to adopt a Freudian model of the unconscious, as there are significant methodological, empirical, and theoretical concerns about the Freudian model.[256] It is clear that unconscious processes influence cognition, but the empirical data and research support the model proposed in chapter two much more readily than Freudian analysis. As such, when unconscious motivations are discussed later, it will not be in the terms Katz's proposes, but rather a reflection of automaticity, heuristics and biases, and emotionally-valenced memory and recall.

[256] In terms of *methodological* concerns, Freud was not research oriented. The case studies he selected were not experiments – they were self-selected case studies designed to develop the theory, not test it. In fact, a recurrent criticism of Freudian models is that they do not translate easily – if at all – into testable variables. There are *empirical* questions as well – Freudian psychotherapy and analysis requires significant time and effort – it is common for patients to see their analyst for years before any insight is drawn. This is clearly beyond the purview of a normal in-patient stay. It is much more likely that Katz is advocating a more superficial variant of Freudian analysis, but even in this abbreviated sense, it remains unclear that the average clinician would have the requisite training or understanding needed to identify unconscious motivations. The *theoretical* concerns raised stem from Freud's own statements – as he approached the end of his life, he raised his own concerns as to whether psychoanalysis was actually helpful. If the founder of the school of thought questions its use, one ought to be skeptical about arguments built from the suspect theory.

Second, the criteria set by Katz for incompetence appear to be too high. It is understandable that he would establish such strict criteria in light of the psychoanalytic model he proposes, which integrates the unconscious, but as that methodology is suspect, it seems reasonable to question the need for such restrictive criteria. This is not to say that clinicians ought to have *carte blanche* in deciding which decisions to accept or to reject, but it certainly suggests that the standards for rejecting bad choices ought to be lowered. It is clear that cognition is dependent on a variety of factors, of which we are only aware of the surface phenomena. It is likewise clear that our cognition can be affected in manners great and small at a variety of levels of reduction. It would therefore seem to be reasonable to suggest that clinicians have more leeway than Katz's proposes in challenging the decision-making process of patients, who by their nature are more vulnerable to influences due to medical illness, pharmacology, and potential psychopathology. I do not challenge the idea that patients have the right to make bad choices; I do challenge the idea that this right is an absolute, especially as the consequences of their decisions increases in severity. As suggested earlier, it seems that a quite compelling case can be made for a sliding scale of autonomy, contingent upon the severity of the predicted outcomes, with the most scrutiny applied to terminal decisions.

Anderson and Lux

Higher cognitive standards are established by Anderson and Lux,[257] who argue that the keystone of autonomy and self-determination is 'accurate self-assessment.' They argue that autonomy is contingent upon an ability to recognize impairments in one's own cognitive capacities.[258] They offer the clinical case of 'John' – a patient who experienced severe frontal

[257] Anderson and Warren Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," *Philosophy, Psychiatry, and Psychology* 11, no. 4 (December 2004): 279-94.
[258] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Authority," 279.

lobe injury, which severed his optic nerves (as a result, he had no perception of light at all). As a result of his accident, John experienced a fascinating cognitive impairment: despite the severing of his optic nerves, John was unaware that he was blind. Consequently, he would attempt to navigate his way around as he would were his vision normal, with the result that he would walk into walls, trip over furniture, and found himself in various dangerous situations for one who cannot see. Anderson and Lux argue that his actions ought not to be considered autonomous, not because of his visual impairment, but because of *his cognitive inability to recognize that he had a visual impairment*. This is to say, they argue, "[a]t least with respect to those actions, he was deeply alienated from himself as an agent."[259] There are a number of types of agnosognosia (being unaware that one is unaware of a deficit) – visual, auditory, etc. – each of which pose the same kind of problem for one's self-concept. Further, there are multiple conditions which produce similar deficits in one's sense of self – V.S. Ramachandran, Oliver Sacks, and others describe neurological conditions in which a patient experiences a disconnect between sense data and association cortices, sense data and perception, perception and association cortices, sense data and emotional valence, etc.[260] Clearly it is possible to meet previously proposed criteria for autonomy and yet experience a profound deficit in self-perception. As such, it makes eminent sense for clinicians to examine self-perception for accuracy before asking patients about treatment preferences – if their self-perception is unrealistic or bizarre, there is reason to believe that decisions made upon these perceptions will also be compromised.

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[259] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 280.

[260] Interestingly, Ramachandran describes a procedure that temporarily alleviated post-stroke agnosognosia. Checking for nystagmus involves injecting cold water into the left ear (one of the test performed in some brain-death protocols). Ramachandran found that individuals with a variant of agnosognosia regained an accurate picture of their physical condition (albeit temporary) following the water treatment. See .S. Ramachandran and Sandra Blakeslee, *Phantoms in the Brain: Probing the Mysteries of the Human Mind* (New York: Quill, 1998). for more information.

Anderson and Lux draw parallels to the category of 'insight into illness' in establishing their criterion of accurate self-assessment.[261] A variety of conditions manifest decreased insight – there are several psychiatric illnesses in which the patient categorically denies any illness.[262] Inaccurate self-assessment in Anderson and Lux's sense has three criteria.[263] First, the patient must intentionally undertake a given task. Several authors have noted that intentional action is a requisite part of autonomy and self-determination; accidental actions are not intentional, and as such, are not dependent upon an agent's belief about their skill in performing said action. The second criterion is that the agent believes that she will be able to perform the given task as it is intended. That is to say, the agent believes that she possesses the requisite skill and ability to complete the task. The third criterion is that this self-assessment of capacity must be inaccurate. Specifically, the agent objectively must not possess the requisite skill or ability in question. It must be demonstrable that the agent possesses a deficit that she does not believe she has. This lack of insight is quite relevant to the sense of autonomy developed in this dissertation, a topic we will return to later.

When erroneous beliefs are examined, these self-perceptions are not understood in terms of whether they are subjectively reasonable, but rather whether they correspond with the facts of the case. This lack of insight does not translate into global incompetence – rather, it is a task-specific deficit.[264] As such, we see that clinicians assessing insight must possess an accurate understanding of the degree of skill necessary to complete the task in question – if the evaluator's criteria for normal function are set too high, it is entirely possible that competent

[261] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 280.
[262] For instance, I worked with a patient for several years who maintained vociferously that while he was the son of a famous martial artist, was engaged to/married to/dating a pop starlet (the relationship would change from day to day), was a commander in the Navy, Air Force, and Army, and was designing ships for NASA, all while playing with the band Metallica, he was most assuredly not schizophrenic.
[263] Anderson and Lux, 281.
[264] Anderson and Lux, 281.

individuals will be judged incompetent. This is not the only continuum involved in testing accurate self-assessments – in addition to standards varying with the task, the self-assessment itself is a statement of probability. Further, Anderson and Lux argue that there is no single threshold for accuracy, and hence no threshold for autonomy – for most individuals and for most occasions, a general self-assessment of one's capacities should suffice. They suggest that the cases in which inaccurate self-assessment produces non-autonomous actions will be severe enough as to be immediately recognizable (e.g., stumbling into furniture that one cannot see, but claiming no visual impairment). Some agents are able to recognize that they are experiencing cognitive deficits, and can act to correct them or to incorporate them into their cognitive modeling. Anderson and Lux argue that the capacity (and hence the autonomy) of these individuals is still compromised in some degree, but less than it was before (maintaining the continuum approach to autonomy.[265] They further note that just as individuals with cognitive deficits can overestimate their abilities, so too can they underestimate their abilities.[266]

Anderson and Lux stress that the establishment of non-autonomous actions requires more than simple demonstration that the patient is making poor choices or has some unjustified beliefs. They suggest that autonomy does include the ability to make mistakes. As such, they stress that in utilizing their proposed criteria, it must be clear that the deficit in question is preventing the agent from exercising self-governance – i.e., there must be something inherent in the deficit that prevents autonomy itself.[267] There are several methods by which this may be assessed, and Anderson and Lux focus on two. First, it is possible to explore the causal link between the action and the source of the action – if the action occurs in such a way as to prevent

[265] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 282.
[266] In fact, this is a frequent topic in therapy sessions, both physical, occupational, and psychological.
[267] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 282-3.

evaluation of the motives behind one's action, then the causal pathway has been disrupted, preventing the agent from taking ownership of the action.[268] This is a key concept, and one which will be revisited later. The second method by which ownership of the action can be disrupted concerns problems in integrating the action with its motivations – the agent cannot make sense of his motives or is alienated from them (i.e., the agent experiences a baffling "Why did I do that?" moment). If the agent cannot understand and reconcile his motivations with his actions, there is reason to believe that they are non-autonomous. Anderson and Lux note that these two concerns demonstrate the need for integrated actions, as well as a means of registering that integration has not occurred – a feedback mechanism, in short. They note that this feedback mechanism "must be constituted in such a way that the unintelligibility surfaces. For to the extent to which one is unable to note the internal tensions, one is without this compass, which is so crucial for guiding one's actions in the manner we dub 'autonomous.' And this is why rigidly inaccurate self-assessments undermine autonomy."[269] In short, absent this feedback mechanism, our compass is broken, and we have no way of knowing whether we are moving in the right direction. For all we know, instead of reaching our goal, we could be simply traveling in circles. The primacy of accurate self-assessment carries with it a three-fold advantage: first, it is neutral in regards to competing theories; second, it is more plausibly linked with self-direction in autonomy; and third, it is more empirically supported in clinical neuroscience.[270]

The aspect of Anderson and Lux's analysis that is most crucial to the argument developed in the present work is that they extend it to cover mental as well as physical incapacities.[271] As the analysis in chapter two notes, automaticity, cognitive heuristics and biases, and emotional

[268] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 283.
[269] Anderson and Lux, 284.
[270] Anderson and Lux, 285.
[271] Anderson and Lux, 286.

valencing occur outside of our awareness, and constitute significant but correctable sources of error and distortion. It would seem that these types of errors dovetail with Anderson and Lux's analysis; it is necessary to note, however, that they focus their analysis on traumatic brain injuries, rather than on phenomena of cognitive psychology. However, as the psychological phenomena in question have empirical bases, it seems evident that such considerations as Anderson and Lux propose ought to be extended to them as well. In fact, Anderson and Lux note that the criteria they develop can be applied outside of traumatic brain injury. They note that "[o]ur self-assessments are sometimes off-the-mark, but to the extent to which we can reduce the inaccuracies in our self-assessments, we are better able to guide our actions in a fully autonomous sense."[272]

As with the other cognitive models proposed, there are significant strengths in Anderson and Lux's model. Meaningful self-direction is impossible if one's compass is flawed and there is no way to check it. To the extent that we can become aware of our own cognitive shortcomings, we can correspondingly increase our personal autonomy.

There are weaknesses to be found, however. First, it is unclear how far back they are willing to extend their analysis cognitively. As was noted earlier, the literature on cognitive phenomena discussed in chapter two provides clear indications of the kinds of error that can systematically creep into one's cognition. The kinds of deficits produced by the conditions Anderson and Lux consider also produce systematic error, since they produce a recurring mistaken belief. It is unclear, however, whether Anderson and Lux intend for their argument to be extended to the automatic and backstage elements discussed in the present argument. If they are unwilling to extend their analysis to the types of cognitive errors discussed in chapter two, it

[272] Anderson and Lux, "Knowing Your Own Strength: Accurate Self-Assessment as a Requirement for Personal Autonomy," 291.

would seem a rather arbitrary distinction, and the autonomy model proposed would certainly require clarification.

The second weakness is that while the model raises compelling arguments, it does not establish a clear metric for establishing non-autonomous actions. They do specify some criteria, but they also place these criteria upon continua, which allows for significant room for interpretation. For the autonomy standard to be meaningful, it would seem that a little more structure or clarity is needed for clinical application beyond claims that distortions and corresponding non-autonomy will be immediately recognizable.

A third concern is that this is not a fully-developed theory of autonomy. To be fair, it does not appear to be intended as such, but the criterion of accuracy in self-perception is a necessary, but not sufficient, element of autonomy. It is quite clear that individuals can act in non-autonomous ways while maintaining accurate perceptions of their abilities. Additional criteria, as have been explicated in the previously discussed models, are critical to an accurate and meaningful picture of autonomy.

CONCLUSION

The model that emerges from this discussion must necessarily take into account multiple factors drawn from the strengths of the homuncular and cognitive models of autonomy. Four key categories of autonomy criteria emerge – foundational, medical, psychiatric, and psychosocial. Each of these categories is necessary for an autonomous action, but none are sufficient. Each will be explored in turn.

Before presenting them, however, there are several caveats. First, it must be made clear that this model ought only to be considered applicable to end-of-life decisions. It is quite clear that this kind of decisional process has little day-to-day validity – it will not pass the 'buying

bread' test. However, as has been suggested earlier, a compelling argument can be raised that as the consequences of our decisions become more severe, greater evidence is needed that the action is autonomous. In terminal decisions, it is unclear why a lower evidentiary standard should be preferred. Second, this model is intended for use in cases when a patient is awake, aware, and able to voice her own preferences. Last, quite obviously this should not be understood as a fully developed theory of medical ethics, nor should it be seen as anything other than criteria necessary for autonomous action as evidenced by the theoretical and empirical challenges raised to the autonomy models found in contemporary theories. It is quite possible to incorporate this understanding of autonomy in existing models (e.g., substituting a cognitive model of patient autonomy would not fundamentally undermine Beauchamp and Childress's principlism), albeit in some more than others (this model *does* present a fundamental challenge to models giving disproportionate weight to autonomy, e.g., Veatch).

Foundational Criteria of Autonomy

Foundational criteria of autonomy refer to underlying psychological structures, in the sense developed in chapter two and the current chapter. Foundational structures are primary and fundamental – absent these criteria, significant doubt can be raised about the autonomy of the patient's decision. There are five criteria in this category: the ability to consider, make, and make known one's preferences (which I will refer to as capacity for preference); intentionality in action; accurate self-assessment; awareness of common sources of cognitive error (which I will refer to as bias vigilance); and dialogue aimed at self-discovery, which includes the willingness to participate in dialogue. There is no lexical priority for these criteria, and they fit into both absolute and continuum scales.[273] Each of these requires further exploration and clarification.

[273] As a necessary caveat and matter of clinical significance – I realize that these proposed standards are theoretical, and may have some difficulty translating well into clinical settings (e.g., discussions of backstage cognition). This is

Capacity for Preference

In this criterion, the moral agent engages in reflection upon the treatment options open to her, weighs their strengths and weaknesses as she understands them, and makes her preferences known in some manner to the clinician (ideally through a contemporaneous statement). By its very nature, this will post challenges, as the interpretation the patient gives to the treatment option will be contingent upon her perception and understanding, which may require further discussion and dialogue with the clinician, to ensure as much accuracy as possible. This capacity for preference is not absolute, in that patients will differ in both the degree of their preferences as well as their ability to communicate them. Patients unable to weigh information or express preferences due to cognitive impairment or illness ought not to be considered autonomous agents, and treating clinicians should defer to a best-interest standard until the impairment is resolved or a proxy decision-maker is identified.

Intentionality

Several theories have noted the necessity of this criterion. For an action to be personally meaningful and autonomous, it must be intended and not accidental or reflexive. It is entirely possible to act without meaning to act, and a number of neurological and psychiatric conditions have demonstrated that involuntary actions can be physical or verbal. As has been demonstrated in chapter two, mental actions are also driven by automaticity, and therefore the agent may find herself acting or thinking in a manner she does not desire. Following earlier theories, this is an absolute scale – either one intends to act or one does not, and it is quite possible to discern between the two. Unintended actions ought not be considered autonomous.

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a barrier faced by cognitive therapies in psychology, as well – the theoretical concepts will be dependent upon the underlying cognitive capacity of the patient in question. This can be resolved by using age-, understanding-, or education-appropriate terms (e.g., switching "People frequently make systematic cognitive errors in information processing." with "Sometimes we can get so used to thinking about things some way that we forget there are other ways to see it.")

Accurate Self-Assessment

Following Anderson and Lux's argument, agents must have insight into their illness. If a patient demonstrates agnosognosia, whether correctable or resistant, their autonomy has been weakened. Following Anderson and Lux's analogy, if a patient demonstrates a consistent source of error germane to their medical decision-making process, they cannot process the information necessary to make the judgment (or can only do so in a diminished capacity), and as such lack the insight necessary to be self-directing. This analysis extends not just to awareness of physical injury, but also to persistent cognitive errors and distortions, per the earlier discussion of Anderson and Lux's theory. This criterion exists along a continuum, as the degree of accurate self-assessment increases, so to does autonomy increase.

Bias Vigilance

Given that cognitive biases and sources of error are so prevalent in 'normal' cognition, and that special circumstances may exist in patients with depression, patients must be educated regarding common sources of cognitive error. This does not mean that the patient must hold a doctorate in psychology, but she must be made aware of the ways in which we frequently misinterpret information, emotional information, and memory. This is a continuum criteria, as patient understanding is variable. If a patient demonstrates an inability to understand backstage cognition (i.e., an inability to recognize that thought can be influenced by other conditions [environmental triggers, personal biases, heuristics, etc.]), there is reason to question her autonomy.[274] This criterion ties in directly with Dialogic Self-Discovery.

[274] This argument will no doubt raise significant questions, and so I feel it requires further clarification. I am not arguing that if the patient is *skeptical* about the information they are not autonomous – simple examples can demonstrate heuristical thinking, which should permit the patient to at least be willing to entertain the idea, in an effort to facilitate Dialogic Self-Discovery. If a patient demonstrates a profound *inability* to conceptualize backstage cognition, there is reason to suspect compromised autonomy.

Dialogic Self-Discovery

As has been demonstrated earlier, it is quite common that we are unaware of the idiosyncratic and systematic slants we place upon the information we take in, or upon the memories we selectively recall. These biases and slants can be explored in a shared decision-making model as proposed by Katz. While the content is somewhat different than Katz's model, in that the clinician and patient are not attempting to explore the Freudian unconscious, the aim is similar – dialogic interaction can provide illumination on those processes that evade self-exploration and reflection. This criterion exists along a continuum for two reasons: first, patients will have varying degrees of insight, so the amount of benefit from dialogic interaction will vary from patient to patient; and second, patients will have varying degrees of willingness to participate in dialogic self-discovery. The more open a patient is to self-discovery, the greater the likelihood of an autonomous action resulting. If a patient categorically refuses to engage in dialogic self-discovery, there is reason to suspect compromised autonomy, but not necessarily proof.[275]

Medical Criteria of Autonomy

Medical criteria concern issues that are the traditional purview of medical treatment; i.e., these are routine elements that recur in many theories of medical ethics. There are two key medical criteria for patient autonomy: the absence of a medical condition which directly affects cognition (which I will refer to as Structural Integrity), and access to the information typically required for informed consent. Both of these criteria are continuum-based, as disease processes

[275] There is also the possibility that the patient simply does not want to discuss the matter any further for a variety of reasons (e.g., irritation with the clinical staff, fatigue, pain, personality disorder, desire for privacy, guilt, crisis of faith, etc.). In the event that a patient expresses unwillingness to engage in dialogic self-discovery, it would behoove the clinical staff to identify and document the reasons for refusal, alleviate whatever conditions are immediately preventative (e.g., fatigue or pain), and attempt at a later time, when the patient may be more receptive. Reluctance or refusal are not necessarily indications of compromised autonomy.

result in different degrees of impairment, and some pieces of information might be more relevant or available than others.

Structural Integrity

The most significant challenge to patient autonomy in the models discussed is a physical impairment which prevents the patient from taking in information or processing it. Dementia, delirium, traumatic brain injury, cerebrovascular accidents, etc., can exert profound effects on the ability of the patient to take in new information, make their preferences known, form associations between concepts or words, etc., all of which are necessary elements of cognition. Clearly any illness which fundamentally disrupts this process prevents the patient from making a meaningful decision. However, because the effects of these illnesses are not uniform, it would be inappropriate to make blanket statements about the degree to which subsequent actions are autonomous or non-autonomous. As such, a threshold point would need to be established, which could employ any of a number of psychiatric and neurological tests (e.g., the Mini Mental Status Exam).

Informed Consent (or Refusal)

The standard protocol for medical intervention involves securing the informed consent of the patient. While the standards of this vary from state to state (e.g., whether the 'batting average' – the clinicians success rate with the suggested treatment – is required disclosure), there is enough commonality to require that the patient be provided with information concerning the nature and purpose of the intervention, alternative interventions (including non-intervention) and their outcomes, risks, probable outcomes of the intervention proposed, etc. This information should be presented in normal language, and should not require the patient to have extraordinary

education to understand it. State standards of informed consent could suffice for threshold points (and due to variance, this criterion exists along a continuum).

Psychiatric Criteria of Autonomy

There is only one principle psychiatric criterion of autonomy: the minimization of any psychiatric comorbidity (which I will refer to as psychiatric minimization).

Psychiatric Minimization

Given the documented underdiagnosis of depression and other depressive disorders in common medical illnesses, given the effect of depression on morbidity and mortality, and given the influence depressive disorders can exert on a patient's cognitive process, it is important to identify and account for any psychiatric comorbidities, and to attempt to minimize their effect on the patient's thought process. This may employ a trial period on an anti-depressant or mood stabilizing medication, cognitive therapy or another talk-based intervention, etc., in an effort to isolate and control thought processes stemming from a depressive disorder instead of the patient's own expressed values. This criterion exists along a continuum, as the severity of depressive disorders varies. This criterion is linked with the psychosocial criterion of authenticity.

Psychosocial Criteria of Autonomy

Psychosocial criteria of autonomy refer to the relational individual – i.e., it recognizes that the individual exists as part of a network of relationships which can exert influences – as well as referring to the narrative individual – i.e., the individual as she exists over time. There are two essential psychosocial criteria: the minimization of external coercion (which I refer to as coercive minimization) and the ownership and congruence of the individual's choices

(authenticity). Both of these criteria are based on continua – recognizing that coercion and authenticity are not all-or-none principles.

Coercive Minimization

Moral agents do not exist in a vacuum – even the choice to forgo medical treatment involves at least two people (physician and patient). As such, it makes no sense to fiat a model of radical individualism, as there is significant empirical refutation of this idea. The choices that we make in life affect other individuals in a variety of ways, some strongly and others weakly. This is not unidirectional, however – the relationships in which we engage, personal and professional, influence how we approach problems and decisions. Some relationships can exert significant influence – our motives can shift from egoistic to altruistic, focusing more on how a decision affects someone else than how it affects ourselves. Further, our decisions can be manipulated by others, through bad information and deception, emotional appeals and threats, etc. Most systems of medical ethics reject such manipulations as fundamentally undermining autonomy, a position advocated here as well. This is not to attempt to argue for radical individualism, as this seems to be untenable. However, it does seem plausible that a proper accounting of personal autonomy should attempt to minimize the coercion applied to any individual – it is unlikely that *all* forms of coercion can be accounted for and prevented, but in a decision as serious as the choice to forgo medical treatment – a terminal decision – it seems clear that one would seek to minimize any *undue* influence.

Authenticity

The authenticity criterion is complicated – on the one hand, it is intuitively reasonable to desire for decisions to reflect the values and choices an individual has taken to be her own; on the other hand, humans have the capacity to change, and that inherent plasticity makes it difficult

to insist that the individual act in accordance with the same principles at every point in his or her life (e.g., changing faiths from Roman Catholicism to agnosticism, or vice versa). A compromise position would seem to have individuals explore their contemporaneous values, in light of the other cognitive criteria, and in a dialogic process, in an effort to establish which principles should be considered authentic. The individual's decision could then be examined in light of the congruence between contemporaneous, reflected values and the decision made, with incongruence suggestive of compromised autonomy.

The autonomy model proposed above is no doubt open to criticism, as some claims (e.g., authenticity) have been controversial in the literature. However, they are reasonable criteria, when examined in light of the homuncular and cognitive models of autonomy discussed earlier – there is a compelling reason for each element, and the absence of any of them raises fundamental questions as to the autonomy of the action in question. However, as most of these criteria exist on a continuum, it may be unclear as to how this can be used clinically. This is the purpose of the last chapter, which will use a case-based approach to provide a continuum of patients, ranging from compromised to uncompromised autonomy.

CHAPTER FIVE: FACES OF CHOICE AND VOLITION – A CASE METRIC APPROACH TO ASSESSING DECISION-MAKING CAPACITY

At this point, it is useful to consider and briefly review the cognitive arguments made over the previous chapters. The recurring concern is that the choice to forgo medical treatment is subject to a variety of influences at a variety of levels. The patient is susceptible to influences from the cellular to the psychosocial level – and each of these influences can produce automatic, backstage cognition upon which conscious experience is founded. This necessitates the use of metrics sensitive to the complex cognitive causal phenomena of which patients may be unaware or to which they may be inattentive. Further, the behaviors manifested as a result of a depressive disorder may be masked by their illnesses' symptomatology. To overcome this diagnostic barrier, it is necessary both to raise the awareness that reactive depression affects morbidity and mortality, as well as employing a metric capable of discerning comorbid depression in common medical illnesses. There are three tools which immediately spring to the forefront of relevancy to the current argument: the Automatic Thoughts Questionnaire, the Dysfunctional Attitudes Scale, and the Hospital Anxiety and Depression Scale. Each of these scales will be discussed in turn and contact information pertaining to all of the scales discussed can be found in the Appendix.

The Automatic Thoughts Questionnaire

The Automatic Thoughts Questionnaire (ATQ), like the Beck Depression Inventory below, was derived from Aaron Beck's cognitive model of depression. Hollon and Kendall found a deficit in cognitive and cognitive-behavioral therapies and treatment – they noted that there were no "suitable specific measures of cognitions associated with depression."[1] In

[1] Steven D. Hollon and Philip C. Kendall, "Cognitive Self-Statements in Depression: Development of an Automatic Thoughts Questionnaire," *Cognitive Therapy and Research* 4, no. 4 (1980): 384.

response to this shortcoming, they developed a 30-item self-statement metric highly sensitive to differences in the cognitions of depressed and nondepressed criterion groups in men and women.[2] The patient is asked to rate the frequency with which a particular sample thought has occurred to them over the previous week, e.g., how frequently a patient has thought to herself "I'm a loser" or "Nothing ever works out for me." Dobson and Breitner found the ATQ to be very reliable and sensitive, and declared that "[if] there were no other considerations, the ATQ would be the instrument of choice for assessing cognitions in depression."[3] Harrell and Ryon describe similar results in the literature, noting significant specificity for depressive cognitions, significant correlation with the Beck Depression Inventory, and adequate concurrent validity;[4] they did, however, note that there are questions about the theoretical basis of the questionnaire – they suggest the possibility that the ATQ and BDI may simply share a tendency to access an aberrant cognitive response set instead of actually assessing it.[5] More recently, Glass and Amkoff have reiterated the wealth of supporting evidence demonstrating the ATQ's concurrent validity, specificity, and sensitivity.[6] Further, Netemeyer, et al, have demonstrated that shorter versions of the ATQ can be employed which retain high levels of reliability and nomological validity.[7]

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[2] Hollon and Kendall, "Cognitive Self-Statements in Depression: Development of an Automatic Thoughts Questionnaire," 390; Torbjorn Ohrt and Lars-Hakan Thorell, "Ratings of Cognitive Distortion in Major Depression: Changes During Treatment and Prediction of Outcome," *Nordic Journal of Psychiatry* 52, no. 3 (1998): 240; Richard G. Netemeyer, Donald A. Williamson, and Scot Burton, "Psychometric Properties of Shortened Versions of the Automatic Thoughts Questionnaire," *Educational and Psychological Measurement* 62, no. 1 (2002): 126.

[3] Keith Dobson and Hans J. Breiter, "Cognitive Assessment of Depression: Reliability and Validity of Three Measures," *Journal of Abnormal Psychology* 92, no. 1 (1983): 108.

[4] Thomas H. Harrell and Nancy B. Ryon, "Cognitive-Behavioral Assessment of Depression: Clinical Validation of the Automatic Thoughts Questionnaire," *Journal of Counseling and Clinical Psychology* 51, no. 5 (1983): 724.

[5] Harrell and Ryon, "Cognitive-Behavioral Assessment of Depression: Clinical Validation of the Automatic Thoughts Questionnaire," 733.

[6] Carol R. Glass and Diane B. Arnkoff, "Questionnaire Methods of Cognitive Self-Statement Assessment," *Journal of Counsulting and Clinical Psychology* 65, no. 6 (1997): 917.

[7] Netemeyer, Williamson, and Burton, "Psychometric Properties of Shortened Versions of the Automatic Thoughts Questionnaire," 125.

The Dysfunctional Attitudes Scale

Like the Automatic Thoughts Questionnaire, the Dysfunctional Attitude Scale (DAS) was derived from Beck's cognitive theory of depression; it is designed to measure vulnerability to depressive disorders outside of an actual depressed state.[8] As Beck's theory is predicated on cognitive schemata – stable and persistent cognitive states – the DAS is a measure of beliefs indicating predispositions to depression.[9] These predispositions are triggered by personally meaningful events – e.g., illness, loss, etc. DAS scores have been found to be associated with poor responses to cognitive and pharmacological interventions.[10] The DAS is a rather long scale – it was originally a 100-item measure, which contrasted significantly with many other metrics. Later revisions scaled it down to 66-items, then to a 40-item questionnaire.[11] Scores range from 40 to 280, with higher scores indicating dysfunction. Several studies have noted that the DAS has significant test-retest reliability,[12] and that it is recommended for use in the general adult population.[13] Some studies, however, have suggested that the DAS is not as powerful a metric of depressive cognitions as the ATQ.[14] More recent research, however, has suggested that the DAS is in fact a reliable and valid metric of depressive schemata,[15] and that the DAS and ATQ may be

[8] Gary P. Brown, et al., "Dimensions of Dysfunctional Attitudes as Vulnerabilities to Depressive Symptoms," *Journal of Abnormal Psychology* 104, no. 3 (1995): 431.
[9] J.M. Oliver and Elayne P. Baumgart, "The Dysfunctional Attitude Scale: Psychometric Properties and Relation to Depression in an Unselected Adult Population," *Cognitive Therapy and Research* 9, no. 2 (1985): 162.
[10] Ohrt and Thorell, "Ratings of Cognitive Distortion in Major Depression: Changes During Treatment and Prediction of Outcome," 239.
[11] Sharon K. Calhoon, "Confirmatory Factor Analysis of the Dysfunctional Attitude Scale in a Student Sample," *Cognitive Therapy and Research* 20, no. 1 (1996): 81-91; Ohrt and Thorell, "Ratings of Cognitive Distortion in Major Depression: Changes During Treatment and Prediction of Outcome."
[12] Test-retest reliability is a measure of how consistent the results of a given research tool are; specifically, it refers to whether the same test will yield similar results when the test is repeated.
[13] Oliver and Baumgart, "The Dysfunctional Attitude Scale: Psychometric Properties and Relation to Depression in an Unselected Adult Population," 165; Ohrt and Thorell, "Ratings of Cognitive Distortion in Major Depression: Changes During Treatment and Prediction of Outcome," 239.
[14] Dobson and Breiter, "Cognitive Assessment of Depression: Reliability and Validity of Three Measures," 108.
[15] Linda D. Nelson, Stephen L. Stern, and Dominic V. Cicchetti, "The Dysfunctional Attitude Scale: How Well Can It Measure Depressive Thinking?" *Journal of Psychopathology and Behavioral Assessment* 14, no. 3 (1992): 222.

sensitive to different aspects of depressive disorders and cognitive distortions.[16] Brown, et al, found further support for Beck's proposed diathesis-stress model of depression and the utility in the DAS in screening for said vulnerabilities.[17] As such, it seems plausible that the DAS may make for an appropriate screening tool for vulnerability to depression in medical inpatients.

The Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale (HADS) was designed by Zigmond and Snaith as a means of employing the anhedonic state, the central characteristic of depressive psychopathology,[18] to screen patients for clinically significant anxiety and depression.[19] It is a 14-item self-report scale, with seven questions keyed to depression and seven questions keyed to anxiety. Each subscale has a maximum score of 21, with scores of 11 or higher on either subscale indicating the probable presence of the corresponding psychopathology,[20] although some have found lower cut-off scores to have a more optimal balance between sensitivity and specificity.[21] By separating the emotional from the somatic aspects of depression, they sought to minimize the number of false positives that would plague metrics which relied on the endorsement of both physical and psychological criteria. Studies began to demonstrate the utility of the HADS in screening – for instance, in comparison to other scales like the Beck Depression Inventory and the Hamilton Depression Rating Scale, Kenn, et al, found that the HADS quite

[16] Torbjorn Ohrt, Ingemar Sjodin, and Lars-Hakan Thorell, "Cognitive Distortions in Panic Disorder and Major Depression: Specificity for Depressed Mood," *Nordic Journal of Psychiatry* 53, no. 6 (1999): 463.
[17] Brown, et al., "Dimensions of Dysfunctional Attitudes as Vulnerabilities to Depressive Symptoms," p. 434-5.
[18] Some have challenged this characterization, however. For instance, see David A. Clark, Allan Cook, and Dean Snow, "Depressive Symptom Differences in Hospitalized, Medically Ill, Depressed Psychiatric Inpatients and Nonmedical Controls," *Journal of Abnormal Psychology* 107, no. 1 (1998): 45.
[19] A.S. Zigmond and R.P. Snaith, "The Hospital Anxiety and Depression Scale," *Acta Psychiatrica Scandanavica* 67 (1983): 364.
[20] G. Johnson, et al., "Screening Instruments for Depression and Anxiety Following Stroke: Experience in the Perth Community Stroke Study," *Acta Psychiatrica Scandanavica* 91 (1995): 253.
[21] Ingvar Bjelland, et al., "The Validity of the Hospital Anxiety and Depression Scale: An Updated Literature Review," *Journal of Psychosomatic Research* 52 (2002): 71; Bernd Löwe, et al., "Diagnosing ICD-10 Depressive Episodes: Superior Criterion Validity of the Patient Health Questionnaire," *Psychotherapy and Psychosomatics* 73 (2004): 133.

useful in screening elderly patients, who normally have significant somatic complaints. They noted that "the Hospital Anxiety and Depression Rating Scale is felt to relate well to the anhedonic state and to be influenced as little as possible by concomitant physical illness."[22] Razavi, et al, found it to be quite useful in screening cancer in-patients for a variety of psychological conditions (e.g., general psychological distress, adjustment disorders, and major depressive disorders),[23] and Hamer, et al, found it to be applicable across a range of clinical situations.[24] Koenig, et al, noted some discrepancies, however, noting that in their study the General Health Questionnaire identified depression more reliably than the HADS.[25] Johnson, et al, found similar results.[26] These findings however, have been challenged.[27] Recently, Savard, et al, found the HADS to have good sensitivity in addition to "excellent internal consistency and test-retest reliability, as well as a very good convergent validity" in their study involving screening for depression in the presence of HIV symptomatology.[28] While they note that there is some debate about the factor structure, i.e., whether a two or three subscale methodology is more accurate, the HADS has generally been found to be reliable and valid. They note that "because it is simple and brief to administer, the HADS may easily become an integral part of routine HIV care. It may thus be considered the best currently available self-report scale to assess anxiety

[22] Chris Kenn, et al., "Validation of the Hospital Anxiety and Depression Rating Scale (HADS) in an Elderly Psychiatric Population," *International Journal of Geriatric Psychiatry* 2 (1987): 191.
[23] Darius Razavi, et al., "Screening for Adjustment Disorders and Major Depressive Disorders in Cancer In-Patients," *British Journal of Psychiatry* 156 (1990): 82.
[24] D. Hamer, et al., "Using the Hospital Anxiety and Depression Scale to Screen for Psychiatric Disorders in People Presenting with Deliberate Self-Harm," *British Journal of Psychiatry* 158 (1991): 784.
[25] Harold G. Koenig, et al., "Screening for Depression in Hospitalized Elderly Medical Patients: Taking a Closer Look," *Journal of the American Geriatrics Society* 40 (1992): 334.
[26] Johnson, et al., "Screening Instruments for Depression and Anxiety Following Stroke: Experience in the Perth Community Stroke Study," 256.
[27] Martin Härter, et al., "Screening for Anxiety, Depressive and Somatoform Disorders in Rehabilitation - Validity of HADS and GHQ-12 in Patients with Musculoskeletal Disease," *Disability and Rehabilitation* 23, no. 16 (2001): 742.
[28] Josee Savard, et al., "Evaluating Anxiety and Depression in HIV-Infected Patients," *Journal of Personality Assessment* 71, no. 3 (1998): 362.

and depression in the context of HIV."[29] Further support is found in Berard, et al., who note that the HADS has been found to be reliable in oncology settings in studies in the United Kingdom, Europe, the United States, and India,[30] Johnston, et al., who note that it can be an acceptable and not unduly burdensome metric of emotional distress for a variety of physically compromised patients,[31] and Herroro, et al., who note that the scale demonstrates reliability and sensitivity when translated out of English.[32] The HADS can also be employed as part of a battery of screening tools – for example, Fossa and Dahl suggest that the HADS be used as a complementary depression-specific tool when evaluating other aspects of cancer-patients' psychological state.[33] Some concerns remain about the scale, however. McCue, et al., raise a criticism of the HADS, suggesting that the two-factor/three-factor debate raises a fundamental question about the validity of the scale in chronic fatigue syndrome patients.[34] As McCue, et al.'s concern is limited in scope to a small subset of the overall patient base, however, it seems as if the majority of the evidence favors the HADS, and suggests it to be quick to administer, valid and reliable in construct assessment, and quite useful in screening a variety of patients.

Other Common Scales

The above discussion is not to suggest that it is inappropriate to use other scales – there is empirical support for a number of other metrics, and several of the more prominent are discussed below. The concern, however, is not simply diagnosing the presence of a depressive disorder,

[29] Savard, et al., "Evaluating Anxiety and Depression in HIV-Infected Patients," 364.
[30] R.M.F. Berard, F. Boermeester, and G. Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management," *Psycho-Oncology* 7 (1998): 113.
[31] Marie Johnston, Beth Pollard, and Peter Hennessey, "Construct Validation of the Hospital Anxiety and Depression Scale with Clinical Populations," *Journal of Psychosomatic Research* 48 (2000): 583.
[32] M.J. Herrero, et al., "A Validation Study of the Hospital Anxiety and Depression Scale (HADS) in a Spanish Population," *General Hospital Psychiatry* 25 (2003): 282.
[33] S.D. Fossa and A.A. Dahl, "Short Form 36 and Hospital Anxiety and Depression Scale: A Comparison Based on Patients with Testicular Cancer," *Journal of Psychosomatic Research* 52 (2002): 86.
[34] P. McCue, et al., "An Investigation Into the Psychometric Properties of the Hospital Anxiety and Depression Scale in Individuals with Chronic Fatigue Syndrome," *Psychology, Health & Medicine* 8, no. 4 (2003): 425-39, p. 436-7.

but more specifically, if one is present, identifying what effect it has on cognition and decision-making. These criteria seem to be best met with tools discussed above. However, successful screening and diagnosis are possible using other tools. Three of the most common are summarized here: the Beck Depression Inventory, the Hamilton Depression Rating Scale, and the Geriatric Depression Scale. Each will be addressed in turn.

The Beck Depression Inventory

The Beck Depression Inventory (BDI) is directly derived from the theories and therapeutic modalities of Aaron Beck's cognitive-behavioral model of depression.[35] The BDI itself, presently in its second iteration, is a multiple-item, multiple choice inventory of symptoms of depression (the first version conforms to the depressive symptomatology of the DSM-III, while the more recent version utilizes the criteria of the DSM-IV). The BDI is a 21-item self-report inventory scored on a scale from 0 to 48 with higher scores indicating more severe depressive symptoms; it has been repeatedly demonstrated to be a reliable and valid screening tool for depression in a variety of patient demographics and treatment settings.[36] Some studies have recommended the BDI be included as part of a battery of screening tools, including a full psychiatric interview.[37] Norris, et al., suggest caution in the use of the BDI in assessments of

[35] C.K.W. Schotte, et al., "Construct Validity of the Beck Depression Inventory in a Depressive Population," *Journal of Affective Disorders* 46 (1997): 115; Patricia J. Moran and David C. Mohr, "The Validity of Beck Depression Inventory and Hamilton Rating Scale for Depression Items in the Assessment of Depression Among Patients with Multiple Sclerosis," *Journal of Behavioral Medicine* 28, no. 1 (February 2005): 35.

[36] Jack T. Norris, et al., "Assessment of Depression in Geriatric Medical Outpatients: The Validity of Two Screening Measures," *Journal of the American Geriatrics Society* 35 (1987): 991; Koenig, et al., "Screening for Depression in Hospitalized Elderly Medical Patients: Taking a Closer Look," 324; Schotte, et al., "Construct Validity of the Beck Depression Inventory in a Depressive Population," 123; Berard, Boermeester, and Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management," 113; James C. Cole, et al., "Multimethod Validation of the Beck Depression Inventory-II and Grossman-Cole Depression Inventory with an Inpatient Sample," *Psychological Reports* 93 (2003): 1125.

[37] Berard, Boermeester, and Viljoen, "Depressive Disorders in an Out-Patient Oncology Setting: Prevalence, Assessment, and Management," 117; Lefteris Lykouras, et al., "Beck Depression Inventory in the Detection of Depression Among Neurological Inpatients," *Psychopathology* 31 (1998): 219.

geriatric patients, however (see below), and Kenn, et al., echo this concern.[38] Schotte, et al., found conflicting evidence; they note that while the BDI is a valid measure, there are some psychometric weaknesses in it and other self-report measures.[39] Richter, et al., suggest that the BDI is not appropriately used in frequent testing, but ought instead to be used to assess change over a larger time interval (e.g., several weeks).[40] The heart of their concern is that they found repeated testing to influence the BDI score – the test became a reaction to itself, instead of a reaction to depression. Savard, et al., raised other concerns, specifically the vulnerability of the BDI to artificial inflation due to the inclusion of somatic symptoms. They noted that severely ill AIDS patients endorsed a significant number of somatic items, which may have inappropriately elevated their scores.[41] Clark, et al., found similar results for other patient populations.[42] Despite the concerns about the inclusion of somatic symptoms, Viinamaki, et al., note that the metric has still been useful in screening depression in diabetics, Parkinson's patients, chronic pain patients, and multiple sclerosis patients, although the screening cut-off scores have varied.[43] Further support for the BDI is found in Vittengl, et al., who found the BDI to be an accurate assessing tool for changes in depressive symptoms over time,[44] Furlanetto, et al., who found that even

[38] Kenn, et al., "Validation of the Hospital Anxiety and Depression Rating Scale (HADS) in an Elderly Psychiatric Population," 191.
[39] Schotte, et al., "Construct Validity of the Beck Depression Inventory in a Depressive Population," 123.
[40] Paul Richter, et al., "Measuring Treatment Outcome by the Beck Depression Inventory," *Psychopathology* 30 (1997): 234-40, p. 239-40.
[41] Savard, et al., "Evaluating Anxiety and Depression in HIV-Infected Patients," 363.
[42] Clark, Cook, and Snow, "Depressive Symptom Differences in Hospitalized, Medically Ill, Depressed Psychiatric Inpatients and Nonmedical Controls," 45.
[43] Heimo Viinamaki, et al., "Is the Beck Depression Inventory Suitable for Screening Major Depression in Different Phases of the Disease?" *Nordic Journal of Psychiatry* 58, no. 1 (2004): 51.
[44] Jeffrey R. Vittengl, et al., "Multiple Measures, Methods, and Moments: A Factor-Analytic Investigation of Change in Depressive Symptoms During Acute Phase Cognitive Therapy for Depression," *Psychological Medicine* 35 (2005): 693-704, p. 700-1.

shorter versions of the BDI are sensitive screening measures in medical inpatients,[45] and findings that the BDI is a reliable screening tool across both demographic and socioeconomic status.[46]

The Hamilton Depression Rating Scale

Moran and Mohr note that the Hamilton Depression Rating Scale (HRDS) is "one [of] the most frequently-used and well-validated clinician-rated measures of depression severity."[47] The HRDS dates back to 1960 and was designed to be used with patients who have already been diagnosed with a depressive disorder.[48] As such, its use as a primary screening tool (i.e., in examining a patient to determine whether he or she is depressed) may not be appropriate, but it may certainly be an indicator of the severity of the depressive disorder. The scale contains 17 items which vary in answer weight – some answers are on a scale of increasing intensity, while other responses are weighted equally. Hamilton notes that scoring is ideally performed by two raters – the total score is the aggregate of the two; although it is possible for one person to use the scale.[49] There is some potential concern, however, in that the HRDS includes somatic symptoms as well – these can pose problems in diagnosis; as has been emphasized repeatedly, physical symptomology may be either somatic or psychiatric in origin. In light of this, Kenn, et al., recommend that clinicians employ the HADS instead, as it is does not rely on physical symptoms.[50] Benazzi notes, however, that there is strong correlation between HDRS and GAF

[45] Leticia M. Furlanetto, Mauro V. Mendlowicz, and J. Romildo Bueno, "The Validity of the Beck Depression Inventory-Short Form as a Screening and Diagnostic Instrument for Moderate and Severe Depression in Medical Inpatients," *Journal of Affective Disorders* 86 (2005): 90.
[46] Dennis P. Carmody, "Psychometric Characteristics of the Beck Depression Inventory-II with College Students of Diverse Ethnicity," *International Journal of Psychiatry in Clinical Practice* 9, no. 1 (2005): 27; Karen B. Grothe, et al., "Validation of the Beck Depression Inventory-II in a Low-Income African American Sample of Medical Outpatients," *Psychological Assessment* 17, no. 1 (2005): 113.
[47] Moran and Mohr, "The Validity of Beck Depression Inventory and Hamilton Rating Scale for Depression Items in the Assessment of Depression Among Patients with Multiple Sclerosis," 35.
[48] Max Hamilton, "A Rating Scale for Depression," *Journal of Neurology, Neurosurgery, and Psychiatry* 23 (1960): 56.
[49] Hamilton, "A Rating Scale for Depression," 57.
[50] Kenn, et al., "Validation of the Hospital Anxiety and Depression Rating Scale (HADS) in an Elderly Psychiatric Population," 191.

(Global Assessment of Function – Axis V in psychiatric diagnoses) scores in major depressive episode outpatients.[51] It should be noted, however, that Benazzi is examining the HDRS-10, a revised version of the original HDRS. Meyer, et al., recommend the inclusion of the HDRS in combination with other cognitive metrics (e.g,. the Mini Mental State Examination) in vascular headaches, noting that the sensitivity and specificity of the combined screening tools has already been demonstrated to be efficacious in longitudinal studies of cognitive decline during aging.[52] In their study of depression in multiple sclerosis, Moran and Mohr found that a majority of the content of the HDRS (12 of the 17 items) were able to distinguish accurately changes in depression severity,[53] which may make it useful in monitoring a patient's progress once a depressive disorder has been diagnosed. Vittengl, et al., had similar conclusions, noting that the HDRS was sensitive to changes in depression symptom severity over time.[54]

The Geriatric Depression Scale

The Geriatric Depression Scale (GDS) is a commonly used clinical measure for detecting depression in the elderly. Norris, et al., note that it is valid and reliable in the normal elderly and in those exhibiting psychopathology, and that validation studies demonstrate it to be superior to some other scales.[55] They note that diagnosing physicians would benefit from employing the GDS in screening elderly patients for depression, instead of simply relying on their own instincts; they suggest that while the Beck Depression Inventory may have higher concordance

[51] Franco Benazzi, "A 10-Item Hamilton Depression Rating Scale to Measure Major Depressive Episode Severity in Outpatients," *International Journal of Geriatric Psychiatry* 13 (1998): 571.
[52] John S. Meyer, Y.-S. Li, and John Thornby, "Validating Mini-Mental Status, Cognitive Capacity Screening and Hamilton Depression Scales Utilizing Subjects with Vascular Headaches," *International Journal of Geriatric Psychiatry* 16 (2001): 434.
[53] Moran and Mohr, "The Validity of Beck Depression Inventory and Hamilton Rating Scale for Depression Items in the Assessment of Depression Among Patients with Multiple Sclerosis," 39.
[54] Vittengl, et al., "Multiple Measures, Methods, and Moments: A Factor-Analytic Investigation of Change in Depressive Symptoms During Acute Phase Cognitive Therapy for Depression," 701.
[55] Norris, et al., "Assessment of Depression in Geriatric Medical Outpatients: The Validity of Two Screening Measures," 991.

with the DSM-III criteria for a depressive disorder, some of the attributes make it less preferable as a screening tool.[56] Studies have raised some concerns about the GDS (e.g., some studies have demonstrated lower sensitivities to depression in the GDS than in other screening tools),[57] but more recent research has reaffirmed that it has reasonably sensitivity (approximately 80%).[58]

In addition to the six scales discussed here, there are a host of other metrics available germane to the present discussion.[59] To discuss them all is well beyond the purview of this work, and would require significantly more space than can be afforded here. It should suffice to note that significant effort has been undertaken to measure and understand the complex cognitive phenomena accompanying depressive disorders, and clinicians would be remiss to ignore or underemphasize their utility in screening and diagnosis.

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[56] Norris, et al., "Assessment of Depression in Geriatric Medical Outpatients: The Validity of Two Screening Measures," 994.

[57] Koenig, et al., "Screening for Depression in Hospitalized Elderly Medical Patients: Taking a Closer Look."

[58] Johnson, et al., "Screening Instruments for Depression and Anxiety Following Stroke: Experience in the Perth Community Stroke Study," 256.

[59] See, for instance, William T. Riley and Edward W. McCraine, "The Depressive Experiences Questionnaire: Validity and Psychological Correlates in a Clinical Sample," *Journal of Personality Assessment* 54, no. 3&4 (1990): 523-33; Steven K. Huprich, et al., "The Depressive Personality Disorder Inventory: An Initial Examination of Its Psychometric Properties," *Journal of Clinical Psychology* 52, no. 2 (March 1996): 153-59; Paul Chadwick, Peter Trower, and David Dagnan, "Measuring Negative Person Evaluations: The Evaluative Beliefs Scale," *Cognitive Therapy and Research* 23, no. 5 (1999): 549-59; David J. Hellerstein, et al., "Rating Dysthymia: An Assessment of the Construct and Content Validity of the Cornell Dysthymia Rating Scale," *Journal of Affective Disorders* 71 (2002): 85-96; Madeleine J. Groom, et al., "Assessing Mood in Patients with Multiple Sclerosis," *Clinical Rehabilitation* 17 (2003): 847-57; Yvonne Birks, Alun Roebuck, and David R. Thompson, "A Validation Study of the Cardiac Depression Scale (CDS) in a UK Population," *British Journal of Health Psychology* 9 (2004): 15-24; Zeynep Hamamci and Sener Büyüköztürk, "The Interpersonal Cognitive Distortions Scale: Development and Psychometric Characteristics," *Psychological Reports* 95 (2004): 291-303; Stephen Joseph, et al., "Rapid Assessment of Well-Being: The Short Depression-Happiness Scale (SDHS)," *Psychology and Psychotherapy: Theory, Research and Practice* 77 (2004): 463-78; Löwe, et al., "Diagnosing ICD-10 Depressive Episodes: Superior Criterion Validity of the Patient Health Questionnaire."; Gregory H. Mumma, "Validation of Idiosyncratic Cognitive Schema in Cognitive Case Formulations: An Intraindividual Idiographic Approach," *Psychological Assessment* 16, no. 3 (2004): 211-30; Vicki J. Naumann and Gerard J.A. Byrne, "WHOQOL-BREF as a Measure of Quality of Life in Older Patients with Depression," *International Psychogeriatrics* 16, no. 2 (2004): 159-73; Mark Zimmerman, Thomas Sheeran, and Diane Young, "The Diagnostic Inventory for Depression: A Self-Report Scale to Diagnose DSM-IV Major Depressive Disorder," *Journal of Clinical Psychology* 60, no. 1 (2004): 87-110; Juan V. Luciano, et al., "Development and Validation of the Thought Control Ability Questionnaire," *Personality and Individual Differences* 38 (2005): 997-1008; and Maria Vuorilehto, Tarja Melartin, and Erkki Isometsä, "Depressive Disorders in Primary Care: Recurrent, Chronic, and Co-Morbid," *Psychological Medicine* 35 (2005): 673-82, among many, many others.

Having presented several diagnostic and psychometric tools, it is useful to see how they can augment the autonomy model proposed in chapter four. Because patients present idiosyncratic challenges, it is necessary to discuss a variety of patients in order to offer a compelling cross-section of the patient base. However, it is also necessary to balance this idiosyncrasy with generalizations – recurrent themes and conflicts that have appeared, regardless of the individual differences between patients. In what follows, nine patients are discussed along a continuum, in an effort to delineate key aspects of their underlying cognitive structure and processes which either support or detract from their expression of genuine autonomy.

Case Metric Format

The cases that follow have been set into a standardized format, in which salient information is presented as concisely as possible. The format covers several elements germane under the autonomy model explicated in chapter four. These cases are meant to be paradigmatic and quick references for practicing clinicians; they are amalgams of patients encountered in literature and in person.

Introduction

The introduction provides a thumbnail sketch of the patient and his or her medical crisis.

Demographics, Personal and Family History

Background information germane to discussions of support networks, salient events in the patient's life, and psychological motivations are discussed here. It is not a complete sketch or life history, but does delineate those events which are both personally meaningful as well as explanatory of how particular patients ended up facing the dilemma they face.

Condition and Prognosis

This section provides a brief summary of the patient's diagnosis and estimations of recovery or mortality.

Case Treatment

This section denotes the treating clinician's thought process which brings about the autonomy discussion. It denotes the steps taken, as well as any psychometric tools employed.

Test Results

This section refers to any salient findings of the psychometric tools employed. It does not contain laboratory values or other physical screenings.

Dialogic Content

This section conveys any salient information speaking to the patient's psychological state, attitude towards treatment, motivations, and areas of exploration paralleling dialogic approaches to medical decision-making.

Heuristics and Biases

This section discusses any salient heuristics and biases covered in chapter two which are evidenced or suggested by the patient's disclosures.

Authenticity

This section discusses apparent motivations – i.e., authentic values – which appear to be strongly represented in the patient's expressed belief system. This section will also spell out apparent conflicts in motivations, as well as potential means of addressing authenticity concerns in autonomy. Sometimes authenticity creates problems for the agent; there are times when authentic decisions may be self-destructive, which will pose difficulties for clinicians.

Accuracy of Self-Perception

This section denotes any salient evidence of cognitive distortion preventing the patient from appreciating relevant aspects of his or her condition.

Resolution

This section notes the outcome of the conflict in question.

Variations

This section notes possible variations on the case presentation, and other concerns which may be germane (e.g., changing age, prognosis, etc.) in the assessment of the choice to forgo medical treatment.

Case #1: Alice

The first patient under consideration is Alice, a 48-year-old Caucasian woman. Alice faces a difficult personal challenge – she has recently been diagnosed with a malignant tumor in her right breast, a condition complicated by other comorbidities. As of yet, it appears that her cancer has not metastasized. Alice is visibly distraught during interviews with her treatment team and psychiatric consult liaison. She currently faces a difficult decision – the degree of malignancy favors mastectomy over lumpectomy; alternatively she could do nothing, and risk metastasis. At present, Alice is convinced that her condition is terminal; she states that any surgery will be disfiguring, which will alienate her friends and family. She states that she doesn't believe that she will ever find love bearing the resultant physical and emotional scars so frequently associated with breast cancer. She believes that even if the cancerous breast were removed, because she didn't find the cancer immediately, it is likely to spread – she believes that intervention at this point is futile, a case of too little, too late. She has stated that she does not

wish to undergo surgery, chemotherapy, or radiation. She states that she "wants to live out my days in peace as a whole woman," regardless of the consequences.

Demographics, Personal and Family History

Alice is a college-educated administrative assistant at a mid-sized law firm. She is divorced, and is presently out of a failed relationship. She is moderately overweight but maintains an active lifestyle, exercising two to three times per week. She is a middle class mother of three; her children and ex-husband live out of state.

Alice's life has been difficult at times. Following the break-up of her marriage due to her husband's infidelity and her children moving away for work and college, she began drinking as a coping mechanism. She returned to dating, but found herself repeatedly in relationships that were destructive, emotionally and physiologically – she contracted hepatitis C several years prior to her current admission, which was exacerbated by her alcohol abuse. Upon receiving the diagnosis, Alice set her life back on track. She became a regular figure at Alcoholics Anonymous meetings, and developed a significant support network of sponsors and friends. She also began a dietary and exercise regimen in an effort to avoid other health complications, and has maintained her regimen since her diagnosis.

Although Alice is health-conscious, she did not perform regular self-exams for lumps in her breasts. She happened to notice an irregularity in the shower a few weeks prior, but initially attributed it to stress, after discussing 'stress knots' with her colleagues at work. At that time, her firm was nearing the end of a significant trial – most of the staff were required to up their work schedules by at least an additional 10 hours per week, so she assumed that it was trial-related. When the 'stress knot' didn't recede following the conclusion of the case, she became

more concerned, and began checking the 'stress knot' several times per day. After an additional two weeks, she consulted her primary care physician, who referred her for testing.

When she got the news, she relapsed, and spent the entirety of the previous weekend intoxicated. She was admitted to the hospital to detoxify her system on a Librium protocol. Her treatment staff approached her about medical treatment for the malignancy, and presented her with her options. Emotionally labile, Alice disclosed that her grandmother died of breast cancer, and that she has two friends who had undergone personal cancer scares, one of which resulted in a radical mastectomy, while the other was benign. She notes that her friend who underwent the mastectomy described her recovery as painful, awkward, and socially uncomfortable for her and her husband.

Condition and Prognosis

Alice currently has a stage IIB malignancy. Upon biopsy, the malignancy was determined to be 5.5cm in size, but had not reached the lymph nodes. Her treating physician has given her a five-year survival chance of 65% with treatment. Her hepatitis flared again following her relapse, but her physician believes it should respond to interferon therapy.

Case Treatment

An ethics consult has yet to be called; being aware of the possibility of comorbid depression, the treating clinician contacted the psychiatric consult liaison. The consult liaison noted Alice's anhedonia, an understandable reaction to an acute hepatitis flare and cancer diagnosis. Suspicious that there might be more to the case, the consult liaison asked for formal testing by the hospital's clinical psychologist, who administered the Hospital Anxiety and Depression Scale and the Automatic Thoughts Questionnaire.

Test Results

The HADS indicated that Alice likely carried a comorbid depressive disorder. She endorsed a number of items indicating anhedonia and feelings of worthlessness and helplessness. She could not complete the ATQ, as she burst into tears when she began reading the self-statements, saying that she was thinking "all of them, all of the time."

Dialogic Content

Alice was willing to talk with the clinical psychologist – in the course of her interviews, she disclosed significant feelings of being controlled – the negative events in her life made her feel "helpless against fate." She stated that even when she tried to do right, she couldn't win – just when she thought her life was back on track, something else went wrong. She noted that she frequently thought of her friend who underwent the mastectomy. The pain of treatment weighed especially heavy on her – she didn't want to suffer, especially if she wasn't sure that it would do any good. When she heard that she had a 65% chance of living another 5 years, she stated that she only heard that she had a 35% chance of dying, even if she underwent treatment. She stated that she normally was a happy person, and that before her diagnosis, she thought that she would have a happier life. Now, she stated, "life doesn't mean much to me anymore, and it probably never will."

Heuristics and Biases

Several key issues are apparent in this case, all of which should give pause in the decision to support Alice's choice to forego medical treatment. The first issue are the test results – both of which indicate that she is presently depressed, which as noted in previous chapters, can exert significant influence over cognition. The second concern is elements disclosed during the dialogue with the clinical psychologist. Alice notes three key heuristics which ought to raise

flags concerning potentially compromised autonomy. The first is the disclosure of the impact of her friend's bout with cancer on her present cognition. Alice is demonstrating the availability heuristic – the information that came to her mind most readily, and as a result, the information which exerted significant influence on her decision-making process was that cancer recovery was a painful process. By focusing on this information, Alice believes, rightly or wrongly, that her own recovery from cancer must be similarly painful and socially awkward. Alice also demonstrates the durability bias by believing that her emotional and physical state will continue unabated into the foreseeable future. In employing both availability and affective forecasting, Alice is ignoring key elements that can affect her recovery, specifically her support network of friends and children. While Alice possesses insight into her life that is unavailable to others, her proximity also blinds her to salient aspects of her situation. While she faces major hurdles, she has major support in overcoming them. A third heuristic bias exhibited is that of anchoring – Alice stated that she only heard "35% chance of death," a clear instance of latching on to one particular outcome, and ignoring the 65% chance of five-year survivability. Her endorsement of most of the items on the ATQ offers further indication that she is experiencing 'inside interference' – uncontrollable negative thoughts.

Authenticity

In addition to the heuristics and biases discussed above, Alice gives key indications as to her value structure and the congruence of her actions with it. Two specific issues deserve attention. First, Alice is concerned about pain and social awkwardness, a legitimate concern. Self-image exercises considerable influence on self-worth and goal-structuring – it is very difficult to attain goals when one believes one cannot. Even when one is successful at achieving one's goals, depression can cause one to attribute the success to outside elements or actors. A

second clue is that Alice notes that she normally isn't so morbid – she described herself as a happy person with goals for the future. Deciding to forgo medical treatment and passively accept death seems radically incongruent with her stated values. Both of these disclosures suggest that her cognition runs counter to her authentic self – the actions, it would seem, stem from her malignancy, not her identity.

Accuracy of Self-Perception

Alice does not believe that her cognition is compromised. Instead, she states that she "just wants to be left alone" and that "I am seeing things clearly for the first time." Both of these suggestions, however, are questionable. Isolation itself is a sign of depression, and it can further increase the malignancy by allowing the sufferer to ruminate upon their current condition. Isolation prevents recovery – as noted earlier, while we possess insights into our selves that are inaccessible to others, we can also become blinded to our state. We experience a kind of agnosognosia – we are unaware that we are unaware of our dysfunctional cognition. This agnosognosia is evidenced in her statement on her clarity – she is unaware that the stressors in her life have changed her perception from her authentic self, and have altered the cognitive processes upon which she based her choice to forgo treatment.

Resolution

A variety of red flags were raised by Alice's disclosures, each of which would suggest a higher evidentiary standard for endorsing her decision to forgo treatment. The clinical psychologist, consult liaison, and treating clinician approached Alice with their concerns, and suggested a course of anti-depressants and brief cognitive-behavioral therapy sessions with the clinical psychologist over a two-week period, at which point the decision to refuse treatment would be revisited. This course of treatment would be initiated during Alice's in-patient stay,

and would be maintained on an out-patient basis following her discharge. Alice was open to the suggestion, and used the therapy sessions to discuss her fears, social and physical, as well as exploring her support network and developing coping skills for automatic thoughts. When the three clinicians met with Alice again, she consented to the mastectomy and adjuvant therapy.

Variations

Alice's case offers challenges in itself, but there are other potential complicating factors. She is presently a middle-aged woman; would her case be considered differently if she were older? In light of the discussion presented in the previous chapters, there would likely be factors affecting the analysis. Age carries with it complicating factors: frequently, older adults have more somatic complaints than younger adults. An absence of chronic medical conditions seems to be the exception, rather than the rule. As such, her assessment of the quality of her life may change as a result. Further, as she gets older, her body may no longer have the same physical resources as it did when she was younger, and as such, physically taxing treatments and recovery periods may become very difficult to endure. Her prognosis remains very favorable, but she may quite reasonably determine that the treatment would be too burdensome for her. If this assessment were shown to be consistent with her values (which may also change from her initial case study presentation, since she is now older and may have had change-inducing experiences), there may be significant reason to endorse her decision to forgo burdensome, and hence morally extraordinary, treatment.[60]

Similar difficulties arise were Alice to elect a different treatment option. For instance, if Alice were to maintain her same value structure and life experience, but elect prophylactic bilateral mastectomy. This decision should raise concerns for a variety of reasons. Although the

[60] Morally ordinary treatments are distinguished in ethics from morally extraordinary treatments. Morally ordinary treatments tend to be seen as obligatory, while morally extraordinary treatments tend to be seen as non-obligatory.

procedure would certainly remove the lump in question and would prevent a recurrence of that particular form of cancer, it still fundamentally conflicts with her desire to live "as a whole woman." Unilateral mastectomies tend to produce significant long-term psychological sequelae, and many women require counseling to restore a sense of self and self-confidence. Bilateral mastectomy carries similar consequences, and as such, represents a decision that ought not to be treated lightly or chosen without thorough introspection and support. If Alice were to elect this opposite extreme, especially in light of the immediate conflict with her authentic values, clinicians should suspect compromised autonomy.

A third complicating factor is her likelihood of five-year survivability. In the case study, Alice was given a 65% chance of living another five years; if this percentage were changed, then it would be reasonable to expect a change in Alice's valuation of her condition and potentially change her decision. If the percentage were increased (from roughly 7 in 10 to 9 in 10, for instance), we might expect her to be more optimistic about her outcome. If it were lowered, however, to 40%, the decision to forgo treatment may seem more reasonable. The 25% shift between survivability percentages can have significant impact, especially as one moves towards the lower range of percentages (e.g., a 95% to 70% shift would likely have less of a decisional impact than a shift of 50% to 25% or 40% to 15%). If the likelihood of five-year survivability drops significantly, her decision to forgo medical treatment may seem more reasonable and objectively accurate.

Case #2: Bill

The second case under consideration is Bill, a 57-year-old African-American with colon cancer and coronary artery disease (CAD). Like Alice, Bill's cancer has not yet metastasized, and, in fact, was detected upon colonoscopy quite early. Bill appears upset at times during

interviews, and has a hard time maintaining eye contact. He has a tendency to speak to the floor, instead of the person asking him questions. Bill is less concerned with the colon cancer than he is with his CAD – he is not happy with the dietary changes his physician is recommending, and he argues that at this point in his life, he has earned the right to eat badly. He argues that if he has cancer, it "shouldn't matter what I eat, since I am going to die anyway." Because of his predicament, Bill is refusing to continue his CAD treatment, and is refusing any sort of intervention for his colon cancer. He has stated that since "the cards are stacking up against me, I might as well enjoy myself." After all, he argues, what is the point of living if you can't enjoy it?

Demographics, Personal and Family History

Bill is a bus driver, and has been for 30 years. Prior to his hospitalization for chest pain, he had been considering retiring and moving across town to be closer to his family (his wife had died two years previously in an auto accident). He has two children, a lawyer and an architect, who visit him regularly. Bill has a fairly sedentary life – when he is not working, he enjoys sitting on his porch and barbecuing with his neighbors.

Bill's life has been interesting. He graduated from high school with honors, but had no college aspirations. He was drafted, and spent a rotation in Vietnam in the infantry. Wounded twice in combat, he was a decorated soldier before returning home. He wanted to stay in his hometown, and took on a variety of jobs in the years following his discharge. During this time he met Janine, whom he fell in love with and married. As a budding photographer, her career required her to move away from Bill's hometown, and in his devotion, he followed her. He found a job as a bus driver, and settled into a comfortable married life. Bill and Janine had two sons, of whom they were very proud; the entire family was shaken by Janine's death. Bill's

experience in Vietnam taught him to enjoy life while he could – because he was unsure what each day would bring, he was determined to enjoy it. He maintained this philosophy when he returned home, but didn't maintain the same activity level. As a result, his waistline grew as his hair receded, bringing with it coronary artery disease.

In the course of a routine physical, Bill disclosed to his PCP that he occasionally had hemo-positive stool, which he attributed to hemorrhoids, the consequence of sitting and driving for hours on end. When his doctor found no hemorrhoids, he recommended colonoscopy as a routine precaution. The colonoscopy discovered polyps, which were later determined to be malignant. Bill's father had died from lung cancer, after smoking unfiltered cigarettes for twenty years. It was later determined that in addition to the pleural malignancies, his father's heart was atherosclerotic; in fact, if he had not died from cancer, it was quite likely that a massive heart attack would have killed him within a year. When Bill heard that he himself had both colon cancer and CAD, he figured his time was up, and that he was going to die just like his father. "Why fight it?" he asked.

Bill's chest began to bother him several weeks before his admission. He stated that it felt as if his left chest and shoulder were cramping, and he would get occasional tingling sensations in his hands and feet. He didn't think much about it in the first few days; he thought he "must have been sleeping on his side a little funny." After a week, he began to worry, but thought that as he had a physical coming up soon, he could just wait it out. Doctors were expensive, he reasoned, so it would be better to kill two birds with one stone. He noticed the blood in his stool the following week, ten days before his annual physical.

Condition and Prognosis

Bill was diagnosed with Stage A (T1N0M0) colorectal cancer and 40% occlusion of the coronary arteries. His PCP and consulting oncologist gave him better than 90% five-year survivability, presuming that he simultaneously reduced his fat intake and engaged in moderate exercise.

Case Treatment

Bill's reluctance to treat the eminently correctable and survivable morbidities caused his treating physician significant concern. The clinician noted that it seemed as if Bill was "giving up far too easily," especially in light of the moderate nature of the illness and his strong social support network. Like Alice's clinician, he contacted the consult liaison psychiatrist, who noted the poor eye contact, apathy and resignation when conversing with Bill. He called for testing by the clinical psychologist, who administered the Hospital Anxiety and Depression Scale and the Dysfunctional Attitudes Scale.

Test Results

Bill scored highly on both the anxiety and depression subscales of the HADS. While Bill did not manifest many outward signs of anxiety, he disclosed that his father was on his mind a lot, in light of the similarities of their diagnoses. The Dysfunctional Attitude Scale similarly noted the likelihood of a depressive comorbidity.

Dialogic Content

Although hesitant initially, Bill was willing to talk to the clinical psychologist following the administration of the diagnostic instruments. When asked about his disclosure of his preoccupation with his father, Bill described how he couldn't help but see the parallels between their two cases. They both had heart problems and they both had cancer, so why shouldn't he expect the same kind of outcome? Why shouldn't he expect the cancer to kill him, or, barring

that, the massive heart attack? "With any luck," he added, "it'll get me in my sleep." Concerning Bill's attitude towards his life, he said that he was comfortable with it, and that he liked being able to eat what he wanted, do what he wanted, and that anything which changed that routine he didn't like. "I am who I am, and I like what I like," he stated. Bill showed some insight into his heart condition, noting that it was possible that his choice of poor diet and lack of exercise were exacerbating his condition, but he said that he didn't see any way around that, because he didn't like to exercise. "Some people like to smoke; I like to eat," he added, "and there ain't nothing gonna change that. It may kill me, but I'll go out happy."

Heuristics and Biases

As with Alice, there are several areas of concern. First, Bill is demonstrating the availability heuristic – he is drawing direct conclusions about his own condition based on his father's experience of cancer and heart disease. While this is reasonable to an extent, Bill has taken it beyond a reasonable level – his condition is eminently treatable, and his path is not intrinsically as damaging or delineated. Bill's prediction about the cause of his death may only be true if he does nothing to change it – it is essentially a self-fulfilling prophecy. Bill has adopted an overly fatalistic attitude, and based it upon connections which are loose, at best. Bill also demonstrates a durability bias, predicting that living a life with dietary restrictions will be too unpleasant for him. This assumption may be unfounded – he will not necessarily know how he feels about a situation until he has experienced it. As such, it seems as if there are reasons to question whether Bill's decision to refuse therapeutic interventions for his cancer ought to be endorsed.

Authenticity

Bill's case raises difficult questions about authenticity – specifically, what should we as clinicians do about authentic values which are dysfunctional or self-destructive? Our bodies contain feedback mechanisms which can build dependencies – we have built in reward mechanisms for eating fatty foods, sweet foods, smoking, abusing drugs, etc., among a litany of directly and indirectly self-destructive behaviors. If a patient's authentic self is self-destructive, it seems to present a Catch-22 situation for the clinician. Attempting to change the behavior invites accusations of paternalism, while doing nothing invites accusations of abandonment and disregard. Bill's authentic self likes to eat fatty foods and maintain a sedentary lifestyle; he wants to enjoy the time he has left. It would be both unrealistic and unreasonable to expect him to begin training for a marathon. One approach to this situation would be to stress the other elements of his life which are personally meaningful – his relationship to his surviving family. Bill is close to his sons, and he was considering moving closer to them after retiring. Clearly he wants to be near his family, to enjoy his life with them as he wants to enjoy his vices. One could explore which of these two motivations is stronger – it is not uncommon to find oneself faced with two competing drives. Choosing medical intervention and changing his diet could be less of an imposition if he views them as means of enjoying his family more; authenticity can thus be saved by exploring the complex motivating elements of his decision-making process. The choice to forgo treatment, in this case, would seem to be incongruous with his desire to be near his family.

Accuracy of Self-Perception

Bill exhibits some insight into the controllability of his situation in that he recognizes that his behavior is linked with his health. However, it seems as if Bill does not see how his long-

term and short-term goals conflict. His devotion to his family is confounded by his desire for short-term gratification, found in maintaining his lifestyle. He views his alternatives as all-or-none; either he can enjoy himself and live a shorter life, or he can deny himself and live a longer, miserable life. This dichotomy is as powerful as it is false; Bill is not entertaining any notions that a middle-ground is possible. Dietary changes would not necessarily require him to become an ascetic; instead, it would simply require more responsible monitoring of what he was eating. He would not have to train for the marathon, but instead could take a walk around the block – an activity he could combine with visiting his neighbors. Bill's lack of insight into his cognition and dichotomous thinking demonstrate likely influence of a depressive disorder, and as such, raise cautionary flags about his choice to forgo treatment.

Resolution

Bill initially did not agree with the recommendations of the treatment team, but was more open to discussion when his sons visited him. They offered to help him move, to eat and walk with him, providing him with a means of both improving his lifestyle and maintaining contact with his family, at which point he consented to immediate treatment for his CAD, and said he would think about treating his colon cancer. He agreed to follow-up with the clinical psychologist as an outpatient. After several weeks of talking about his father's experience of illness, in addition to strengthening his social support network and learning about warning signs of depression, he consented to treatment for his colon cancer.

Variations

Bill's case becomes more complicated when focus is shifted to his CAD by removing his cancer. At this point, the only clinically significant condition is his cardiac disease; the treatment for his condition is quite simple: dietary changes and exercise. As noted in the case a significant

concern for Bill was the perception that he was experiencing a significant amount of medical comorbidity (the CAD on top of the cancer), which he argued rationalized and justified his dietary choices. Absent that rationalization, the choice to forgo treatment based on dietary impact would seem to lose both objective weight and subjective appeal. Bill may still choose to forgo treatment based upon this rationalization, but it should at this point raise significant concern for the clinician, and would suggest that there may be compromised decision-making ability. This is not to suggest that clinicians can overturn a decision based upon objectively poor lifestyle choices; a number of reputable institutions and organizations have amassed a wealth of evidence supporting the link between smoking and multiple health problems, but it does not therefore stand to reason that a clinician can undermine a patient's choice simply because he chooses to continue to live in an unhealthy manner. While clinicians cannot control lifestyles, they can at least raise challenges to decision-making processes that are compromised by lifestyle choices.

A second complication takes Bill to the opposite extreme – in this case, his cancer has advanced, and poses a serious threat to his health. Depending upon the severity of the cancer, Bill's gastronomy may be the only enjoyable part of his life, and as such, requiring him to refrain from the one activity which brings him pleasure may genuinely constitute overly burdensome treatment. Were this the case, refusing to consent to treatment he views as overly burdensome would seem to be a reasonable response, instead of an indication of potentially compromised autonomy.[61]

[61] I admit that I am concerned about this statement, in that what is burdensome has a significant subjective element, and as such, it is quite possible for objectively questionable (even unreasonable) judgments to appear to be justified by this argument. I wish to stress that this idea exists upon a continuum between clearly reasonable and unreasonable extremes, and as such, I would examine the internal consistency of the choice with the patient's value structure (and potential value conflicts) before attempting to generate generalized rules as to what is reasonable versus unreasonable. In the event that a clinician encounters a patient with a value structure which appears at odds

A third possibility is that Bill may disagree with the treatment team's treatment recommendations. As has been noted earlier, simple disagreement with the treatment team does not in itself constitute compromised autonomy. In the case presented above, the resolution notes that Bill ultimately consents to treatment for both his CAD and his colon cancer. Quite obviously this is not the only potential outcome. The case is meant to raise potential concerns about compromised autonomy, but it does not require that he consent to both or either treatment. Patients are free to make bad mistakes; what is incumbent upon us as clinicians is to challenge them, and not simply to overrule those choices with which we disagree. Bill may have chosen to make the dietary changes but not address the colon cancer, or vice versa. Consent to one treatment does not carry with it consent for the other, and each would need to be addressed separately. It does make sense to approach them as part and parcel of the same overall treatment package, but patients may disagree, just as they may divorce consent to CPR from consent to specific follow-up interventions (e.g., any resulting intubation), or divorcing consent to exploratory surgery from consent to remove any malignancies found. Disagreement does not necessarily produce inability to make treatment decisions, but it certainly produces a greater necessity of common language and understanding between clinician and patient, as it may stem from simple miscommunication or obfuscation.

Case #3: Catherine

The third patient under consideration is Catherine, a 70-year-old Hispanic woman presenting with congestive heart failure, diabetes mellitus, and peripheral vascular disease (PVD). Prior to her admission, Catherine noted that her legs were beginning to swell severely, and that her feet, already puffy and weepy, were also discolored. Catherine has been living with

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with common sense, the necessity of dialogic interaction becomes evident – the 'method to the madness' may become evident to the clinician, and/or the source of the error may become evident to the patient.

congestive heart failure for many years, and is used to being short of breath and experiencing edema. She is concerned about the discoloration, but didn't want to make a fuss when she visited her doctor. Testing determined that while the edema could be treated by increasing her Lasix, the discoloration in her feet was being caused by occlusions in the blood vessels near her ankles, which was starving her feet of oxygen. In fact, tissue on the toes of both of her feet were necrotic; she had not noticed because she was unable to see or feel the damage due to her diabetes and poor flexibility. Catherine is unsure how she wants to proceed; her physician is recommending removing the dead tissue, but has not ruled out the possibility of amputation. Catherine has poor mobility as it is, and is sure that any intervention at this point would restrict her mobility too much, as she is convinced that the doctors want to take her feet.

Demographics, Personal and Family History

Catherine is a high-school graduate, and was the first in her family to go beyond eighth grade. Her activity level has dropped off with age and the progression of her illnesses, but she still does her own housework, cooking, and socializing with her neighbors. Her daughter lives a few miles away, and visits her frequently.

Her life has been tumultuous at times; at a young age she lost her first husband in the Korean War, leaving her with a young child and minimal income. She worked a series of jobs from receptionist to retail, always provided for her and her child, and had enough saved to send her daughter to college. Because of her experiences as a young adult, she has developed significant self-reliance and a drive for independence. She had been diabetic since childhood, and had assiduously attended to her blood sugar levels. While she has some retinopathy in addition to the PVD, she has made a conscious effort to minimize the impact of the illness on her life. She was, therefore, quite surprised to hear about the necrotic tissue on her feet.

Prior to her admission, Catherine had noticed that her feet and ankles were swelling up during the day, which she simply attributed to her frequent walks in the house. In the times when she would sit down, she would put her feet up to reduce the swelling, advice she took to heart from her gerontologist. She was able to recognize the signs of edema, and reduced her activity level as a result, allowing her daughter to do some of the cooking and cleaning for a few days. When the edema did not go down and the discoloration appeared, she became concerned and approached her gerontologist, who referred her for treatment.

Catherine maintained strong community ties, and several of her neighbors have visited her during her hospitalization. She remains feisty, and the staff occasionally reprimands her for pacing in her room instead of elevating her feet. "I want to stay active," she tells them, "it makes me feel good to be up and about and doing something." Catherine tells them further that she doubts she'll be able to be active once the doctor takes her feet – she is afraid that she will become entirely dependent on other people, and that she will lose control of her life.

Condition and Prognosis

Catherine's diabetes is currently being managed – she is quite compliant with dietary restrictions, and is faster checking her blood sugar than are the nurses treating her. Catherine's principle ailment is the necrotic tissue, which is threatening to lead to sepsis. Her treating physician has her on several antibiotics while they discuss her treatment options.

Case Treatment

The treatment physician is concerned about her apparent ambivalence regarding her outcome. She does not seem to appreciate the severity of sepsis, nor does she seem to appreciate the precariousness of her health. This seems to be a significant change from her previous state of mind, which was very health minded. While she keeps tabs on her diabetes, her physician is

concerned that this may be due simply to routine, rather than maintained concern. Recognizing that personality changes can be a hallmark of depression, he contacts the psychiatric consult liaison. The psychiatric consult notes that she does display a variety of somatic symptoms typically associated with depression, but wants to make sure that a psychopathology is present. The consult liaison contacts the clinical psychologist, who administers the Hospital Anxiety and Depression Scale, along with the Geriatric Depression Scale.

Test Results

Both the HADS and the GDS indicate borderline depressive symptoms. Although neither test indicates clear psychopathology, her scores were very close to the threshold, and as such, the clinician elects to pursue a dialogue with Catherine, in an effort to find more evidence supporting or refuting a depressive disorder.

Dialogic Content

Catherine is a little reluctant to discuss her case with the clinical psychologist, but speaks more freely after a visit from her daughter. The psychologist notes that her affect brightened considerably following the visit as well. Catherine relates to the psychologist her life and need for independence. She stresses how tough it was being a single mother, but that she raised her daughter to be self-reliant. She relates how difficult it was for her to sit around while her daughter cooked and cleaned; she confides that she would occasionally fold laundry when she was supposed to be sitting down. Her voice gets stronger as she describes the shock of being widowed and the satisfaction of being able to do well by herself when everyone else thought that she couldn't. She describes her fear of becoming dependent if amputation becomes necessary, and wonders if it might not be better for her just to enjoy the time she has left. She loves her daughter, and does not want to think that she would be a burden on her. She has known several

individuals who were less rigorous in their diabetic management, and she doesn't want to end up like them.

Heuristics and Biases

Like Alice and Bill, Catherine is exhibiting the availability heuristic – she is drawing a parallel between other individuals experience with diabetes and her own, which may not necessarily be accurate. While there is a possibility that removing the necrotic tissue may require amputation, it does not necessarily follow that this must lead to greatly restricted mobility. She further notes that she may become burdensome upon her daughter, which may also be an unfounded application of the availability heuristic. Like Bill, she is exhibiting some durability bias – she is forecasting her emotional response into the future, and ignoring possible coping mechanisms which could restore some equilibrium between dependence and autonomy. Further, Catherine has anchored, rightly or wrongly, on the word 'amputation', which carries significant meaning for her. As such, it is clear that it is influencing her cognitive processing and decision-making.

Authenticity

Clearly independence is an important part of Catherine's life – it has been a guiding principle for her since she was a young woman. Any answer to the current dilemma must include an appreciation and understanding of this basic tenet of her existence. It would seem that the best possible recourse would be a treatment that minimizes the impact on this facet of her self-understanding. Removing the dead tissue without amputation would be the clearest step in this direction. Further, discussion of the diagnosis and prognosis with her daughter would clarify any needed assistance Catherine may require, and may in fact assuage Catherine's fears of being burdensome. If a resolution is found which maximizes Catherine's independence, she may be

more amenable to therapeutic intervention. One of the biggest issues that seems to plague consultations is poor communication – ensuring that the relevant parties are on the same page regarding motivations and outcomes can go a long way to overcoming apparent boundaries.

Accuracy of Self-Perception

Catherine's self-perception is demonstrably more accurate than Alice's or Bill's, but there are still areas of concern. Catherine's fear of dependence has caused her to focus on the possibility of amputation and potential restriction, almost to the point of ignoring other possible outcomes and benefits. She has selectively maximized the likelihood of complete dependence and minimized the possibility of diminished, but significantly maintained independence – after all, there are many individuals who have undergone amputations who still maintain active lives. As such, it appears that her decision-making is unduly influenced by this attention to select details, and it is simultaneously preventing her from recognizing both her cognitive barriers as well as other prognoses.

Resolution

After talking with her treating clinician and her daughter, Catherine decided to allow the removal of the dead tissue. She insisted, however, that if further interventions such as amputation were deemed medically necessary, that she would refuse them, and her daughter voiced her understanding of her mother's wishes. Catherine stressed again her desire to live independently; her daughter replied that she would be happy to check in on her mother, but would happily do so as a guest, rather than a housekeeper.

Variations

Catherine's case can be complicated by strengthening her fears of losing her feet to amputation. In this variation, she adamantly refuses both debridement of the necrotic tissue and

amputation of her feet, but demonstrates clearly compromised autonomy. The compromise results, however not from a medical condition, but from her fears of disfigurement. This raises an issue of significant concern – her fears have a legitimate basis. This is not to say that she is automatically autonomous or non-autonomous; rather, we see that there is evidence on both sides of Grisso and Appelbaum's scale. On the one hand, Catherine was approached about the possible medical need to amputate.[62] This directly conflicts with her desire to remain active; amputation will not necessarily reduce her to a moribund state, but it will certainly impact her mobility. Even the use of prostheses would require an acclimation period. This shifts the discussion of the removal of the necrotic tissue away from morally ordinary care and towards morally extraordinary care. In doing so, it suggests that Catherine may be justified in choosing to forgo the treatment. On the other hand, there is significant analysis demonstrating the availability heuristic and the durability bias, both of which suggest sources of cognitive error. This then leads to an uncomfortable dilemma, with compelling evidence on both sides of the evaluative scale. As a means of approaching this situation, part of the original analysis still holds true – any avenue of treatment must necessarily address her concerns of independence, but it must be made clear that any source of cognitive error be identified and corrected as much as possible for the action to be autonomous; as noted above, the underlying motive for the refusal is fear. While fear can be a powerful motivator, it can also be addressed and overcome.

A second complication arises if the psychometrics did not indicate the likelihood of depression influencing cognition. This is not to say that an affective disorder is the only possible

[62] I willfully shift from 'medical necessity' to 'medical need', precisely because necessity may be too strong of a word in this case. While her *medical* best interests may be served from the removal of the necrotic tissue and potentially of her feet, that is not the only potential outcome. For instance, the amputation may be presented as medically favorable, or the intervention which produces a higher long-term gain; this does not therefore translate into the only potentially beneficial course of action. Medical best interests could also be served by removing the dead tissue without amputating the feet, or by providing palliative care and pain management, were sepsis or other secondary medical issues to arise.

compromising factor – as was argued in the last chapter, there are may requisite characteristics of an autonomous action. Rather, a lack of psychometric evidence does undermine arguments that her decision is cognitively compromised. There is still recourse in the heuristical analysis, and dialogic interaction may succeed in bringing out any sources of error. These are not guaranteed results, however, and clinicians may be forced to accept that the only objections they can raise to a decision is their own intuition or impression from interaction with their patients, which may not be enough to justify overturning an endorsement of their patient's autonomy. An absence of quantifiable data does not immediately make a compromised patient uncompromised; all it does is shift the emphasis in patient interaction from psychometrics to dialogic interaction. No psychometric enjoys perfect sensitivity or specificity; as such, clinicians must make a dialogical exploration of personal values a part of the decision-making process.

A third complication can be raised if Catherine refuses to make a choice. Many patients prefer to have decisions made for them. Several authors have noted a willful surrender of authority to treating clinicians on the part of patients; while this may make the decision-making process longer, it does not necessarily make it morally ambiguous. If Catherine were to refuse to make a choice, several avenues remain open possibilities. The refusal may stem from her interactions with the clinical staff – a friend or family member may be able to make inroads towards a decision. This friend or family member would not necessarily make the choice for her, but may make it easier for Catherine to express her wishes. The friend or family member may be a facilitator, rather than a proxy decision-maker. Alternatively, the friend or family member make explicitly take on the role of proxy, and provided that no conflicts of interest occurred or clear differences between the proxy's and patient's wishes were present, the friend or family member may be able to shoulder the burden of responsibility for Catherine. The requirements

for proxy decision-making vary from state to state, so adequate research and/or legal understanding may be required to employ this avenue of decision-making.

Case #4: David

Our fourth patient is David, a 64-year-old Asian-American male presenting with Parkinson's disease, peripheral neuropathy and congestive heart failure (CHF). David's dosage of L-dopa has steadily increased over the years as his Parkinson's has gotten worse. His therapeutic dosage is approaching toxic levels, and he is experiencing auditory and visual hallucinations with increasing frequency as a result. These auditory and visual hallucinations are causing him significant distress, compounded by his increasing difficulty moving. He spends most of his time sitting in his chair at home, "trying to decide what's real and what isn't, and trying not to feel my feet tingling." David isn't quite sure what to do with his life – he finds it difficult to move his hands, which makes it hard to enjoy the puzzles and games his grandchildren want to play. His impaired mobility makes it hard for him to stay active – before his diagnosis, he used to enjoy walking in the park and feeding the pigeons. His family visits him at least once a week, but he feels very lonely when they are not around. Recently his CHF began to act up, and when his family found him struggling to breathe on their last visit, they brought him to the ER. His treating physician has increased his Lasix and his CHF is responding moderately well to the treatment. David, however, is not sure that he wants to continue treatment – although he might breathe better, he is not sure that his life is worth continuing.

Demographics, Personal and Family History

David is a college-educated retired high school history teacher. He is twice divorced, and has children from both marriages who visit him frequently. He was athletic throughout his life, regularly bicycling and running, until he began having some difficulty coordinating his

movements. What he initially attributed to clumsiness was later diagnosed as 80% destruction of his substantia nigra, the onset of Parkinson's disease.

David always did well in school, met his first wife in college, and had two children before his thirtieth birthday. His wife and he grew apart, culminating in a divorce in their early thirties. At thirty-six, he remarried a woman five years younger than he, and had two more children before his fortieth birthday. His wife began an affair a year after the birth of their second child, which eventually spoiled the marriage. He decided that he would try living single for a while before looking again, but he never really felt the drive to become involved in a long-term relationship, the product of two failed marriages. His children harbored no resentments, and they remain closely knit, despite lingering animosity between their parents.

David's life became much more solitary after his Parkinson's diagnosis – he felt that it was a huge change in his life, and that it would prevent him from being the man he was before. He remained active as long as he could, but his lifestyle was further affected by a diagnosis of congestive heart failure – he could no longer exercise as easily as he once did. Frequent walks and free weights gave way to his recliner and porch chair. As his motor skills slid away, he found it more difficult to read, and his thick books and reading glasses gave way to audio books and the television. He isn't happy with his lifestyle, and continues to wish that he could be more active. The visits from his family restore his spirits, but he feels his illness closing in on him when they are not there.

When he woke one morning and found it hard to breathe, he was unsure what to do. On the one hand, he didn't want to leave his family – he loved them dearly and wanted to see his grandchildren grow up. On the other hand, his life was no longer what it used to be, and he was finding it harder and harder to manage to cook, clean, and take care of himself. He knows his

family is considering nursing homes – the family harbors no illusions that the Parkinson's will ultimately prevent David from caring for himself, and as such, he would need skilled care. He is ambivalent about living in a nursing home – he recognizes that his needs will increase, but he doesn't want to depend on others, or leave the house he has lived in for several decades.

Condition and Prognosis

David currently has moderately advanced Parkinson's disease, his neurologist predicts that he will be completely chair-bound within three years. His congestive heart failure is advanced, but responds to treatment; his treatment team believes that it will respond to the Lasix and are recommending that a home care nurse begin visiting him following discharge.

Case Treatment

The treating physician recognizes that David has legitimate concerns about his long-term prognosis. They have discussed the likely outcomes of his conditions, and both are aware of what he can expect within the next five years. Both are cognizant of the significant changes that have occurred in David's life, and they recognize that he has been forced to give up personally meaningful activities. David's physician is concerned that he may be conflicted about his treatment – his consideration of giving up treatment seems to conflict with his desire to be with his family. His clinician contacts the consult liaison psychiatrist, who after a formal evaluation consults the clinical psychologist, who administers the Hospital Anxiety and Depression Scale and the Geriatric Depression Scale.

Test Results

Both the HADS and the GDS indicate borderline depressive symptoms. The clinical psychologist expresses some concerns that the Parkinson's and CHF may confound the somatic items on the GDS, and decides to pursue the issue further through a formal interview.

Dialogic Content

In the course of the dialogue, David expands upon his conflicting desires. His family is very important to him, and he gets a lot of pleasure from visiting them. At the same time, he does not believe that he can impose upon them, or require them to visit him daily. They are busy with their lives and children, and he noticed the strain the more frequent visits had on them. He is deathly afraid of becoming moribund, and the prospect of sitting in a chair and being unable to move greatly alarms the previously energetic and active man. He does not want to die, but he is not sure whether his life is worth living – he feels that he has become simply a shell of the man he was before. He says "it's hard to find a purpose when you can do next to nothing to accomplish it." He notes that he has been having these kinds of thoughts quite frequently, ever since he had been forced to reduce his activity level.

Heuristics and Biases

David has legitimate concerns, and his thought process rightly reflects his conflict. One can note that he is engaging in affective forecasting – he is attempting to predict how he will feel about his situation far in advance, which as has been discussed earlier, is easily mistaken. This is not to say that David might not experience these emotions or that his perception is necessarily inaccurate – however, he is mistaken in conflating questions of probability with questions of necessity. In light of the finality of his decision, it would make more sense to discuss the probabilities of different outcomes, and to attempt to address any misconceptions and uncertainties as possible. In short, while David shows significantly less cognitive biases than Alice, Bill, or Catherine, there are still reasons to explore his cognition further, instead of simply endorsing the decision to forgo treatment, especially in light of his evident ambivalence.

Authenticity

As should be evident, David has two essential personal values that are in conflict. On the one hand, he has his drive and desire to be with his family. It is clear that they are important to him, and that he has long-term goals involving them (i.e., the desire to see his grandchildren grow up). On the other hand, he has a profound desire to avoid a moribund state – he greatly fears the end-stage of his illness, in which he is completely dependent upon others. As such, he wants to avoid this as much as possible. The choice he faces – whether to continue treatment for his congestive heart failure – clearly hinges upon which of these two conflicting ideas has precedence. Any recommendation made must include sensitivity to this fundamental conflict. In this particular case, further clarification of his expectations of his illness may offer insight into a resolution. The clinician may note that while a moribund state is likely, it is not immediate, and as such, it is a decision he can always return to, should his condition worsen. In the meantime, he would still be able to see his family and watch his grandchildren grow. This would allow him to maintain both driving forces in his motivation, and retain the ultimate control which clearly is important to him.

Accuracy of Self-Perception

The only significant challenge to accurate self-perception in David's case is the degree and immediacy of the incapacitation he expects. At present, it appears that his concerns about long-term dependency are making it difficult for him to identify and appreciate the likely short-term gains to be had. As such, it appears that he has some inaccuracies in his self-perception, which would seem to raise potential compromising factors to his autonomy. This does not translate to an outright refusal to honor his wishes, but stresses the need for further discussion to

ensure that he identifies, understands, and appreciates both the short-term and long-term options available to him.

Resolution

Ultimately the treatment team and clinical psychologist convinced David to continue treatment, with the knowledge that he always had the ability to refuse treatment in the future – with the assistance of the hospital ethicist, he and his family discussed his wishes regarding future treatment, clarifying what he viewed as acceptable versus burdensome interventions. The psychiatrist recommended starting David on a mild dose of antidepressants, in light of his scores on the HADS and GDS, which produced an affective improvement following his discharge. The family and social worker arranged a home care nurse to check in with David on the days when the family could not, ensuring that he had daily company.

Variations

David's case can be complicated by advancing his Parkinson's disease. At present, he has a moderate stage of the illness, but is expected to be completely dependent in three years time. Consider for a moment the impact his disease may have were it to be at an advanced stage now, rendering him moribund. If David were still able to voice his treatment preferences, and elected to forgo treatment, that decision would not be made simply upon the prognosis of his CHF, but upon how he viewed his baseline functioning. It might not be unreasonable for him to decide that while he was breathing more easily, his life had gotten to the point where he felt it to be overly burdensome. Per his disclosure in dialogic interaction, it is clear that he feels it hard to find a purpose in his life; restoring his breathing to baseline may not address this concern – the medical treatment may not provide a cure for the existential questions that remain. Treatment might make him physically better, but one could legitimately raise the question as to whether

well-being is defined solely by oxygen saturation – psychological considerations and subjective assessments of burden are requisite elements of the desirability of a particular state of being. This would seem to avoid accusations of succumbing to the availability heuristic, and the present irreversibility of Parkinson's disease weakens arguments that David is experiencing the durability bias; his movement will not get better, and entertaining illusions to the contrary seems more unreasonable than reasonable. Were David to find himself in this state, the question of compromised autonomy is less clear, and the treatment team's proposed therapeutic interventions may legitimately be interpreted as either morally ordinary or morally extraordinary.

Alternatively, the case can be complicated by exacerbating David's CHF. If it is sufficiently advanced that David will never again draw an unlabored breath, one can raise questions about the appropriateness of the intervention. While David would remain alive, he may find that he cannot adjust or adapt to such breathing difficulties, which may increase both his physical and psychological discomfort. David may therefore judge interventions which simply preserve his current level of functioning instead of improving it to be morally burdensome, and hence, may reasonably forgo such interventions. Alternatively, medical management with morphine may decrease his sensation of smothering or drowning, which may increase his subjective quality of life.[63]

A third complication is what to do if David actively wants to die. In phrasing the variation in this manner, it is not meant that David is seeking a more comfortable life, or a life unburdened with unpleasant medical interventions, or that David is actively suicidal. Instead, the

[63] Morphine can reduce the sensation of oxygen starvation, so patients may not experience the discomfort associated with breathing problems. Morphine is contraindicated in patients with breathing difficulties, quite understandably, and at sufficiently high doses it can produce respiratory arrest. This is obviously a significant concern, and finding the balance between keeping a patient comfortable and potentially administering a lethal dose of medication requires significant clinical attention. The option is raised not as a means of euthanizing the patient, but rather as a means of trading a longer but less comfortable survival for a potentially shorter but more comfortable survival period. Clearly the patient must be involved in this kind of decision as much as possible, and in as much of an autonomous capacity as is possible in light of his or her condition.

question is asked, what ought a clinician do when faced with a patient actively looking to die? This is a difficult issue – there are many reasons why a patient may express a desire for death.[64] Clinicians must be sensitive to feelings of hopelessness and helplessness producing these statements – they are hallmarks of affective disorder, and as such, ought to be explored with the patient. There are times when recognizing and validating a patient's sensation of alienation and fear can produce significant changes in how they view the world. By taking such statements at face value, a clinician may be overlooking the underlying problem., By simply dismissing them as meaningless, a clinician runs the risk of converting a borderline depression to a fulminant depression, which demonstrably affects morbidity and mortality of the underlying medical illness. Cases like this underscore the necessity of open and honest dialogue between clinician and patient; the insights offered through such a process will influence assessments of compromised versus uncompromised autonomy significantly.

Case #5: Eugenia

Our fifth patient is Eugenia, a 68-year-old African-American female presenting post-stroke with comorbid coronary artery disease. Eugenia's stroke affected her left motor cortex and Broca's area, resulting in right-side hemiparesis and significant language impairment. Her case is further complicated by a 75% occlusion of her coronary arteries. While her ability to produce meaningful speech has been compromised, she is still able to communicate in writing. She indicates that is afraid of further debilitation, and that she doesn't want to be kept alive by anything "unnatural," but has a hard time explaining what she means by the term. She is clear

[64] Aside from motivations encountered in cases in the literature, I have talked with numerous patients of varying demographics who have expressed a desire to die. Some do so out of fatigue (i.e., illness has worn them down), some do so to escape intractable pain, some do so for attention, some do so to remain hospitalized. Exploration of the desire for death will therefore be a critical part of dialogic interaction – we certainly ought not to accede to the 'autonomous wish to stop treatment' of a patient who voices a desire for death because she feels lonely and wants attention.

that she doesn't want to be intubated, nor does she want "to be hooked up to a bunch of machines." She is uncomfortable with cardiopulmonary resuscitation, especially when she hears that it might require breaking her ribs, and doesn't like needles. She expresses some ambivalence about death, indicating that there are times when she feels ready to die, especially when she gets chest pains. Her treating physician has recommended two coronary artery bypass grafts (CABGs), but she isn't sure if it is worth it, as she feels very physically compromised.

Demographics, Personal and Family History

Eugenia is a high-school educated, former restaurant manager. She found time to start a family, has two sons and a daughter, all of whom live out of state, and her husband is a retired police officer. They have flown in to be with her during her hospitalization, and one of her sons has offered to move back to take care of her. She is moderately overweight, and never really maintained an active lifestyle outside of work.

Throughout her life, Eugenia has been dependent upon others for motivation. Although she ended up in positions of responsibility as a wife, mother, and manager, she has always been indecisive, and usually has required someone else to be her impetus in deciding major issues. After graduating high school, she went straight to work, but didn't really find anything which captured her interest. After ten years of shifting from job to job, she settled in as a waitress at a growing chain of restaurants. With prodding from her husband and family, she rose from waitress to shift leader, to assistant manager, to manager, and was happy in that position until her retirement. She was unsure about having children; her feelings shifted significantly from month to month until she discovered that she was pregnant with her first child. Following the birth of her first son, she and her husband settled into a routine, until her second pregnancy, which produced twins, her daughter and second son. Following a brief maternity leave, she returned to

the restaurant. Between her earnings and those of her husband, they were able to provide a comfortable home. Her parents helped out watching their grandchildren when Eugenia and her husband were at work.

Following their retirements, Eugenia and her husband enjoyed a fairly quiet life. He had occasional poker games with his former coworkers, while she puttered around the house and garden. One Sunday, while washing up the lunch dishes, a plate fell from her hand, breaking on the floor. She noticed that she started to feel very weak on her right side, and found it very difficult to talk and balance, preventing her from calling to her husband. Fortunately he had heard the broken plate, and came into the kitchen in time to prevent her from falling to the floor. He called an ambulance and rushed her to the ER, where a left side ischemic cerebrovascular accident was diagnosed. Following her admission to the hospital, further testing revealed the extent of her coronary artery disease. Her clinician approached her about CABG, but she is feeling a little overwhelmed by everything going on. The motor deficits she is experiencing have shaken her, and being hampered in getting her frustrations out has made her feel helpless and hopeless.

Condition and Prognosis

Eugenia experienced a left side stroke of the middle cerebral artery. Because it was caught and treated relatively quickly, her neurologist is confident that her deficits will be manageable with physical and speech therapy, but stresses that this is not guaranteed. There is significant concern that her coronary artery disease may lead to another ischemic attack, which may be fatal, but this is an unknown. Overall, her clinician rates her likelihood of significant partial recovery at 60%, but believes that vestiges of her neurological deficits will remain for the rest of her life, however long that may be.

Case Treatment

Eugenia's treating physician notes that Eugenia is emotionally labile, which makes him suspect that her stroke is affecting her psychologically as well as physiologically. He consults the psychiatric consult liaison, who after interviewing her concludes that there is a psychiatric comorbidity. He recommends further testing by the clinical psychologist, who administers the Mini-Mental Status Exam, the Hospital Anxiety and Depression Scale, and the Geriatric Depression Scale, offering assistance when needed due to Eugenia's impairments.

Test Results

The Mini-Mental Status Exam indicates good cognitive functioning of the major executive areas. Both the Hospital Anxiety and Depression Scale and Geriatric Depression Scale yield borderline scores, suggesting the need for further interviewing to offer further evidence or absence of an affective disorder.

Dialogic Content

Eugenia finds it difficult at times to express her feelings – aside from her language deficits, she finds it difficult finding words which can capture fears and worries, of which she has many. She worries about her heart, about her children, about her husband, about another stroke. She worries about being bed-ridden or confined to a wheelchair. She worries about never being able to tell her children that she loves them. She worries about rehabilitation, and whether she will ever regain her ability to function. She finds writing to be frustrating, doubly so when she tries to speak and cannot. She feels trapped within her body, a feeling she had never had before in nearly 70 years. She hates having to live this way, and is not sure whether she is willing to go on or attempt to restore her health. She writes that these thoughts whirl around her head every time she tries to move or speak and fails. She writes that even when she tries to calm herself

down, she can never escape these worries. They always linger, waiting for a trigger to spring back to the forefront of her thoughts.

Heuristics and Biases

While Eugenia notes a variety of distressing cognitions, she does not explicitly demonstrate any particular heuristic or bias. Instead, what is presented is significant backstage cognition – she notes that the thoughts are always there, but she isn't always aware of them. Their ability to spring quickly to mind suggests that they are a significant part of her underlying cognitive mechanisms, which invites speculation as to the extent to which they are influencing the mental constructs she produces. As such, one cannot state definitively that her autonomy is compromised on these grounds – one would need to discuss these further with her, and perhaps administer the Automatic Thoughts Questionnaire to attempt to quantify her processing. Therefore, one ought to conclude that there is possible influence in her thought process, but not definite influence.

Authenticity

Clearly Eugenia is conflicted – she demonstrates significant ambivalence in her motivations and concerns. As such, it would be mistaken to simply fiat one guiding principle. Some patterns do emerge, however – her worries fall into concerns about her physical functioning and her relationship with her family. Any proposed solution, therefore, should approach the issue with sensitivity to these important and influential elements. In light of Eugenia's difficulty in making decisions in her life, one could approach her and ask if she felt more comfortable making this choice with her husband and/or children. It is recognized that this may confound the issue – it is not uncommon for family members to disagree. However, as Eugenia is capable of expressing her wishes, she would retain ultimate authority in the decision.

Further, it should be noted that she has been given a favorable probability of recovery to a point where she would not be as hindered as she presently is. It would be important to note that treatment and rehabilitation would allow her to maintain more control over her life, and to engage with others in ways that she finds more meaningful than written notes. This would simultaneously address both her concerns about familial interaction as well as physical impairment. It is not a cure-all, but it is a significant improvement over her current condition, and therefore worthy of consideration.

Accuracy of Self-Perception

Eugenia demonstrates an accurate assessment of her present state, and has legitimate concerns about her recovery. She does not seem to harbor misconceptions about her illness, but does seem overly pessimistic about the likelihood of recovery. This is a far cry from Alice's agnosognosia; Eugenia is aware of that her concerns are potentially influencing her cognition. In light of these, there seems to be little ground on which to challenge any resultant decision to forgo treatment based on self-perception.

Resolution

Eugenia opted to make her decision with input from her husband and children. They favored her attempting rehabilitation and treatment for her CAD, recognizing that both may amount to only temporary fixes. Bolstered by her family and sure of their support, Eugenia elected to try rehabilitation and treatment. Eugenia and her husband used the current crisis to tell their children what they would want done if something else happened to them.

Variations

Eugenia's case can be complicated by changing her underlying motivations – instead of Eugenia seeking to avoid being kept alive via mechanical interventions, she now is concerned

about remaining alive at all costs. She seeks every possible intervention, mechanical, chemical, electrical, surgical, etc. During dialogic interaction, she discloses that her motivations for doing so stem from a dear of death; she doesn't want to die, and believes that by using all of the avenues of medical science available to her, she will be able to postpone her death indefinitely, or at least of pushing off her death for the foreseeable future. This is a significant shift away from the values she disclosed in the initial case study, and at this point, her medical wishes are bordering on unreasonable expectations. This is not to say that medicine cannot stall mortality, but it certainly cannot prolong life indefinitely, nor can it overcome profound insults and injuries.[65] As such, a significant concern in Eugenia's medical management will be establishing reasonable versus unreasonable expectations, and helping her to clarify what, if any, point she would wish to forgo treatment. Issues to consider would include whether she could find her life to be meaningful without consciousness, or without cortical function, or whether she would want her ribs broken repeatedly as CPR becomes more necessary, or whether she would want to be machine-dependent for respiration, hemodialysis, nutrition, hydration, excretion, etc. These are fundamental issues, and some patients are unaware just how much can be done when they say "Do everything." Setting reasonable treatment expectations and treatment boundaries would be necessary in this case.

Her case can also be complicated by eliminating the sequelae from her stroke, reducing her immediate medical concerns to her cardiac problem. In the initial case presentation, Eugenia disclosed that a significant source of her frustration was the impairments she incurred following her stroke, which led to her ambivalence about treatment/non-treatment of her cardiac condition,

[65] Some of the terms employed here require clarification – the kinds of insults and injuries considered here are systemic, e.g., massive trauma, drug-resistant infections, progressive illnesses like AIDS, etc. Interventions exist which can alleviate significant pain and suffering, and can prolong lives significantly, but cannot render death optional.

as well as her sense of helplessness and hopelessness. If these sequelae were removed, but her ambivalence, helplessness, and hopelessness persisted, then there would be significant reasons to suspect cognitive distortion and potentially compromised autonomy. This is not to detract from the seriousness of her cardiac condition – as has been discussed earlier, depression frequently occurs when heart pathologies are diagnosed. Many patients have disclosed significant distress when hearing the diagnosis, and find it difficult to adjust both to the news and to any changes that their heart condition may require. As such, Eugenia's treating clinician should be alert for psychological sequelae to the coronary artery disease, and should be especially vigilant in light of any resultant disclosures of helplessness, hopelessness, or ambivalence regarding treatment.

If Eugenia's prognosis were worse, we would expect her ambivalence regarding treatment as well as her affective state to become more pronounced (i.e., mild to moderate depression giving way to more severe forms). If the likelihood of significant recovery were decreased to 25%, we would expect several possible reactions. It is possible that she would be further overwhelmed by her condition, and may demonstrate even greater inability to decide upon a course of action. It is possible that she may develop a more severe depressive episode, or that she may demonstrate depressive cognition (i.e., interpreting a 25% chance of significant recovery as 0% chance of significant recovery). This latter avenue would open up her judgment to more significant challenges – at the point where she begins to equate 25% with 0%, there is reason to suspect that she is evidencing cognitive distortion, and as such, the clinician should treat the decision to forgo treatment as more suspect. It is not unreasonable to become pessimistic when one hears a low probability of success – but it *is* unreasonable to become fatalistic or irrational. Treating clinicians, therefore, should pay greater attention to underlying cognition in situations like this.

Case #6: Frank

Our sixth patient is Frank, a 75-year-old white male presenting post-stroke with comorbid congestive heart failure (CHF) and emphysema. Frank was struck blind by a cerebrovascular accident (CVA) affecting his posterior temporal artery, compressing his visual cortex. In a way, he was fortunate – he was in the emergency room being treated for labored breathing when he stated that he could no longer see. He was rapidly scanned, and the bleed was found and treated. Frank initially was less concerned about his sight than he was his breathing – he told the emergency department staff that when he woke up it felt like he was breathing through a wet towel, and that things got worse over the course of the day. With the intracranial bleed addressed, Frank faced two difficult issues – he might not ever see again, and his breathing was not going to get any better. This is not what he imagined his life would be like as a young man, and he is unsure whether he wants to continue with the breathing treatments. After speaking with pastoral care, Frank is weighing his options, aware that he has the ability to elect palliative care in place of further treatment.

Demographics, Personal and Family History

Frank is a retired construction worker, who spends his time watching sports on television and reading detective novels. He has a few friends at his nursing home, but he has lost contact with his long-time friends. He has two children by different mothers, but has never been married. He is close to neither woman nor to his children, and it has been nearly two years since he last heard from any of them. He putters around his room at the home, but tends towards inactivity.

His world is essentially the games he watches and the *noir* novels of Micheal Connolly, Dennis LeHane and Keith Ablow. Despite the other health concerns he developed over the

years, his eyes have been fairly reliable. He was a two-pack a day smoker for twenty years before being diagnosed with emphysema, at which point he cut back, but was never able to quit completely. As a result, his breathing has gotten worse over the years, eventually requiring him to use oxygen at night. When his breathing problems were exacerbated by the CHF, he was put on Lasix and again cautioned to stop smoking completely. Frank was able to quit for a month, but found himself reaching for his cigarettes whenever he turned on ESPN.

On the day of his admission and CVA, Frank woke at his usual time. He had had bad mornings before, and brought his oxygen cylinder with him to breakfast. After his usual coffee and low sodium breakfast, he returned to his room, showered, and turned on his television. Sitting in his favorite chair, he noticed that his chest was getting very heavy and it was harder to breathe. Hitting the call button, he expressed his alarm and was in an ambulance shortly thereafter. Upon presentation to the emergency department, he was given 100% oxygen while his medication list was faxed over. Halfway through his physical exam, the world turned to black. Panicking, he informed his physician, who took him for his scan.

When the CVA was managed and Frank was admitted to the hospital for monitoring, his visual deficits remained. The attending pulmonologist and neurologist informed him of the extent of his medical problems, and his treating physician proposed continuation of the breathing treatment and the initiation of rehabilitation with the aim of minimizing his visual deficit. Frank, however, was not convinced that this really was the best course of action for him – he decided it might simply be better to be comfortable, instead of simply waiting while his combined illnesses worsened.

Condition and Prognosis

Frank's neurologist places his recovery of partial vision at 50%, due to the speed with which his CVA was addressed. His pulmonologist is less optimistic about Frank's breathing – he believes that Frank was at a plateau for a long time, and now his condition is likely, but not guaranteed, to worsen. He expects Frank to be on continuous oxygen upon discharge, and even then to experience some difficulties.

Case Treatment

Frank's treating clinician is aware of the profound impact a stroke can have on a patient's mood and recovery, and contacts the psychiatric consult liaison. After interviewing Frank, the consult liaison believes that there may be a psychiatric comorbidity, but Frank exhibited only a handful of symptoms consistent with a depressive disorder (principally somatic). In light of this, the consult liaison requests psychological testing. The clinical psychologist administers the Hospital Anxiety and Depression Scale, as well as the Geriatric Depression Scale and Automatic Thoughts Questionnaire, reading the questions out loud to assist Frank in completing the items.

Test Results

The Hospital Anxiety and Depression Scale indicated subthreshold endorsement of depressive symptoms. The Geriatric Depression Scale endorsed depression, but the clinical psychologist believed this to be due to confounding physical symptoms of the CVA and CHF. The Automatic Thoughts Questionnaire indicated that Frank is experiencing some ego-dystonic automatic thoughts.

Dialogic Content

Because the testing results indicated no clear depressive disorder, the clinical psychologist desired clarification of the results through dialogue. While Frank expressed some

frustration with his present condition, he understood that the breathing therapy and rehabilitation may allow him to enjoy watching sports and reading again. Frank stressed, however, that "if my vision isn't good enough to wear glasses, it really does me very little good, as I could hold my books, but couldn't read them." He noted that his vision was the last thing that he could depend on, prior to his CVA; he stressed that "while I can handle a lot of things, not being able to see really messes up my world." Frank related his concerns about what the neurologist told him were reasonable versus unreasonable expectations, and stated that "50/50 is worth a shot, but if it doesn't work out, I don't want to try anything else." He was disappointed by what the pulmonologist told him, but stated "that so long as I can see, I'll strap the mask on." He understands that he might not be thinking clearly at the moment, as his world has been changed so radically and so quickly.

Heuristics and Biases

Frank exhibits very little bias in his thought – he appears to have incorporated the information from the pulmonologist and neurologist have told him with minimal misconception and misunderstanding. If any critique can be made of his thought process, one might consider discussion about his reliance on sight, or raise concerns about durability bias.

Authenticity

Frank's visual orientation is quite clear, and as such, any approach to his condition must be sensitive to this motivating factor. Frank acknowledges that the return of his vision is not necessarily guaranteed, which may offer an avenue of approach – after all, one does not necessarily have to see a game to enjoy it, and many books are available in large print or audio format. While these responses may not be convincing to him, they do serve to acknowledge the things that are important to him, as well as opening up discussion of means by which he could

still enjoy his life in the presence of impairment. If, however, Frank is not swayed by the argument, one ought to propose a trial period of therapeutic intervention – this will combat the durability bias by seeing how he reacts and behaves in the presence of his deficit.

Accuracy of Self-Perception

Frank appears to have an accurate picture of both his condition and avenues of care available to him. He recognizes that his deficits may cause him to approach problems in a way that may not be advantageous, indicating that he is aware of potential influences on his cognition. There seems to be little ground to challenge his resultant decision to forgo treatment based on poor self-perception.

Resolution

Frank elected treatment, but only on a provisional basis. If his discomfort continued despite the rehabilitation and Lasix, he would stop further treatment, electing palliation instead. His treatment team endorsed his position, and initiated treatment. After two weeks of rehab, Frank demonstrated minimal improvement, and his difficulty breathing continued. He elected to forgo further treatment, and was referred to hospice care.

Variations

The first variation on Frank's case to be considered improves his chances of recovering his vision. As the case was initially presented, Frank had an equal chance of partially recovering his vision as he had of never recovering his vision. He made his choice to continue breathing treatments contingent upon his visual improvement, and elected not to continue the treatments when he had minimal recovery. If, however, he did experience improved vision, to the point where he was able to read large-print books, and still elected to forgo his breathing treatment, questions could be raised about the autonomy of his decision. The values that he disclosed

during dialogic interaction demonstrated the primacy of his vision; he stated that if he were able to see, then he would put up with visual deficits ("So long as I can see, I'll strap the mask on."). As such, the choice to forgo treatment would then apparently conflict with his stated preferences, and such conflicts raise the possibility of compromised autonomy. Clinicians must be cognizant of value conflicts in their patients – a requisite element of an autonomous action is harmony with one's authentic values and a knowledge of one's preferences; the choice to forgo treatment in this case is inauthentic, and therefore ought to be challenged.

The second variation on Frank's case involves a significant improvement in his breathing and a decreased likelihood of partial visual recovery. As the case was initially presented, Frank's pulmonologist does not anticipate Frank's breathing to improve to the point where he will not require oxygen, and partial recovery of vision was estimated at 50/50. In this variant, Frank's pulmonary baseline is higher, and he is expected to make a full recovery, but his chance of partial visual recovery is only 25%. He is expected to experience little to no discomfort when breathing, but it is probable that he will not see again. Frank already voiced his priorities concerning improved breathing and recovery of vision, and a similar analysis can be applied in this case. The decreased chance of visual improvement ought to be weighed more heavily than the improved breathing when considering any expression of a desire to forgo treatment. There is still some ground for questioning his choice based on the durability bias, and clinicians encountering this choice to forgo treatment must make sure that their patients are not equating a diminished chance (25%) with no chance (0%). As noted in previous case variations, an inability to recognize the difference between possible and impossible raises concerns about compromised cognitive processing.[66]

[66] I would argue, however, that this becomes less of an issue as the probability of recovery approaches 0. Clearly there is more distortion present when a patient equates a 95% chance of recovery with 0% chance of recovery than

A final variation raises the possibility of Frank being unable to make a decision about whether or not to continue his treatments. As in previous cases, his decision-making ability may be improved with help from a family member or friend – this is challenging, however, in light of his few current social attachments. One might question whether his ambivalence may be a product of his alienation and isolation, and this might be a fecund avenue of inquiry. If no such facilitator can be found, Frank may benefit from longer and more introspective discussion with someone less close to him, but willing to engage in dialogic interaction (e.g., pastoral care, therapist, etc.). The key to resolving this case hinges upon finding the source of his ambivalence, the values or preferences which are conflicting, and exploring the area of contention between the two. Frank may benefit from having the opportunity to discuss this conflict, which may give one option greater decisional weight, allowing him to voice a preference. This is not a guaranteed outcome, however, and an inability to make a choice may demonstrate compromised autonomy. Indecision alone is not enough to demonstrate an inability to make medical decisions, and as such, it would be inappropriate to pursue guardianship, which would compromise Frank's autonomy further. The most justifiable course of action would be to continue his breathing treatments while engaging in dialogic interaction, attempting to facilitate a decision, as it always remains an option to discontinue them and explore palliative care in their stead.

Case #7: Georgette

The seventh patient is Georgette, an 82-year-old African American female presenting with stomach cancer. What she thought was cramps and gas has proven to be much more severe. She has received the news stoically, which surprised her clinician, who expected a more emotional reaction. Georgette explains that "we're all going to die from something" and that she

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there is when a patient equates a 5% chance of recovery with 0% chance of recovery. In the case of Frank, 25% is still a meaningful chance, even if it is unlikely.

is surprised that she hasn't gotten it sooner, in light of her family history. She admits to being afraid at times to die, but says that she knows she can't avoid it, and that she's had a good life. She states that if she "can live a few more months to see the birth of my first great-granddaughter, I will be able to die a happy woman." Her clinician has discussed the possibility of surgery to remove the cancerous tumor, indicating that that could help improve her chances of seeing her granddaughter born. After discussion with the oncologist, she was told that surgery itself posed a significant risk in someone of her age. Georgette faces a difficult choice – because of the risks of anesthesia and potential complications from the procedure, she may live longer without the surgery. At present she is leaning towards forgoing treatment, as she views it as invasive, and "a hard thing on these old bones."

Demographics, Personal and Family History

Georgette is a lower-middle class nanny to the young children in her neighborhood. She is a frail woman, who prior to her diagnosis maintained a somewhat active life, if only by keeping up with the children in her care. She is widowed, with four children, two of whom live in the same town.

Georgette has lived a long life; she was born in Georgia shortly after the first World War. Her father transplanted the family north when his factory moved. The youngest of six children, she attended school through the eighth grade, at which point she found a job as a waitress. While working, she met her first husband, a mechanic in the Navy. She and her husband moved to Hawaii, where he was killed in the attack on Pearl Harbor. She returned to her family full of shock and grief, and stayed with them for two years before moving out. She met the man who would become her second husband a year later. He was also a military mechanic, and would go on to serve in Korea. He was fortunate enough to return home, where they started a family. She

had four children, two boys and two girls. Her husband provided for the family as a mechanic and working construction part-time, allowing Georgette to remain at home with the children.

Georgette's mother was diagnosed with breast cancer shortly after Georgette's second son was born. Not knowing that the lump was malignant, Georgette's mother did not believe anything was amiss until she began to experience headaches, dizziness, and difficulty breathing. When she checked with her family doctor, there was nothing that could be done for her – she would drop dead walking back from the market shortly thereafter, and an autopsy revealed metastasis of her cancer to her brain and lymph nodes. Her father, a long-time smoker, succumbed to undiagnosed lung cancer two years later.

Georgette herself was no stranger to medical problems – following the birth of her fourth child, she had to have an emergency hysterectomy when she hemorrhaged. Her recovery took a long time, and strained her family, both monetarily and mentally. Her husband was working two full-time jobs to pay for her hospital stay and provide for the children's schooling. Five years after her recovery, he died from a heart attack in his sleep. Georgette's medical life was relatively uneventful after her husband's death. She received her husband's pension, and all four of her children graduated from college, two with the help of scholarships. Georgette spent her time walking in the neighborhood, going to the library, and listening to the radio.

Georgette's stomach began to bother her after a neighborhood pot luck dinner to thank her for her selfless care of the neighborhood children. She initially attributed it to her neighbors' salted brisket, and it went away after a few days. It would return a few days following that, following an on-again, off-again cycle. She attributed it to unhealthy food, and began treating herself with antacids and Pepto-Bismol. She woke one morning with a strong pain in her

stomach, at which point she presented at the emergency department. Endoscopy revealed multiple tumor sites, which proved to be malignant upon biopsy.

Condition and Prognosis

Georgette has been diagnosed with a stage IIIA (T2N2M0) cancer. Her oncologist predicts that with surgery to excise the tumor she has approximately a 15% chance of five-year survival, although she is doubtful as to whether surgery would be an option for a patient of Georgette's age.

Case Treatment

Georgette's clinician is concerned about the near apathy with which Georgette greeted the news, and believes that a depressive comorbidity may be present. She contacts the psychiatric consult liaison, who finds Georgette difficult to read. In order to get a clearer picture, the liaison requests a consult from the clinical psychologist, who administers the Hospital Anxiety and Depression Scale and the Geriatric Depression Scale.

Test Results

The Geriatric Depression Scale indicates some depressive symptomatology, but the clinical psychologist believes it may be confounded by the somatic symptoms of cancer. The depression subscale of the Hospital Anxiety and Depression Scale reveals a subthreshold number of depressive symptoms that are not confounded by somatic issues. Both the clinical psychologist and consult liaison believe that if a depressive disorder is present, it is mild at worst.

Dialogic Content

In discussion with her treating physician, Georgette relates how her family has always dealt with death. Her first husband, her parents, and three of her elder siblings have all died, and

while the family grieves for them, they are all aware that death is not optional, and there is a strong belief in their family that it is just a natural part of life, in both philosophical and theological terms. They miss their dead relatives, but celebrate their lives. Georgette is comfortable talking about death, and states that she understands that forgoing surgery now will likely remove it as an option in the future. She smiles when she thinks about her family, and says she is "looking forward to seeing them again once I move on." She states that if she can do something about the pain in her stomach, she will be happy just to make do as she is. She states that she will "be able to cook for my children and grandchildren, to see them smile and laugh for a little longer," and "maybe see that happy little girl come into the world." She states that while the surgery may help this happen, it is just as likely to prevent it from happening, and "that's a risk I'm not sure I want to take."

Heuristics and Biases

While Georgette is focusing on her family's experience with death, she appears not to be employing any unrealistic heuristics. The conclusions she is reaching are consistent with the available data, and while one might raise questions about some of her predictions of how she will feel in the future, she appears to be exhibiting little bias in her thoughts. Her conclusions seem to be quite reasonable, and offer little ground for suspecting diminished autonomy.

Authenticity

There is little question that Georgette sees her choice as consistent with her attitude towards life and death. Family is clearly an important motivator for her, and her choice appears consistent with what probabilistically will give her the most time with them. There are always some uncertainties in making probabilistic statements, but the available evidence makes her statements and judgments reasonable and authentic. Like the analysis concerning heuristics and

biases, there is little ground for suspecting diminished autonomy from the perspective of authenticity. The only potential concern lies in the nature of probabilities – one should be clear on the reasonably foreseeable and most likely outcomes before making these kinds of judgments.

Accuracy of Self-Perception

Georgette demonstrates a good understanding of her situation and prognosis. She is clear in her priorities and the likelihood of attaining them in light of her condition. There is little ground on the basis of self-perception for suspecting diminished autonomy.

Resolution

Georgette ultimately elects to forgo surgery; she was referred to hospice care and died three months later, just shy of the birth of her first great-granddaughter.

Variations

The first variation on Georgette's case focuses on her age. The case as initially presented detailed the life of an octogenarian, but the analysis and outcomes can shift dependent upon her age. Clearly a woman half her age likely would not have had her experiences (e.g., losing a husband in Pearl Harbor or awaiting the birth of a great-grandchild), but a younger woman could have similar family histories and personal experiences with illness. As such, the authenticity of the decisions may be comparable. There are some important differences, however. For instance, the physical resources of a forty-year-old woman would likely be greater than those of an eighty-year-old woman. This could affect the issue of recovery; Georgette stated in the initial case study that she feels the surgery would be difficult on her, as she is significantly older. While this may hold true for an older person, the same line of reasoning becomes suspect in younger and ostensibly healthier people. The clinician treating Georgette in this case variant should be suspect of the claim that the surgery and recovery will be too difficult. This is not to suggest that

it is impossible for the recovery from surgery to be too taxing for a younger person or that this has never happened clinically; however, it does stand to reason that such instances are the exception, rather than the rule, and as such, should raise cautionary flags in the evaluation of autonomous action. On the other hand, however, the five-year survival rate for this type of cancer does remain low, so it is not unreasonable for a younger patient to question whether an invasive procedure and painful recovery process are off-set by a small survival rate. In these cases, therefore, it would seem prudent to engage the patient in dialogue to explore her important values and goals, and to see whether the risks and potential benefits of treatment are harmonious with these subjective assessments.

A second variation maintains Georgette's age at eighty-two, but increases her five-year survival to 40%. This is a significant increase (+25%), and exerts am important influence on the assessment of her case. As noted in previous cases, it is important for clinicians to be cognizant of the patient's assessment of probabilities. A patient who cannot appreciate the difference between 15% and 40% chance of five-year survival demonstrates potential impairment.[67] As the case was initially presented, Georgette opted to forgo further treatment in favor of hospice care. This decision is still reasonable in light of her other concerns (i.e., difficulty of recovery at her age), but the risk/benefit assessment has changed, and as such, her clinician ought to approach her to ensure that this information is understood and appreciated. As in the first variation, an increase in her probability of five-year survival of this degree warrants exploration of her values and goals. She may decide that the increased likelihood of five-year survival increases the change of seeing both the birth of her first great-grandchild as well as her first birthday and first

[67] Just as in earlier cases, the indication of impairment will be dependent upon the amount of change. Not appreciating a 5% increase demonstrates significantly less potential impairment than failure to appreciate a 50% increase.

words. As her family is clearly an important motivator, these concerns are quite germane, and as such, warrant careful consideration.

A third variation would reexamine her case in light of potential availability bias. Cancer has been part of her family throughout her life, and as such, it is something with which she has great familiarity. However, there is a legitimate question raised as to whether her thinking has become fatalistic – because her parents both died from cancer, and she disclosed that she was surprised that she didn't get it earlier, could she simply be working under the impression that she must die from cancer, too? To be sure, cancer has a genetic etiology, and that first-degree relatives of individuals with cancer have higher risks. However, like many illnesses, cancer appears to follow a diathesis-stress model, in that while she may carry the latent susceptibility to cancer, it requires an impetus before developing into a clinical condition. She may have taken it for granted that she would develop cancer at some point, and may have resigned herself to her illness, instead of approaching the illness more objectively. In short, her thinking may have been "This is going to happen; why fight it?" Such thinking does not automatically produce compromised autonomy, but it certainly opens an avenue for exploration and discussion. It would seem that Georgette has focused on the examples of cancer in her life (her parents), both of whom had progressed too far in their illness to effectively treat it, which has led her to believe that she is necessarily in the same boat (hence, availability bias). Her clinician should challenge this preconception, if only to note that there are qualitative differences between her parents' experiences with the disease and her own, and any subsequent differences in prognosis. The outcome for this variation may be the same as the initial case presentation, but at least a source of potential error would have been raised and addressed.

Case #8: Harry

The eighth patient in our case metric is Harry, a 68-year-old Asian-American male presenting with pancreatitis, gastroesophageal reflux disease (GERD), diabetes mellitus (which resulted in bilateral amputations below his knees), and congestive heart failure (CHF). Harry presented to the emergency department with flare-ups of pancreatitis and congestive heart failure; his breathing had been labored all day, but towards dinner it had become markedly worse and he was diaphoretic, causing his care team to transfer him to the hospital. While his CHF is responding to Lasix, it is very slow going, and Harry appears to have hit a plateau – he is neither improving nor getting worse. The concern at this point is pain control – his pancreatitis is relentless, and is causing him significant discomfort. Despite the analgesia, there is worsening breakthrough pain; his treating clinician is concerned that if he provides any more pain medication, he may end up suppressing Harry's breathing, a significant concern in light of his current CHF difficulties. The team has reached an impasse – do they give priority to Harry's breathing or his pain management? Harry stresses that his primary concern is pain, but he is inconsistent. There are times when he confides a fear of not being able to breathe. He hates the sensation of being smothered, and had a fear of drowning all of his life. The team, as a result, is unclear as to what to do for him.

Demographics, Personal and Family History

Harry is a college-educated retired engineer. He has two younger brothers, but has not maintained a close relationship with either of them. He has never married or had children, and has few close friends. His amputations and physical frailty have led to his housing in a skilled care facility for the past several years, but while he is friendly with the staff, he has not attempted to develop friendships with them.

Harry's life has been, in his words, "uneventful." He was born in Chicago, studied physical engineering and literature at college, and took a job with an architectural firm after graduating. His social life brought him many casual acquaintances and social contacts, but he "never really found anyone who clicked." As such, while he spent many nights out on the town, he frequently went home alone. He never felt strongly about marrying or raising a family, he always said that it might be nice, but it didn't really cross his mind much.

Harry has had significant medical problems – his diabetes has hit him hard, causing bilateral amputations below the knees, as well as advancing retinopathy. His socialization dropped off significantly after the first amputation, but it occurred relatively late in his life – he spent most of his time on the internet, a habit he was able to continue after diabetes took his other leg and he moved into the skilled care facility. His retinopathy made it difficult to see the screen, but he made do. It wasn't until his pancreatitis and CHF started giving him difficulties that he began thinking about the quality of his life.

On the day of his admission, Harry awoke as normal. He was wheezing slightly in the morning, but he attributed it to the air conditioning, and not having a lot of fresh air. His appetite diminished after breakfast as he began to experience abdominal pain, which wasn't relieved by his usual PRN medication. As evening approached, both his pain and his breathing worsened. An aide found him in his room heaving his chest for air when they were gathering their residents for dinner. At that point, he was transported to the emergency department.

Harry hates pain, and has said so on numerous occasions. At his skilled care facility, he has repeatedly asked for increases in his PRN pain medication, but did not demonstrate any signs of addiction or dependency – for him, it was genuinely a pain management issue. In the hospital,

he has generally maintained this attitude, stating that if he had to choose between difficult breathing and abdominal pain, he would choose the difficult breathing.

Condition and Prognosis

Harry's pulmonologist believes that Harry will be dependent upon continuous oxygen following discharge, which would offer further difficulties for his mobility. He is not confident that even with Lasix and oxygen Harry will be able to draw an unlabored breath. Harry's pancreatitis is chronic and debilitating – his attacks are becoming more frequent and more severe.

Case Treatment

Harry is quite lucid, despite his compromised condition. In discussion with his clinician, he admits to fears of smothering and of pain, but he more often than not says that he would prefer to be pain-free than to worry about his breathing. He states that he would prefer to be healthy, but he would settle for being comfortable; his pain causes him more discomfort than his CHF. His apparent resignation raises some concerns in his treating physician, who suspects that there may be a psychiatric comorbidity. He contacts the psychiatric consult liaison, who is concerned about pain masking depression. Focusing on the somatic diagnostic criteria, the consult liaison notes recurrent anhedonic elements in Harry's statements, and recommends that the clinical psychologist evaluate him formally. The clinical psychologist administers the Hospital Anxiety and Depression Scale along with the Geriatric Depression Scale and the Dysfunctional Attitudes Scale.

Test Results

Both the GDS and DAS note the possibility of depression, but the clinical psychologist suspects that there may be confounding due to the somatic content of Harry's primary illnesses. The HADS subscale for depression indicates subthreshold depressive elements.

Dialogic Content

The clinical psychologist opens a dialogue with Harry about his preferences in treatment. Harry discloses that he would prefer that his pain be managed over his breathing, although it would be nice if both could be resolved. Since he knows that won't happen, he would at least like to be able to sleep, which he finds next to impossible with his breakthrough pain. He discloses that he's relatively happy with the life he led, and that he doesn't really worry about death – at his age, he is becoming more used to the idea, and it doesn't make him afraid. "After all," he reasons "I'm not going to know when it happens." Instead, he is more afraid of pain and suffering. Death may not be unpleasant, he adds with a small smile, since he "doubts his pancreatitis will bother him much at that point." He states that he is aware that his current condition may make him "feel a little down," but "it's nothing I haven't dealt with before, and I haven't changed my mind when it's happened then." Harry even noted that he didn't want CPR if something happened, because it would be too rough on him if it worked – the added pain of cracked ribs would simply be too much for him.

Heuristics and Biases

If Harry is demonstrating any particular bias in his cognition, it is a durability bias about his pain. This, however, is questionable, as he has significant experience with his pancreatitis, and has no reason to suspect that it will abate in the future. Further, his conclusions are reasonable, and he is basing them upon conditions of which he has significant confirmatory

experience. His pain may be an influential element of his cognition, but it is not unreasonably so, and as such, there is little ground for claims of compromised autonomy.

Authenticity

Harry has affirmed and reaffirmed his basic principle: avoidance of pain and suffering, not necessarily avoidance of death. One might raise challenges to this, but it appears to be a consistent pattern in his life, and appears to be a genuine constituent of his self-identity – it is something he endorses repeatedly, outside of his current medical crisis. As such, it would seem reasonable to endorse decisions based upon this guiding principle as autonomous. One might challenge this based on his statements about a fear of smothering, but there are also contemporaneous statements placing fear of pain as a higher concern than fear of smothering. Thus, there is some uncertainty in this case, but not enough to ground conclusions of compromised autonomy based on authenticity.

Accuracy of Self-Perception

Harry seems to possess an understanding of his condition based upon his experience with chronic illness. He does not seem to be harboring any unreasonable expectations concerning his prognosis with and without treatment, and he seems to be aware of the likely outcomes of favoring pain management over respiratory control. As such, there seems to be little grounds upon which to base claims of diminished autonomy.

Resolution

Harry ultimately elected increased attention to pain management over interventions designed to improve his breathing. Four days after making this decision, he went into cardiac and respiratory arrest. Per his instructions to staff, CPR was not performed, and he was declared dead shortly thereafter.

Variations

The first variation on Harry's case involves his diabetes; the bilateral amputations profoundly affected his mobility and his retinopathy has affected his socialization. This disease has put him in a place where he is very dependent upon others, and has little to focus on save his own physical state. If diabetes were removed from the equation, he would still have significant medical issues (the pancreatitis, GERD, and CHF), but he would not necessarily be as dependent upon others, and may have other aspects of his life upon which he could focus his attention. Harry may find this quite relevant to his long-term preferences in medical care. Restoring his ability to be active and socialize may cause him to place more emphasis on one treatment versus the other, as he may elect to tolerate some pain and avoid compromising his breathing, in order to be more active and social. This is not guaranteed, to be sure, but it would likely facilitate the decision-making process; allowing for some novelty in an otherwise routinized situation can make otherwise intolerable conditions tolerable. To wit, Harry's day involves little more than focusing on his somatic concerns; increasing his mobility and ability to socialize may decrease the attention he pays to his pancreatitis. Further, the case as presented noted the possibility of Harry demonstrating a durability bias; by introducing an avenue by which he can change his surroundings and experiences (since removing the diabetes from the case presentation has restored his mobility and vision), it is possible that he may not exhibit the same affective forecasting.

The second variation on Harry's case restores his diabetes and its comorbidities, but involves a complete absence of pancreatitis and it's corresponding abdominal pain. This can exert a significant influence over his ultimate treatment preferences – pain is a significant motivator in electing to forego medical treatment. Harry still faces a dependent existence; his

daily routine is tied to the schedule and availability of his care staff, and his breathing is likely only to worsen. He may be unsatisfied with the quality of his life, a topic that is addressed in he psychometrics discussed earlier in the chapter, and certainly fecund ground for discussion in dialogic interaction. As his health worsens, he may view breathing therapies to be burdensome, which then raises the question as to whether he ought to continue with the treatments, or shift his attention towards palliative care. If Harry were to elect to forgo treatment at this point, there is a significant amount of evidence suggesting that this is reasonable, and not necessarily the product of cognitive distortion. The clinician should approach him and discuss the values and goals that are meaningful to him, and then to offer treatment modalities congruent with those preferences, whether potentially curative or palliative. Alternatively, Harry may elect to continue to receive breathing treatments when they are no longer of any real medical or psychological benefit – at this point, it would be appropriate to discuss with him reasonable versus unreasonable expectations of therapy, and why he may feel that continued 'treatment', if that is even the appropriate term, is warranted.

The third variation removes all of his medical comorbidities except his pancreatitis. He maintains his mobility, no longer has his breathing difficulties, but still experiences severe abdominal pains. The question at this point becomes pain management, and a variety of treatment options exist, whether in the form of medications, analgesic patches, nerve blocks, etc. The standard of care would require an examination of his lifestyle choices to see whether there are any exacerbating elements (e.g., alcohol abuse), but pancreatitis is a manageable condition. If he were to elect to forgo treatment at this point (say for an acute illness), it is reasonable to suspect that his cognition is being influenced unduly, and therefore the decision to forgo treatment ought to be challenged. Pain can be a powerful motivator, and in some cases, can

require dosages of analgesic medication in the toxic and lethal ranges to manage it; it is not the purpose of this case variation to suggest that pain alone is insufficient grounds to forgo medical interventions, but it certainly should raise cautionary flags and suggest avenues of dialogue about treatment.

Case #9: Irene

The last case in the metric is Irene, a 70-year-old Hispanic female who presents with pancreatic cancer. Irene is no stranger to cancer, having gone though skin and colorectal cancer earlier in life. She has endured several surgeries and rounds of chemotherapy and radiation in the process. The pancreatic mass was discovered quit recently, but it was discovered to have metastasized to her lymphatic system. Irene has made it quite clear that she does not want further medical interventions beyond palliative care – she states that she is "too old and frail to have to go through all that nonsense again." Her treating clinician felt that it was his responsibility to discuss potential therapeutic interventions, as well as palliative measures.

Demographics, Personal and Family History

Irene is a middle class former schoolteacher. Aside from her multiple cancer scares, she is in quite good health. She walks frequently, and socializes with other cancer survivors. She has two younger sisters, along with numerous nieces and nephews. A hysterectomy early in life prevented her from having children of her own. Her husband died the previous year from pneumonia.

Irene has been very vocal and opinionated throughout her life. She developed a reputation for voluminous research and meticulous attention to detail in topics that interested her. This drove her to academic success, where she earned Master's Degrees in both Education and Biology. She taught science classes in many grade levels, and was quite resourceful for her

honor's students. She retired late, arguing successfully that while her body might be slowing down, her mind was as sharp as ever, a point demonstrated unequivocally by questioning by the school board.

After her skin cancer diagnosis in her mid-thirties, she focused her considerable faculties on understanding the disease. She read information generally available, as well as delving into some of the trade publications for research articles. She maintained a library on cancer in her home that would rival that of some clinicians. She felt that by understanding the disease, she could exert some mastery over it – "know thy enemy" as she related to her treatment staff. She experienced remission and recurrence of the skin cancer over the following decade, but it seemed to go into a permanent remission in her mid-forties.

As she approached fifty, she noticed that she was experiencing recurrent hemo-positive stool. She became alarmed, knowing that it was potentially symptomatic of colorectal cancer, and promptly underwent examination and biopsy. When it was confirmed to be malignant, she underwent several rounds of treatment which left her tired, weak, emotionally and physically exhausted, but victorious and in remission. She would experience a recurrence five years later, and underwent further rounds of chemotherapy.

Her diagnosis shook her at first – her interest in understanding cancer had not waned as she grew older, and she recognized the significant mortality rate present in the pancreatic form. She had taken stock of her situation, and decided that at this point in her life, it was more important to her to feel comfortable than to survive – although she was still active, her age was wearing at her spirit. She tired easily, and felt that while she might be able to give a good fight, she no longer possessed the physical and emotional resources she had in her youth.

Condition and Prognosis

Irene was diagnosed with stage II pancreatic cancer. Her oncologist predicts that her chances of five-year survival are approximately 1%.

Case Treatment

Irene's clinician is concerned with Irene's adamant refusal of potential medical intervention; he suspects that there may be a psychological comorbidity affecting her decision. He contacts the psychiatric consult liaison, who evaluates Irene. Noting little obvious depressive symptomatology, the consult liaison elects to cover all the bases and asks the clinical psychologist to evaluate her. The clinical psychologist administers the Hospital Anxiety and Depression Scale, as well as the Geriatric Depression Scale and the Dysfunctional Attitudes Scale.

Test Results

All three scales indicate subthreshold depressive symptoms. The GDS and DAS scores are higher, but the clinical psychologist suspects that these may be false positives due to the somatic content of the some of the scale items.

Dialogic Content

Irene notes that while she understands that her clinician is concerned with promoting the well-being of his patients, he may be ignoring her experience and knowledge of cancer. She reiterates that she has gone through medical interventions for the disease in the past, and found them to be quite taxing on her mind and body. She notes that she doesn't have the same resources and strengths once available to her – she is a lot older now, which has affected her strength and resiliency. She had a difficult time bouncing back from her previous illnesses, and believes that were she to attempt it again, that those difficulties would be compounded.

Heuristics and Biases

The only potential sources of bias that seems apparent are potential applications of the availability heuristic (drawing upon the most salient information) as well as potentially misremembering her previous experiences with illness. Neither of these, however, seems particularly likely – a recurrent theme in Irene's case is her efforts to understand both the illness and her experience of it. This suggests that any information which may exert influence would likely be objectively accurate and relatively free from personal bias. As such, there appears to be little heuristic ground for suggesting diminished personal autonomy.

Authenticity

A key component of Irene's value structure is her evident understanding of herself and her illness. Throughout her life, she has made an effort to research and explore her areas of interest, amassing information and organizing in a manner that makes it highly relevant to her. It is clearly important to her that her analysis of her situation be respected – aside from the abstract knowledge provided by years of reading and research, she has personal experience which are quite salient. She defines herself through her understanding; any recommendation, therefore, must take this into consideration. Her voiced preference to forgo further interventions is based upon a thorough understanding, not only of the disease in question, but of her own abilities and resources. Her choice to forgo treatment reflects this understanding and assessment, and as such, is congruent with fundamental aspects of her self-identity. As a result, there is little ground for suggestions of diminished autonomy based on authenticity.

Accuracy of Self-Perception

It seems quite evident that Irene is aware of her condition, her quite likely prognosis, and her self. She seems quite aware of the effect age has had on her ability to recoup – she

understands that who she is now is not who she was the last time she fought the battle. She knows what the fight requires, and knows that she will come up lacking. While one may raise the question as to the objective accuracy of this self-knowledge, it is congruent with the available evidence, making her self-judgments quite reasonable and defensible. As such, there is little reason to suggest that her autonomy has been compromised due to lack of accurate self-perception.

Resolution

Irene elected to refuse medical intervention for her pancreatic cancer, and was referred to hospice care. Her growing pain was managed with morphine, and five months after her diagnosis, she died peacefully in her sleep.

Variations

The first variation on Irene's case shifts her pancreatic cancer to an earlier stage. This carries with it an increased chance of five-year survivability, and may make treatment less taxing on her physically and emotionally. This is not to suggest that it will be easy, however, it does raise the possibility that her risk/benefit analysis of treatment may change, which is an important consideration. If her survivability were increased to 40%, she would be remiss to dismiss a therapeutic intervention out of hand. This is not to say that she would be incompetent or that her autonomy has necessarily been compromised, but dismissal of that significant of an improvement ought to raise cautionary flags in the assessment of her autonomy. As has been noted in earlier cases, an inability to recognize significant differences in probable treatment outcomes suggests a compromised cognitive process.

The second variation provides a bit of a twist – instead of electing to forgo treatment, Irene now insists upon it, despite the risks and very low five-year probability of survival. This

suggests several different avenues of approach. On the one hand, there may be a meaningful future event in her life that she wants to witness, and as such, is willing to take risks that have small chances of success. For instance, she may desire to witness the birth of a relative, a wedding, a graduation, etc.[68] This is reasonable, and has occurred frequently in clinical practice. As such, it would be remiss of a clinician to dismiss out of hand a patient's desire to pursue treatments with diminishing probabilities of success. On the other hand, a clinician must be cognizant that sometimes patients seek out treatments because of irrational expectations – they are 'hoping for a miracle.' These cases are difficult, in that hope can be a powerful motivator, and can help to ward off psychological comorbidities (e.g., depression), which has been demonstrated to affect morbidity and mortality. However, it is important that a patient have reasonable expectations in treatment outcomes – entertaining impossible outcomes as serious possibilities ought to raise a cautionary flag in assessing autonomy. If a patient maintains an unreasonable expectation in the face of mounting evidence to the contrary, the clinician should suspect that some element of the patient's autonomy has been compromised, and engage in the necessary dialogic interaction to explore the patient's thought process. As has been argued earlier, it is important to address as many identifiable sources of error as possible in light of the decisions being made.

The third variation on Irene's case changes little of her case except her source of information. It is entirely possible that Irene has found incomplete or inaccurate information regarding cancer, and is using that information in making her choice.[69] She is highly intelligent,

[68] This is not an uncommon desire – I have both worked with patients and have followed patients in the literature who have found personally meaningful events in the future which they desired to experience. A case local to Pittsburgh involved a man with advanced illness who wanted to remain alive long enough to see his son graduate from high school. He was able to witness the ceremony, and died shortly thereafter.

[69] This is a significant concern in the contemporary "Information Age" . The World Wide Web allows individuals to access an extraordinary amount of information, but does not use uniform standards of peer review or critical appraisal of the information provided (if the website in question does any fact-checking *at all*). I have worked with

but may be led astray. There is a clear external source of error which has translated into systematic internal errors in her cognition. As was discussed in Chapter Two, backstage cognitive processes build new cognitive schemata from existing data. If the data is questionable to begin with, the resultant constructs (which become reinforced and automatic over time) are equally questionable. Any decision to be made, therefore, must make clear what is accurate versus inaccurate, and as a result, what constitutes reasonable versus unreasonable expectations. Her decision to forgo medical treatment in this scenario may be made for the inaccurate reasons, and as such, ought to be questioned, even if the outcome ends up being identical. While the end result is the same, it makes a significant amount of difference how one reaches it.

Conclusion

What emerges from these cases is a sliding scale of influence and potential of compromised autonomy. The cases have been summarized and scaled below, in an effort to indicate quickly and clearly the conditions in which treating clinical staff should suspect that a patient's autonomy has been compromised.

CASE METRIC OF INFLUENCE ON AUTONOMY								
SIGNIFICANT INFLUENCES			MODERATE/ MINIMAL INFLUENCES			LITTLE/ NO INFLUENCES		
1	2	3	4	5	6	7	8	9
Alice	Bill	Catherine	David	Eugenia	Frank	Georgette	Hal	Irene
Autonomy Compromised			Autonomy Possibly Compromised			Autonomy Not Compromised		

This scale is not meant to serve as the sum total of evaluation – i.e., it is not meant to supplant the role of the consult liaison or clinical psychologist. It is meant, however, to note those instances in which clinicians should raise the evidentiary standard of autonomous choice when a

patients who have entertained ideas gleaned from questionable sources in both print and electronic formats which have defied what we know about anatomy, physiology, and biochemistry.

patient elects to forgo potentially beneficial treatment. Personal autonomy is a relevant and meaningful concept in medical ethics for many compelling reasons; however, it is a very complex phenomenon, and requires great attention to detail for it to be genuine. Autonomy can be a mask, an attractive veneer placed over incomplete and unrealistic cognitive models, an ethical 'out' when situations require more analysis than the dominant philosophical model can muster. If we genuinely want our patients to make autonomous decisions, we must attend to as many relevant details of that decision as we possibly can – the opportunity for error and the finality of the decision warrants nothing less.

CONCLUSION

A significant amount of ground has been covered in the preceding chapters in a number of fields. It has been demonstrated that consciousness, our day-to-day perception, our sense of self and identity, judgment, emotions, and intuitions are all predicated upon a number of causal cognitive elements that are outside our awareness – the bulk of our cognition is deterministic and preconscious. This determinism opens up avenues of undue influence into processes we normally assume to be under our control – it should be clear that this assumption is mistaken at best, inhuman and pernicious at worst. We should not abandon ourselves to blind determinism, however – we possess the ability to reflect upon our motivations, and to engage in dialogic interaction with others, who may bring aspects of ourselves to the fore which would remain otherwise inaccessible. As a result, we can take back a measure of control, but only if we engage in honest dialectic and dialogue with others.

In the context of patient autonomy and decision-making, the necessity of this dialogical process is especially evident – patients are already physically compromised, potentially in ways that can exert conscious and unconscious influence over their decision-making processes, above and beyond the normal potential sources of error found in heuristics and biases. Any clinician who genuinely cares for the welfare of her patient will be alert for such influences, recognizing that a medical illness can easily mask a deeper psychopathology. Affective disorders are very common, occur more in patients than in the general population, and tend to go unrecognized or dismissed as a normal reaction to their illness. The effect of these disorders, however, is quite pernicious. They fundamentally affect the efficacy of therapeutic interventions, morbidity and mortality, and rate of recovery – ignoring, dismissing, or failing to identify a comorbidity

compromises the treatment of the obvious illness. By only treating the surface pathology, we potentially ignore the deeper wound.

Many contemporary models of autonomy suffer from similar shortcomings – while ethics seeks to inform itself of philosophical, legal, theological, and medical constructs, it all too easily ignores the psychological, an unfortunate irony in light of the fundamental connection between cognitive and clinical psychology and ethical ideals of autonomous choice. Ethical theories that dismiss or fail to address psychological constructs are simply so much story-telling; models derived from inhuman absolutes are so much fancy and fiction. What good is it to describe models of cognition that have little resemblance to how we actually think?

The present autonomy model suggests that decision-making is a complex construct necessarily containing rational and emotional elements, intuitive judgments, and, as a result, potential sources of error. This seems to gel with day-to-day experience – many decisions are made by gut instinct and intuition, instead of a Cartesian rational process methodically and algorithmically exploring all possible influences, outcomes, and variables. This deterministic model gels with the phenomenon of basing day-to-day decisions upon distal causes – early education and environment, role models, learned behaviors, etc. This model suggests that as the severity of the outcomes increases to terminal, increasing reflection upon the causes and motivations of the decision is required – that a genuinely autonomous choice will explore the agent's motivations, identifying and judging the appropriateness of each influence, determining if it is congruent with the value system adopted by the agent as a whole. Decisions stemming from inauthentic elements of the self fundamentally are not expressions of autonomy; if a patient is forgoing treatment, whether to avoid suffering or actively to choose death, we would be remiss not to ensure that it is *her*, and not *her pathology* making the choice. Anything less would

surrender autonomy to expediency, would surrender authenticity to apathy, and would surrender insight to obfuscation. The capacity for self-reflection appears to be a defining characteristic of being human – we would do well to use it when we face terminal choices.

APPENDIX: PSYCHOMETRIC SCALES

HOSPITAL ANXIETY AND DEPRESSION SCALE

This metric can be found in:
"The Hospital Anxiety And Depression Scale."
Acta Psychiatrica Scandinavica.
1983; Volume 67, pages 361–70

AUTOMATIC THOUGHTS QUESTIONNAIRE

DYSFUNCTIONAL ATTITUDES SCALE

GERIATRIC DEPRESSION SCALE

These metrics can be found in:
Measures for Clinical Practice: A Sourcebook (3rd Edition)
Joel Fischer and Kevin Corcoran
New York: The Free Press, 2000

BECK DEPRESSION INVENTORY

Harcourt Assessment, Inc.
19500 Bulverde Road
San Antonio, Texas 78259-3701
1-800-211-8378 Customer Service
1-800-228-0752 Administration

HAMILTON RATING SCALE OF DEPRESSION

This metric can be found in:
Sourcebook of Adult Assessment Strategies
Nicola S. Schutte, and John M. Malouff
New York: Plenum Press, 1995.

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