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Editorial

The pattern of journalism in this publication has been that of selecting an area of medical interest, researching it thoroughly, and bringing together a dissembled mass of knowledge into a clear, concise, logical, and lucid abstract. Ideally, the result should arouse the interest of both undergraduate and physician alike. Why has this not been the effect? Article writing develops the art of self-expression, encourages clear and precise thought, and is a good way to learn: however, only a few of the undergraduate body take the opportunity so freely offered.

A recent article in *The Lancet* by N. R. E. Fendall (January 29, 1966) reflects on the problems of the growing body of medical publications. He asks, "How much of all that is written and published is of genuine interest to the reader, and how much is merely repetitive? How much actually contributes to an advance in knowledge? . . . General medical journals will before long need to abandon original papers in favour of articles reviewing recent discoveries and their application. Much of what is written is purely in the interest of the author: Maegraith captures the picture neatly in his article, *The Gekochteundgebrocheneeirschale Phenomenon,* lampooning pretentiousness and futility in publications." Perhaps it is this sense of self-indulgent futility which first besets the retiring freshman medical student during the acquisition of a mass of basic science facts (rather than concepts) from the fifty pound standard text-book, facts which he discovers can only with great difficulty be applied in the diagnosis and treatment of diseased human beings, facts which, save for examination purposes, are best relegated to the limbo of the lab.

Organization and classification are abilities acquired by the mature investigator. The organized approach is essential for good medicine, for by it we achieve clear vision, critical analysis, and honest judgement. As a profession we are all student investigators who tend to be impatient and disinterested in the muck of irrelevant detail. Most of us leave to our teachers the problem of digesting the morass of journals and textbooks and of procuring some practical bit of understanding for the student. The student is required to regurgitate these pearls at examinations whether or not he understands and can apply them: the art of observation and critical assessment of what a lecturer or patient presents for our digestion is not taught. Instead, the patient's symptoms must be made to fit a text of *the* book or *the* lecture. The ingraining of an attitude of uncritical acceptance has made reading and learning a boring pastime. and the effort required for lucid writing an unappetizing chore. Isn't it time for a change?

The intangible quality of psychiatric illness demands an enthusiastic, imaginative, and critical approach. The topic of this journal, "mood disorders", is of this nature. The articles in this journal are four. *Mood Mechanisms* discusses and applies recent experimental data in defining the esential clinical features a mood disorder might show. *An Overview of the Depressions* deals with a syndrome often seen in routine medical practice, depressed mood. *Adolescent Behaviour Disorders* discusses some of the emotional problems to be met by the practitioner dealing with the adolescent group. *The Plastic Personality* reveals the problems of the integrative maturation of the psyche in the child, correlating mood and "self".

I would like to thank Dr. G. E. Hobbs, Professor and Head of the Department of Psychiatry and Preventive Medicine, for perusing these articles and offering helpful suggestions as to content and style. Appreciation is also extended to Miss Marg Demeny of the U.W.O. Medical Art department for the diagrams included in this issue.

MECHANISMS OF MOOD

by DAVID A. CLARK '67

INTRODUCTION

The purpose underlying this article is to discuss the neurophysiologic basis for mood and affect, and to use this information to analyze the primary role of emotional or affective imbalance in the genesis of certain psychiatric disorders. The major impetus for attempting such an approach arises from several areas:

Firstly, there has been a change in the orientation of psychiatric practice since the discovery of the psychotropic drugs. These are available for use by those who know as little about them as the drug salesman's pamphlets tell them. The fact that these drugs work has led to their wide use on an empirical basis. Like most of therapeutics in psychiatry, how and why they work and when they should be used is not clearly defined. Compounding this problem is the commercialism which pervades the manufacture and distribution of these new drugs. To support claims of the superiority of drug A to drug B, voluminous pseudoscientific documents deluge the office of the physician. "The indication for our drug is any patient presenting as a fierce Macabaw monkey!" has been one pitch. Much of the information provided by these pamphlets attempting to explain the mechanism of the drug by its action on some vestigial nucleus is dodging the basic issue of mechanism. Most of the information thus provided is confusing and irrelevant, for no one really knows what it all means. The task is sizable for anyone hoping to glean for himself a model of mental function to explain why a certain set of symptoms is pathognomonic of a specific disease meriting a specific drug or drugs.

Second, is the problem of organicity in mental disease which has produced two opposite camps, the organic psychiatrists and the analytical psychiatrists. The analytic group has built a model of mental function and malfunction by drawing upon the observed secondary or intellectual elaborative processes of mental illness. The patient on the couch is an intelligent creature who can think, feel, behave, and communicate, and his abnormal functioning can be "explained" in terms of "well-understood" psychological models. The "cause" of mental illness is therefore bound up with intercurrent psychological problems and the defence mechanisms mobilized to protect the ego or self. The organically oriented group, on the other hand, quote proven examples where an organic lesion caused a set of symptoms identical to a so-called functional illness. They therefore argue that the majority of mental disorders will be eventually shown to be caused by metabolic defects. If we look on the biochemical level, surely all mental illness will have an organic basis. There is little doubt that organic mechanisms must subserve all normal and abnormal brain function, and whether a lesion affects a certain set of interacting pathways or whether a functional disorganization of these pathways takes place is immaterial to the symptoms of the resulting disease. For example, cardiac fibrillation may be caused by such factors as, electric shock, toxic sensitization (epinephrine, hypercarbia, and chloroform), or an irritable focus of decaying tissue following an infarct: the outcome is the same in terms of immediate symptomatology. Although the primary defect leading to the disordered electrical physiology may vary, therapeutics aim at restoring the physiology to

normal. It is obvious that either organic and functional illness may masquerade as the other, and that a clear understanding of the pathophysiology will, in each case, lead to a better understanding of the etiology of mental illness. Insofar as the functional aspects of illness are the most nebulous and poorly characterized, the examiner must first rule out the "organic", especially in the prodromal stages.

Thirdly, over the past fifteen years, neurophysiology has begun to delineate the intriguing mental mechanisms and pathways with which we are concerned clinically. Within this framework, the divergent approach of the organic psychiatrist and the analyst may be resolved, psychiatric disorders may be established on a more scientific basis, and the proper therapeutic regimen for such diseases will follow logically.

DEFINITIONS

In order to discuss the scientific basis of clinical terminology, we must first define the meanings of these terms.

(a) Emotion

Emotions may be regarded as abstractions constructed by the perceptual processes upon a basic affect, and incorporated into the conscious processes. The ever insistent perceptual organizing tendencies of the intellect make the emotional experience sensible with respect to the surrounding situation. An emotion is a transient experience that need be attached to cognitive processes and, if it is not, a situation is invented so that emotion will make sense: i.e. small frustrations lead to emotional experience of overwhelming anger and this is blamed on the next antagonist who comes along, thus giving 'rhyme and reason' to the outburst.

(b) Affect

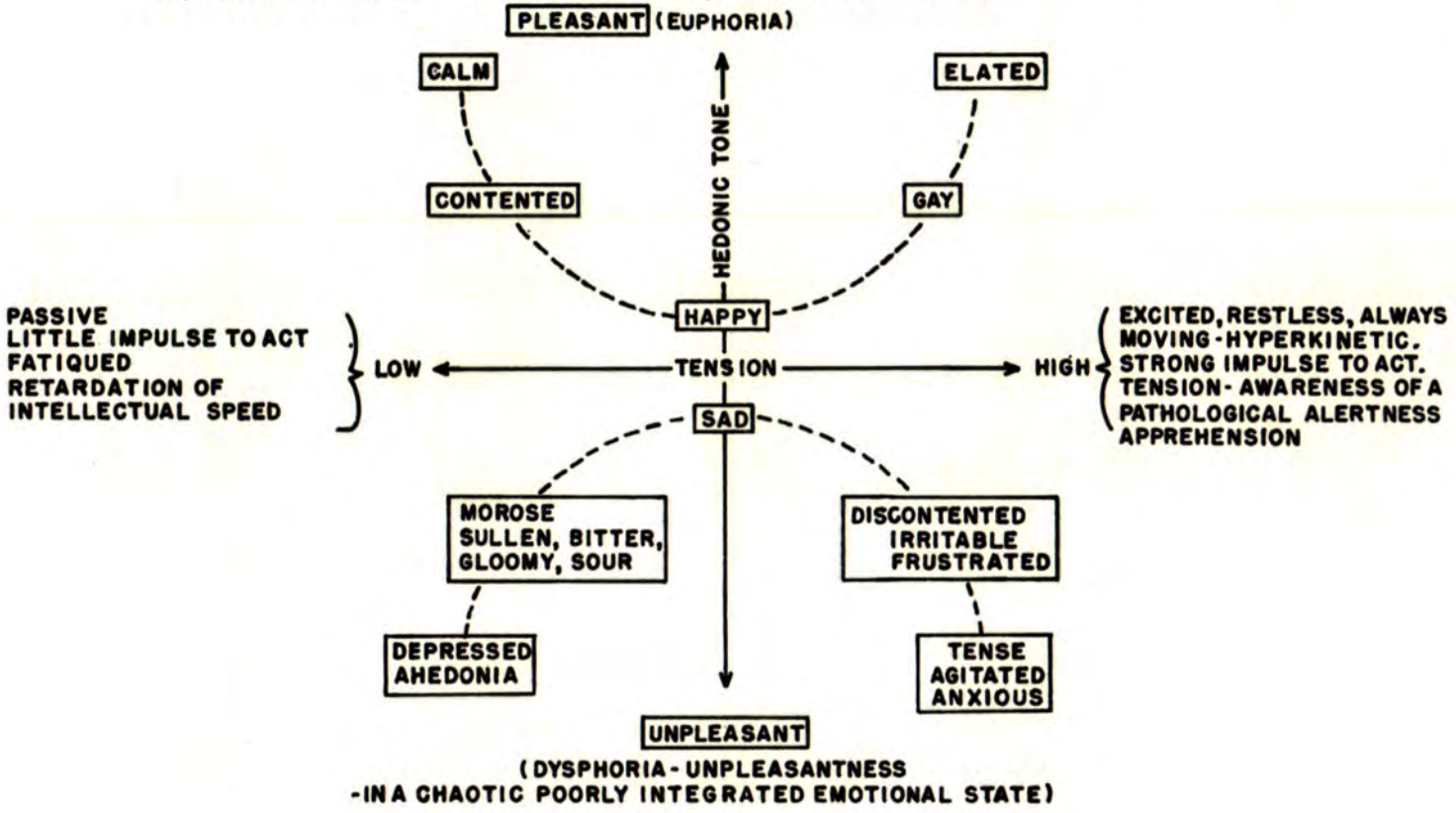
Affect is the feeling tone from which the emotion is constructed by the perceptual or cognitive apparatus. Affect is a less transient physiological quality with autonomic and motor components. It provides directives to the cognitive processes as to what qualities the emotional experience must have. Physiologic upsets, such as produced by injection of adrenaline, may produce what is called "cold emotion", lacking in genuine emotional quality: a person may feel afraid, yet he is calm. However, were there ongoing emotional processes or a constant infusion of the drug, alteration in the qualities of the affective colouring would reorient the perceptions and emotions appropriate to the altered affective colouring. This may be the case in the mental disorders associated with Cushing's syndrome, Addison's disease, thyrotoxicosis and pheochromocytoma.

(c) Mood

For the purposes of this discussion, mood may be regarded as a physiologic state giving rise to affects of similar tone. The sum total of the current pattern of affects ongoing in the person establishes, for the clinician, the nature of the mood. While emotions arise in relation to conscious experience of the outside world, there is little awareness of the basis of moods in the concrete aspects of situations, only an awareness of control which moods may have over the quality of those emotions. Since mood is to be dealt with as a physiologic state, a feature common to all physiologic regulations may be introduced, the concept of homeostasis. A traditional concept states that there is a baseline about which regular fluctuations of mood occur in a cyclothymic manner. Fluctuations may be of a greater or lesser magnitude, depending on the person being examined, and so mood in that person may exhibit more or less cyclothymic variation and more or less stability. As well, the baseline in any individual may be above or below the average norm. All these factors in temperament may be represented in two dimensions as shown in the diagram[1]. An individual has a "homeostatic point" rather than a "baseline" about which his moods

VECTOR-GRAM OF MOOD

THE HOMEOSTATIC POINT IS THE NET RESOLUTION OF ALL VECTORS OVER A 24 HOUR PERIOD. DISTURBANCES FROM NORMAL PRODUCE WHAT IS KNOWN AS A "FUNCTIONAL SHIFT". CHRONIC DISTURBANCES LEAD TO SECONDARY PERCEPTUAL ELABORATION. AT THE EXTREMES, THE RANGE, VARIETY, AND USEFULNESS OF AFFECT IS DIMINISHED, AND THE PERSON MAY BE TRAPPED IN A UNIDIRECTIONAL PATTERN OF BEHAVIOUR, FEELING OR THINKING.



fluctuate. The two dimensional concept has important neurophysiologic implications as will be seen. A third element in this scheme to be kept in mind is cybernetic control or feedback regulation, but a thorough discussion of this feature is beyond the limits of this article.

NEUROPHYSIOLOGIC CORRELATES

Anatomy

Evolutionary process characteristically builds upon and modifies anatomical and physiological substrates which have gone before. With this in mind, certain principles in the organization of the more primitive components of the brain may be outlined. The primitive vertebrate brain consisted of a longitudinal cylinder containing a central tube of grey matter encapsulated in an outer sheath of white matter from which the primitive spinal nerves departed towards the periphery of the body. In the encephalization process, certain features of the primitive brain have been maintained: firstly, the cranial nerves maintain the pattern of primitive spinal nerves; secondly, the brain stem has retained the core of central grey matter. In man, this lines the aqueduct of Sylvius and spreads forward into the third ventricle to form the medial thalamic and hypothalamic nuclei. About this central grey, organized in vaguely delineated paralled longitudinal columns, lies the reticular formation, - a central transactional core between the strictly sensory and strictly motor systems. Central areas of the midbrain tegmentum constitute a major termination point for ascending reticular pathways. At the caudal border of the thalamus, this non-specific pathway bifurcates, the dorsal limb turning into the intralaminar cell groups of the thalamus (between medial and lateral nuclei), the ventral limb turning into the subthalamus and lateral hypothalamus, preoptic area and medial septal nucleus.

In general, influences extend upward from the midbrain to arouse the waking animal and to change brain wave patterns from that of a sleeping to a waking animal, and downward to control sensory thresholds and patterns of neuromuscular tone.

The dorsal limb of the reticular formation serves to filter out unimportant stimuli at the thalamic level, thus serving to direct attention. Thus, "the function of the dorsal limb of the reticular system in normal adaptive behaviour may be in the nature of prevention of general arousal to all stimuli with a control of selective responses to significant stimuli".[2] The cardinal psychological change produced by arousal is called the arrest reaction, a central excitatory state depending for its intensity on reticular activity, peripheral stimuli, cerebellar stimuli, and stimuli from temporal cortex. Interference of the ongoing function of the thalamic reticular formation occurs: the animal shifts to a state of alertness and attention, preempting existing circuits and suppressing selectively all incoming sensory information. Prolonged arousal leads to tension, anxiety, and hypomania. Also, representative of the filtering function is the habituation reaction. This is a form of adaptation to sensory stimuli so that they are ignored rather than examined in minute detail as occurs in the arrest reaction. Studies of the filtering process have shown that arousal may be produced by stimulation of thalamic neurones which inhibit other areas of the thalamus: this effectively suppresses irrelevant sensory input by selective inattention. In the EEG, the alpha rhythm or so-called "neuronal shutter" is replaced by a beta activity of desynchronization, an alteration probably accomplished through cortical inhibition. While this reduces sensitivity of the cortex, it increases specificity to specific signals, increasing the signal-to-noise ratio. Concomitant with this is an increase in the ability of the brain to discriminate between rapidly presented similar sensory stimuli. Excessive arousal due to extreme emotional responses may desychronize to the point of intellectual impairment;

awareness is restricted, attention divided, confusion, inefficiency, and disorganization result.

The ventral bifurcation is vitally important in the genesis of affect, mood, motivation, learning, drive, and the control of the associative processes as directed by memory and emotion. Ascending pathways from the reticular formation enter the lateral hypothalamus, and pathways from the limbic system descend through it by long and short fibre tract systems.

Removal of the olfactory forebrain or rhinencephalon in fish has been shown to produce apathy and loss of initiative, or loss of nose brain control over the behaviour of the beast. In monkeys, removal of amygdalae bilaterally has produced a passive hypersexed animal. Certain investigators feel that the rhinencephalon subserves mutually antagonistic self versus species survival systems. The amygdala is part of the self survival system: electrical stimulation produces defence reactions, aggressiveness, increases tension, reduced gonadotropin and increased ACTH output. The fornix, cingulate gyrus subserve species survival; stimulation has produced pleasurable moods, a decrease in tension, rise in gonadotropins, fall in ACTH, and reproductive behaviour. More research has tended to contradict this view. What can be said, in fact, about the rhinencephalon, is that it appears to afford head brain control over the drive systems of the lateral hypothalamus: the amygdala increases hypothalamic activity and hence, increases drives and affects originating in this structure. The septal area, to which the formix contributes afferents, damps down hypothalamic activity. Reciprocal connections exist between the amygdala, cingulum, temporal lobe cortex and orbitofrontal cortex, septal area, mammillary bodies. The function of the hippocampal cortex is surmised from the fact that bilateral removal in man has led to striking defects in memory. During a conditioned learning process, large amplitude low frequency waves arise in the hippocampus and spread to the entorrhinal coxtex: similar potentials spread in the reverse direction during the forgetting or unlearning process. This would imply that one important function of the hippocampus is memory.

McLean proposes the term "schizophysiology", to represent the dichotomy of function between the neocortex and the rhinencephalon. He feels that the two are independent, that the rhinencephalon reacts as a primitive writhing impulsive reflexive animal, outside the incisive control and awareness of the abstract mind, subject to little of its control. To substantiate this, he cites some of the irrational affective disturbances of limbic epilepsy, - powerful feelings of hunger, thirst, nausea, suffocation, choking, retching, coldness, need to defecate and urinate and feelings of terror, fear, sadness, familiarity, strangeness, unreality, wanting to be alone, persecution. These represent strong *mood* disturbances sometimes schizophrenia may be simulated. Organic disturbances mimicing functional psychoses suggest common physiologic pathways underlie both. Certain objections must be made to regarding the limbic system and hypothalamus as functionally separate from the neocortex. Deep electrode studies of epilepsy have revealed cortico-hippocampal connections to the posterior frontal, anterior parietal, and temporal lobes. Hippocampal activity may aggravate epilepsy. Similar connection from other parts of the rhinencephalon (i.e. amygdala to frontal lobes via thalamus) also exist. The cingulum communicates with the amygdala and hippocampus, and intracortical association fibres spread from the cingulum to other parts of the cortex. The frontal lobes are reciprocally connected to the hypothalamic nuclei through the dorsal medial thalamic nucleus and lesions of this nucleus and lesions of the pathway to the hypothalamus produce severe apathy, loss of affective responses, and loss of anxiety feelings.

Therefore, the concept of independent functioning of the neocortex and limbic system is not substantiated. Just as the dorsal reticular formation is vital to consciousness, the hypothalamus and the central reticular bifurcation appear vital to emotional consciousness and integration.

SUBSTRATES OF MOOD

The bulk of the preceding discussion suggests that experimental observations have produced only more confusion in attempting to understand mood. One of the major discoveries made within the past twenty years is that there are primary areas subserving pain and pleasure within the hypothalamus.

Olds[3] states that three basic affective systems are present:

(i) those areas giving negative reinforcement or pain,

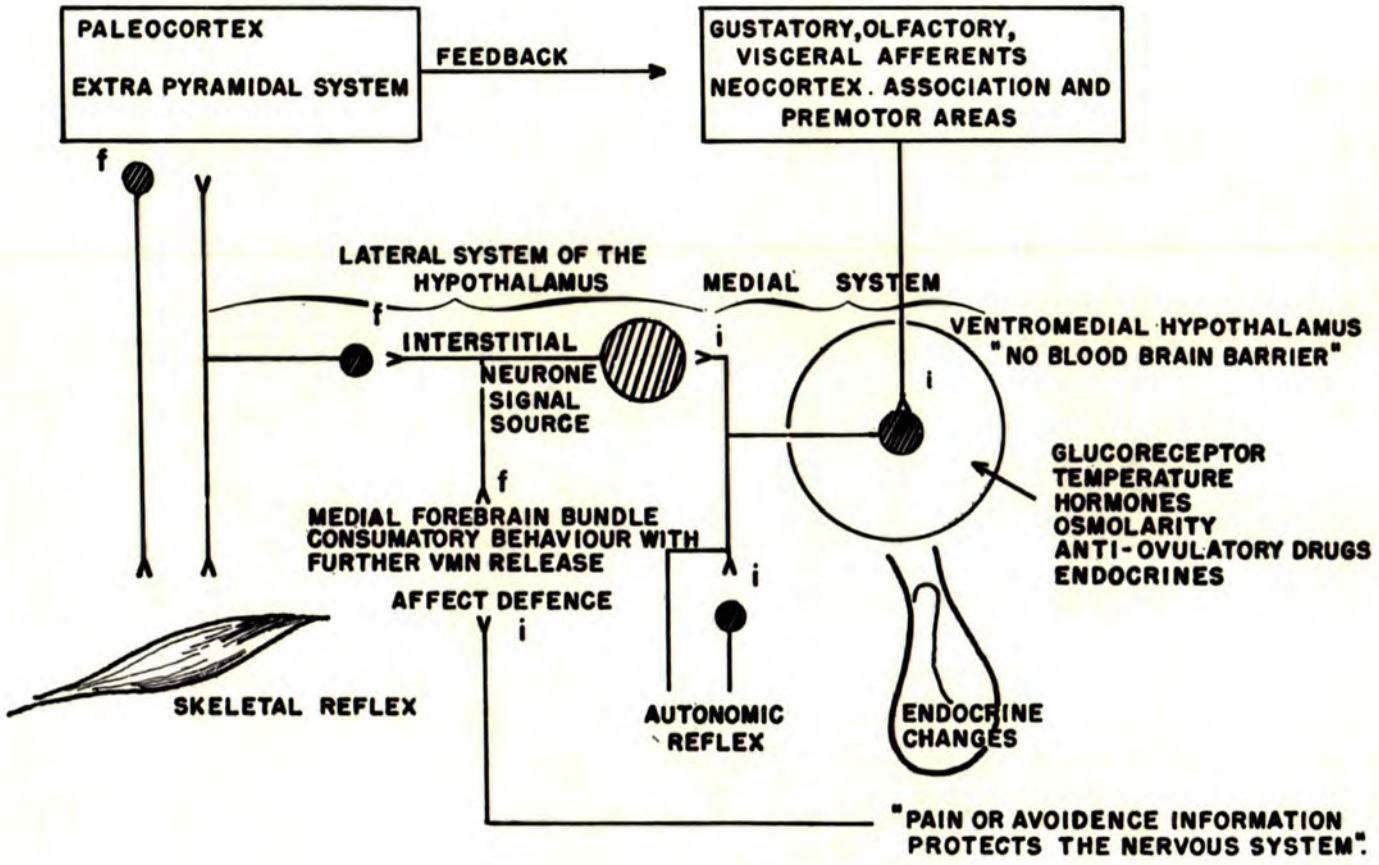
(ii) a physiologic arousal system producing sleep, alertness, tension, and extreme tension, having neutral affective tone and no motivational effects.

(iii)those areas giving positive reinforcement or pleasure.

The reward zone is present as a U shaped area with the base of the U in the ventral midbrain and the two arms extending parallel to the central canal into the lateral hypothalamus as the median forebrain bundle. When electrodes were inserted into this tissue, animals would work to self-stimulate themselves at rates up to 20,000 bar presses per hour, highest rates being obtained from the lateral posterior hypothalamus where the animal would self-stimulate to exhaustion, foregoing all else. In other areas such as the septal area, orbital frontal cortex, less marked responses have been obtained and animals will self-stimulate only to a certain maximum number of bar presses in one day, no matter how long the animal is left on the circuit: thus the animal satiates in these areas and will not be dropped in an unidirectional pattern of behaviour leading to exhaustion. Very extensive investigation of this area has shown that the effects of stimulation is not simply limited to motivation and pleasure. Discriminitive learning (i.e. learning to discriminate between two similar auditory or visual stimuli) has been investigated. Animals learn best under conditions of indiscriminate negative reinforcement and when reward stimuli are given, disruption ensues. This is felt to be a real confusion in the associative processes and illustrates the control exerted by this area over associative processes that guide mental behaviour at points of choice. Olds[4] has reported cortical learning associated with self-stimulation. Electrodes were inserted into reward areas of the hypothalamus and the rhinencephalic cortex. Pulses were delivered to both electrodes, and recordings from the rhinencephalic electrode made. A striking increase in the discharges recorded was noted: repeating the reinforcement alone reactivated a shower a local discharges from the rhinencephalic site. Apparently a form of local learning had occurred. No similar phenomena could be demonstrated in the neocortex, which apparently is left to circumspect and evaluate, at its leisure, incoming sensory information, to consider past experience, and to react in many more possible ways to the perceptual data.

The avoidance area extends slightly dorsal to the pleasure area in the region of the thalamus, and caudally back into the midbrain tegmentum. Continuous stimulation produced cowering, biting, hostility towards the experimenter, withdrawal into a corner, and refusal to eat. This could be reversed by putting the animal on the reward circuit. In the region of the hypothalamus, the "affective defence" portion of this area has been defined by the aggressive, active, self-defence behaviour exhibited by animals when stimulated here.

Much evidence assembled by Olds[5], suggests that the integration and control of these affective substrates is of the nature


f - FACILATORY
i - INHIBITORY
PALEOCORTEX
EXTRA PYRAMIDAL SYSTEM
FEEDBACK
GUSTATORY, OLFACTORY,
VISCERAL AFFERENTS
NEOCORTEX. ASSOCIATION AND
PREMOTOR AREAS
LATERAL SYSTEM OF THE
HYPOTHALAMUS
MEDIAL SYSTEM
INTERSTITIAL
NEURONE
SIGNAL
SOURCE
VENTROMEDIAL HYPOTHALAMUS
"NO BLOOD BRAIN BARRIER"
GLUCORECEPTOR
TEMPERATURE
HORMONES
OSMOLARITY
ANTI-OVULATORY DRUGS
ENDOCRINES
MEDIAL FOREBRAIN BUNDLE
CONSUMATORY BEHAVIOUR WITH
FURTHER VMN RELEASE
AFFECT DEFENCE
SKELETAL REFLEX
AUTONOMIC
REFLEX
ENDOCRINE
CHANGES
"PAIN OR AVOIDENCE INFORMATION
PROTECTS THE NERVOUS SYSTEM".

of a double inhibition of the reward areas. (see diagram).

Interstitial neurones of the median forebrain bundle (reward centre) are spontaneously active: their discharge rate is influenced by local ionic balances, drugs, neuromodulators and by inhibitory neurones. Reinforcing effects are projected to paleocortical areas which by feedback, may further inhibit the inhibitors of the interstitial neurone. Consumatory behaviour reflex is then released. Anticipation of a reward associated with a certain pattern of behaviour motivates the animal to perform and thus to achieve full reward (pleasure). This initial anticipation may be of the nature of partial activity of the reward areas due to lack of satiety, either due to internal homeostatic imbalance or, in more complex animals such as man, due to perceptual imbalances. The control or feedback loops exist to suppress spontaneous reinforcement and prevent behavioural runaway which would destroy the animal: this control is vital for efficient thinking, feeling, behaving. The ventromedial nucleus is an important link in this circuit. Electrical stimulation in this nucleus —

(i) simulates satiety and diminishes eating,

(ii) sometimes produces aversive and escape behaviour,

(iii) inhibits self-stimulation in the reward areas.

On the other hand, lesions of this nucleus -

(i) removes satiety control and produces hyperphagic behaviour,

(ii) produces an "emotionally labile" animal which will self-stimulate,

(iii) produces an irritable animal.

Stimulation of tegmental areas of the avoidance system inhibits the reward areas, thus serving to redirect the activities of the animal away from painful consequences. This is further under the control of the paleocortex: paleocortical entorrhinal stimulation reduces avoidance effects of tegmental stimulation and produces moderate reinforcement as would a reward-stimulus from the environment. The amygdalae form part of this circuit serving to regulate hypothalamic thresholds. Stimulation or irritation of this nucleus bilaterally leads to hyperinquisitiveness, loss of habituation and increased consummatory drives. The septal area, also part of the feedback loop, is known to damp hypothalamic activity, suppress inappropriate behaviour, and therefore, to increase the ability of animals to discriminate. In man, other areas such as temporal cortex and frontal cortex have taken over much of the feedback control of the hypothalamic substrates.

Mood mechanisms are, therefore, complex in their control and integration; but simple in that the primary centres are located in the hypothalamus. The result of the integrating process is a homeostatic physiologic state. The normal variation in a person's mood represents temporary imbalances in this circuitry. Large variations produce far more than changes in the predominant emotional tone experienced by that person, evidence showing that all aspects of feeling, thinking, learning, remembering, behaving may be altered. A model paraphrasing these conclusions is presented by Rosenzweig.

"The theory holds that normal mental activity is dependent upon the appropriate interaction of two fundamental functional systems. The more primitive affect system, functionally reminiscent of Freud's primary processes, represented anatomically in the rhinencephalon and the subcortical structures with which it connects, and the abstract system.

The affect system receives messages related to ongoing bodily processes. A homeostatic shift will cause the affect system to scan the perceptual stream for those cues which have come to be associated with return of equilibrium. The sig-

nificance of the percept is thus determined by the relationship of its presentation to the internal state, i.e. potential gratification or potential danger. The conscious counterpart of this relationship is the affect. While the affect system reserves the vital functions of establishing the relevance of experience, it is limited to qualitative discriminations and is autistic in orientation. The abstract system is related to the neocortex and is roughly analagous to Freud's secondary processes. It is this system which receives and correlates stimuli from various sense modalities and by means of such consensual validation of experience, serves to test reality. By elaborating and abstracting perceptual data, it functions normally to define experience sharply, and makes available to the affect system a broader base for decision-making (the grey tones of affect), and enables the organism to meet the demands made upon it in the environment."

"Ordinarily, the sensory input, after initial correlation and elaboration by the abstract system, is communicated to the affect system, which seeks to establish the relevance thereof for current and prior needs and for ongoing processes. This information is then fed back to the abstract system where it serves as the basis for further elaboration. The back and forth communication between the two systems permits a sequence of meaningful associations which is at once reality oriented and useful in problem solving."[6]

ROLE OF DREAMING IN MAINTENANCE OF MOOD HOMEOSTASIS

One of the features often noted in disturbed and psychotic patients is a disturbance of sleep, either too much or too little. Until recently, we equated sleep with a state of peaceful insensibility accompanied by an E.E.G. like that seen in the different stages of anaesthetic depression. The cause of this normal diurnal variation in consciousness has remained a mystery. In 1953, it was discovered that four or five periods during an average night's sleep were associated with rapid conjugate eye movements, an E.E.G. typical of arousal, alterations in respiratory rate, blood pressure, and movements such as smiling, retching, grimacing, twitching and crying out during sleep. Subjects awakened during these periods almost always reported that they had been dreaming. Surprisingly, it was more difficult to arouse the dreamer when he was dreaming than when he was in a state of deepest sleep, and so, it was called the paradoxical phase of sleep, or the "D-state". In man, the percentage of sleeping time spent in the D-state decreases from 50 - 80% in premature infants to 13 - 18% in the elderly.[7]

Other features of this state have been reported.[8] It has been found in all mammals but not in reptiles or lower forms. It is characterized by physiologic arousal during which time the animal is protected by complete cessation of muscle tone or neuromuscular suppression. A nucleus in the central pons serves as the triggering centre both for the arousal and for the neuromuscular suppression. Some feel the posterior hypothalmus is equally a trigger.

Four types of evidence suggests that D-periods are biologically essential for maintenance of normal homeostatic balance within the nervous system and within the circuits already described as regulating mood or affective balance.[9,10] Firstly, human subjects deprived of up to 90% of their D-time become irritable, increase their appetite, develop low level anxiety and experience difficulty concentrating. Personality changes have been noted with schizoid traits, paranoid flights of speech, flighty irrelevant giggling being observed. To some colours looked more intense. Secondly, cats were similarly deprived. Some cats became ravenous, restlessly searching for food. Exploratory behaviour increased. Impaired ability to learn discrimination and strong impulsivity became noticeable. In general, these animals were

hyperexcitable and showed an increase in drive oriented behaviour. Thirdly, after deprivation of D-time ceases, the animal or man makes up lost D-time by spending a greater percentage of his time dreaming. In his dreams, he is even more active, twitching, increasing rapid eye movement, and may even show myoclonic seizures. It appears that explosive pressure had built up, the safety valve opening just in time. Finally, Jouvet has selectively destroyed the pontine nucleus responsible for D-states in cats. These animals continued to have ordinary-looking waking states and sleep states, but no longer had D-periods. These animals lived only a few months during which time they manifested a condition characterized by increasing tachycardia, agitation and episodes during which the cats looked about curiously, wiped non-existent objects with their paws and appeared to the observers to be probably hallucinating. The cats finally died in an apparently toxic state after a period of coma.

It has been suggested that a metabolite derived from gamma-aminobutyric acid is the biochemical trigger of D-states. Analogues of this substance have produced D-states when injected into the pontine nucleus responsible for D-periods. This substance is an unproven inhibitory central nervous system neuromodulator. Assuming that the activities of our pacemakers or interstitial neurones are controlled solely by inhibition, and assuming that the inhibitory substance is gamma-aminobutyric acid, a hypothesis may be stated. Constant inhibition leads to gradual loss of sensitivity to the inhibitory effects of this substance: therefore, it accumulates in increasing amount. When a threshold is reached, the animal becomes tired and goes to sleep (probably by suppression of the posterior hypothalamus). D-periods then begin: in the D-state, the production of the neuromodulator temporarily ceases; a discharge of the reward centres occurs; the concentration of the neuromodulator falls and sensitivity to it returns to normal. During the whole process, the animal is protected by inhibition of motor neurones. Thus, the defences of the mind are re-constructed.

CLINICAL APPLICATIONS

This model may be applied in two areas: the first is in an explanation of the physiology underlying certain of the functional psychoses; the second is in the explanation of drug actions.

It is not a great leap to suppose that loss of control over the reward centres or their breakaway would lead to at least five possible consequences:

1. A disorder of affect characterized by apathy or lack of emotional response, inappropriate or delayed emotional or affectve changes dues to very slow and blunted response of the "pleasure" centres, volitional disturbances, "self-stimulatory excitement" due to spontaneous uncontrolled discharge, and loss of emotional contact and response to the outer world. The patient may look depressed and preoccupied, but the painful affect of depression is re-replaced by the wistfulness of no affect at all.
2. A disorder of thinking primarily related to the association characterized by loss of precision, failure to discriminate, inability to solve a problem logically or to recognize when a solution had been correctly reached (due to failure of reward for problem solving to appear), gross loss of insight, poor judgment, loss of sense of what is real and spontaneously appearing delusional material due to spontaneous reward of a casual idea. Hallucinations might arise from the uncontrollable directives of the abnormal mood state given to the perceptual apparatus for elaboration. No other disturbance in consciousness is expected, for none have been observed in self-stimulating animals.

3. An increase in the time spent in the D-state prior to a psychotic episode is expected, since inhibitors are being poured out in an attempt to control incipient breakaway of the reward centres during the awake hours and this pool must be dissipated by D-periods. Once release occurs, the inhibitor will no longer effectively accumulate and D-periods will return to normal, although they may not be as clear cut electrophysiologically.

4. The disorder produced responds clinically to drugs suppressing self-stimulation but not suppressing the response to avoidance stimuli once the latter stimuli are given. These drugs are the phenothiazines: a series of dose response curves was determined in self-stimulating animals and the order of effectiveness in inhibiting self-stimulation corresponded to their clinical efficacy in treating schizophrenic psychosis.

5. Mood elevators of iminodibenzyl type apparently sensitize the reward centres to the effects of catecholamines. This would be expected to facilitate the breakaway in cases where discharge of the reward centre rather than spontaneously decreased electrical activity was occurring.

When the cues of reason are thus disturbed, the diagnosis of schizophrenia may be made. More properly, this should be called schizophrenic syndrome for there may be a variety of causes underlying this disorder of physiology. It is therefore likely that the schizophrenic syndrome is a primary disorder of mood.

CONCLUSION

The preliminary development of a model of mental functioning based on current neurophysiological data has been presented. The usefulness of this model has been determined by its ability to explain the pathophysiology of the schizophrenic syndrome. Due to limitations of space and of good research information, other psychiatric disorders such as the manic depressive group of psychosis (including depressions) and the toxic psychoses, will not be discussed here.

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OVERVIEW OF THE DEPRESSIONS

By CORRINE DEVLIN '67

It has been stated by many authorities that quantitatively, the depressive disorders are among the most common aberrations from health with which the family doctor must deal. Further, their qualitative nature is often such that their recognition and management may never come to consultative and/or hospital treatment. These two considerations, then, are offered by way of justification of this monograph in which an attempt will be made to present, in a succinct though admittedly oversimplified way, an overview of the depressions.

DEFINITION

The term "depression" has a myriad of connotations; to some it means sadness, to others dejection, fatigue, or just a "let-down feeling". For the physician it should imply dejection of mood, and "psychiatrically . . . depression . . . is a morbid sadness, dejection, or melancholy; to be differentiated from grief, which is realistic, and proportionate to what has been lost". A depression may be a symptom of any psychiatric disorder, or may constitute its principal manifestation. Neurotic depressions are differentiated from psychiatric depressions in that they do not involve loss of capacity for reality testing.[1] That any depression, regardless of cause, might become psychotic in intensity is a matter for personal consideration and open debate.

CLASSIFICATION

In order to place the depressions in proper perspective with regard to the spectrums of psychiatric disorders, the following classification is offered.[2]

I PSYCHOSES

A. organic

(1) acute brain syndromes (deleriums)

(2) chronic brain syndromes (dementias)

B. functional

(1) schizophrenic syndromes

(2) affective "Mood" disorders -

(a) manic-depressive psychoses

(b) involutional depressions

(c) reactive depressions

II PSYCHONEUROSES

A. anxiety states; including phobias

B. hysterias; dissociative reactions and conversion hysterias

C. obsessive-compulsive disorders; obsessions and compulsions

D. psychoneurotic depressions.

III MENTAL DEFICIENCIES

A. by intelligence level

(a) mild 70-85 I.Q. level

(b) moderate 50-69 I.Q. level

(c) severe - 50 I.Q. level

B. by clinical syndrome; Mongoloid, P.K.U., etc.

IV PERSONALITY DISORDERS

A. personality pattern disorders "psychotic-like personality"

(a) inadequate personality

(b) schizoid personality

(c) cyclothymic personality

(d) paranoid personality

B. personality trait disorders
"neurotic-like personality"
(a) emotionally unstable personality
(b) passive-aggressive personality
(c) compulsive personality

C. Sociopathic personality disturbance
(a) antisocial personality
(b) disocial personality
(c) sexual deviant personality
(d) addict - alcohol or drugs

V PSYCHOSOMATIC DISORDERS

A. functional clinical syndromes; headaches, mucous colitis, etc.

B. psycho-dependent organic disorders; duodenal ulcer, ulcerative colitis, etc.

NOTE: In Psychiatry, as in Physical Medicine, the possibility that two or more disorders may coexist is a real consideration, and neither are mental and physical illnesses mutually exclusive.

ETIOLOGY

The presence of fluctuation of mood can scarcely be heralded as having diagnostic significance. Depression is really a syndrome with numerous component symptoms. It is possible, however, to introduce some order by expressing the symptomatology in terms of a formula made up of the classical triad of physical, emotional, and psychic symptoms. "Emotional" in this context is to mean having to do with feeling in affect, while "psychic" will refer to aspects of cortical synthesis.

While it is unsound logically to predicate complexity of cause to explain the diffuse symptomatology of depression, it would appear that this may indeed be the case; at the present time it is not possible to state THE cause of depression, but only

(1) to consider the genesis of depression within the framework of the soil-and-seed theory, and

(2) to discuss those variables which may operate to effect the development and course of depression disturbances.

Frank J. Ayd in his book "Recognizing the Depressed Patient"[3] makes several useful observations in this regard, the essential of which are summarized below.

(1) **Role of Heredity:** There is an increased incidence of emotional and mental disorders, and in particular depression disorders, in the families of patients with psychotic depressive reactions.

(2) **Role of Physique:** Kretschmer and Sheldon have both described a "type" of body build which is seen more often among manic-depressives than in the general population. Thus the pyknic (Kretchmer) or endomorphic (Sheldon) body habitus - average height, short bull neck, round face, thick chest, protuberant abdomen and slender extremities - has been associated with this illness.

(3) **Basic Personality:** According to studies done before the onset of, and between exacerbations and remissions of, affective disturbance, it would appear that the cycloid temperament, is related to this illness. This temperament may be manifest in the depressive, hypomanic, or cyclothymic personality type.

(4) **Physiologic Factors:** Kraines[4] states that physiologic changes occurring in depressions are linked to altered functioning of the hypothalamus, rhinencephalon, and reticular systems. The reasoning behind the postulate is this; at the clinical level it has been observed that depressed patients exhibit altered appetites, weight, gastrointestinal mobility, menstrual, sexual and sleep patterns - neurophysiologic studies have demonstrated hypothalamic centres for central regulation of appetitie, weight, gastrointestinal mobility, menstruation, sex and sleep. Symptoms related to these centres are referred to as "diencephalic signs".

(5) **Psychologic Factors:** In this context, Ayd only cautions against immediate acceptance of the patient's (or his family's) explanation for the development of the illness. He states "Whenever a person selects a symptom as the etiologic agent, it can be assumed he is quite ill, and more reliance should be placed on this facet of the clinical picture in estimating the degree of depression than almost any other symptom".[5]

(6) **Endocrine Factors:** No definition cause-effect relationship has been demonstrated with regard to endocrine dysfunction in affective disorders. That one is suspect springs from four empirical observations

(a) Sex incidence: women are effected three times more commonly than men.

(b) the appearance of depressive state post partum and during female climacterium.

(c) alteration of menses during the course of depression.

(d) alteration of the course of depression disorders by menses.

(7) **Seasonal Factors:** Patients suffering from manic-depressive disorders tend in general to become ill in the spring or fall, and on an individual basis, they seem to have, a particular seasonal "time" for the recurrence of their illness.

(8) **Somatic Factors:** Concomitant diencephalic disease, cerebral infections, and systemic disease tend to precipitate affective attacks.

(9) **Age of Onset:** In the manic-depressive group, women at first presentation are usually between the ages of thirty and forty; for the men, the common age group lies between forty and fifty. As age increases, depressive attacks occur more frequently than manic attacks: i.e. involutional melancholia.

(10) **Time of Onset:** Owing to the insidious nature of depressions, an astute history frequently reveals that the actual onset of the depression (called the "starting point") predates the clinical presentation.

(11) **Fluctuations:** Depressive states may be self-limiting: Ayd states that a depression attack has a duration range of 6 - 18 months. If the attack be depicted graphically, one would see a symmetrical depression and elevation of mood below a baseline as the major mood fluctuation, and superimposed upon this, the minor fluctuation of mood which occurs from day to day and within a twenty-four hour period. The decline to the depth, and "reentry" to the baseline are in essence mirror images.

(12) **Duration of Attack:** Age of onset has prognostic importance and it is generally agreed that depressions, at first presentation and untreated, tend towards the following duration patterns:

Age Group	Duration
Childhood and adolescence	3 months
Adolescence to age 30 yrs.	6 - 12 months
30 - 50 yrs.	9 - 18 months
- 50	protracted periods; perhaps 3 to 5 years

DIAGNOSIS

The examination of the patient whom the physician suspects has a depressive disturbance proceeds along the same line as those involved in establishing the diagnosis of any disease. There is, however, a shift in emphasis to the history of the illness and this, together with "inspection" becomes of paramount importance.

At was mentioned earlier, the characteristic picture of depression may be described under three headings, these being physical, emotional and psychic symptoms; a short account of each will be given in turn.

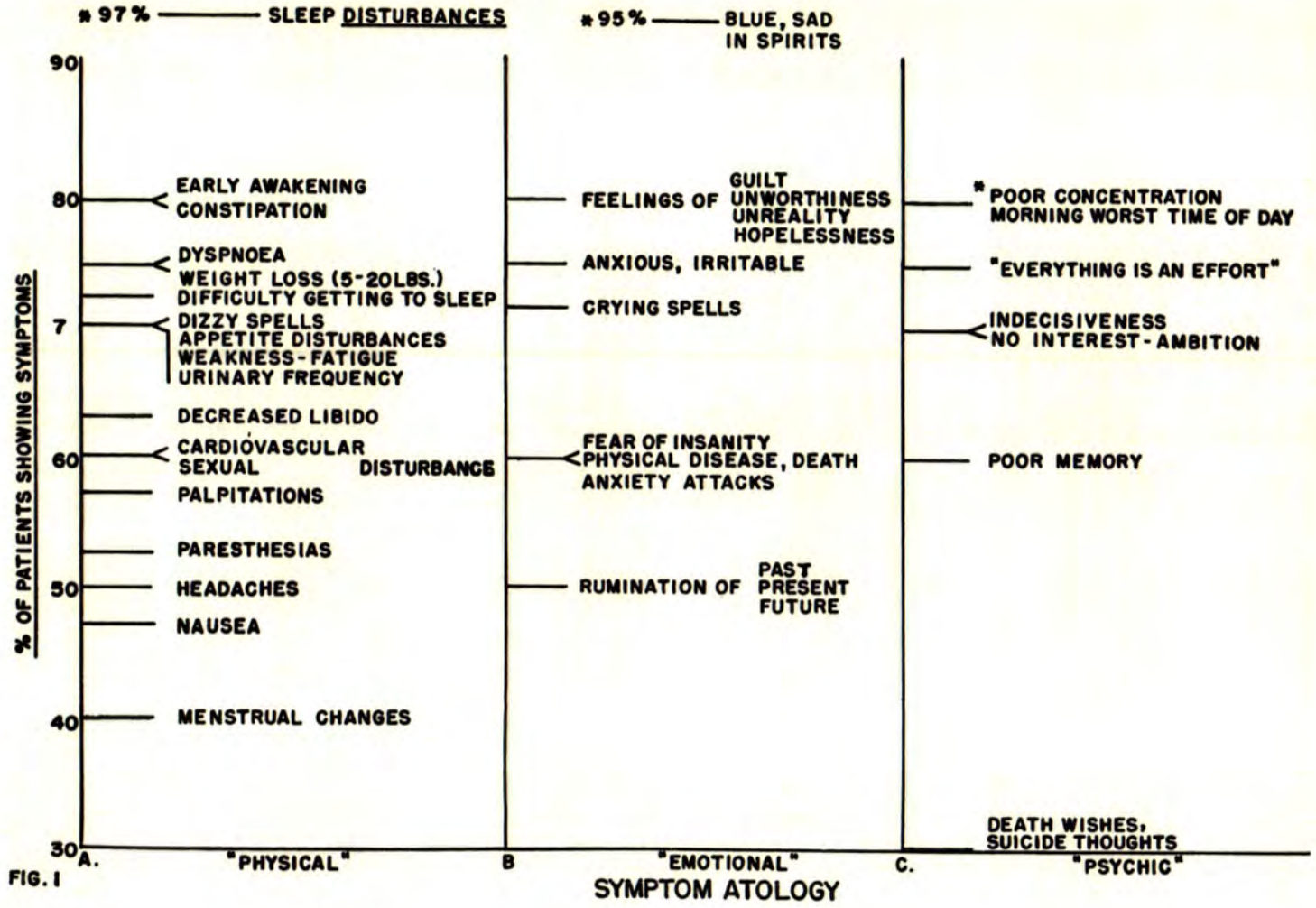
* 97 % SLEEP DISTURBANCES
* 95 % BLUE, SAD IN SPIRITS
% OF PATIENTS SHOWING SYMPTOMS
90
80
7
60
50
40
30
EARLY AWAKENING
CONSTIPATION
DYSPNOEA
WEIGHT LOSS (5-20LBS.)
DIFFICULTY GETTING TO SLEEP
DIZZY SPELLS
APPETITE DISTURBANCES
WEAKNESS-FATIGUE
URINARY FREQUENCY
DECREASED LIBIDO
CARDIOVASCULAR
SEXUAL DISTURBANCE
PALPITATIONS
PARESTHESIAS
HEADACHES
NAUSEA
MENSTRUAL CHANGES
FEELINGS OF GUILT UNWORTHINESS UNREALITY HOPELESSNESS
ANXIOUS, IRRITABLE
CRYING SPELLS
FEAR OF INSANITY
PHYSICAL DISEASE, DEATH
ANXIETY ATTACKS
RUMINATION OF PAST PRESENT FUTURE
* POOR CONCENTRATION
MORNING WORST TIME OF DAY
"EVERYTHING IS AN EFFORT"
INDECISIVENESS
NO INTEREST-AMBITION
POOR MEMORY
DEATH WISHES,
SUICIDE THOUGHTS
A. "PHYSICAL"
B "EMOTIONAL"
C. "PSYCHIC"
SYMPTOM ATOLOGY

FIG. I

PHYSICAL SYMPTOMS

"Nothing in the human organism remains the same from the inception to the termination of the illness".[6]

As might be expected, the physical symptomatology stems from altered vegetative functions, and is elicited in the history under astute questioning. The more common diencephalic - determined physical complaints are shown in Figure 1 (a), as is the relative frequency with which they occur in depressed patients. When these features are elicited (a) from a patient who appears preoccupied about a concert of pansystemic complaints and (b) by a physician with a high index of awareness of the high incidence of affective disorders, the diagnosis of depression is at hand.

EMOTIONAL SYMPTOMS

Again, the cardinal symptom of depression is dejection of mood and from figure 1 (b) it is seen that 95% of patients experience this symptom. It is important to realize that the patient may not flatly state "I am depressed and feel dejection of mood", and so the physician may have to rely on other, more subtle, clues. It is left to the power of observation of the clinician to be impressed by such things as retardation of thought, hesitancy of speech, slow stilted movements, drooped shoulders, apathetic handshake, sallow complexion, and lacklustre eyes, in his assessment. It is also left to the clinician to realize that the "classical picture" of depressions seldom presents itself - that such features as anxiety may be operating within the depression to cause gross motor restlessness, and an agitated appearance. One or many symptoms may be absent, present but not marked in degree, or present and prominent - a patient may complain of anorexia, nausea or bulemia - it is left finally to the accumen of the clinician to assess each patient individually on the merits of the findings.

PSYCHIC SYMPTOMS

"There are two aspects of every major category of psychiatric disorders. One is the form of the illness, which is constant, and the other is the content, which is variable".[8]

The form of the psychic symptoms to watch for and evaluate include psychomotor retardation, impaired ability to concentrate and remember, indecisiveness, loss of or diminished interest, suicidal thoughts and death wishes, misinterpretations, illusions, hallucinations, delusions, changes in behaviour patterns, and strained interpersonal relationships. The content, being more variable, assumes a less important role in the evaluation of psychic symptoms. A Roman Catholic may have a religious core in his delusional system, while an atheist may express delusions which are finance-oriented; what matters is that the physician recognize the existance of delusional material.

GUIDELINE

Depression *per se* is simply a finding - a genetic blob; in the "sorting out" the differential diagnoses, the following simple schemata may prove useful.

Think in Terms of Degree

. . . and decide where, on the locus between health and illness, the presenting depression is located. It is probably worth mentioning that depressions are thought of by some as being a continuum, while others view them as being distinct entities wherein variables such as heredity, family background, etc. operate to determine what "kind" of depression the patient will develop.

Consider the Mode of Presentation

This may affect the treatment of the disorder.

(1) Straight depression: showing the triad of depression symptomatology.

(2) Masked or smiling depression: easily missed - patient may go on to suicide which is the most serious complication of depressions.

(3) Depression defending Schizophrenia or almost any psychotic disorder, or secondary to other disease which may be functional, psychosomatic, organic, or mental.

Locate The Patient Within The Classification of Depressions

This is usually easily done if one considers the Depression disorders as falling into two groups.

(1) Exogenous or Reactive = secondary to an event occurring external to the patient,

(2) Endogenous = secondary to some event within the patient, currently thought of as dysfunction in the diencephalic - rhinencephalic-reticular formation axis.

This is further subdivided into neurotic and psychotic depressions, and as a general rule these two are differentiated on the basis of reality testing.

TREATMENT

Having decided upon the diagnosis, it remains now for the physician to design a regimen which will best attain the three basic goals of any therapeutic programme.

These are

(1) to shorten the course of the illness,
(2) to minimize suffering,
(3) to prevent complications - in this case - suicide.

In the achievement of these goals, both general and specific measure are employed - the setting for treatment will vary with the individual case; hospital treatment will be indicated for some, while others will be treated as an outpatient basis.

General Treatment

(1) nutrition - adequate protein, fat, carbohydrate, vitamins,
- adequate hydration.

(2) elimination - adequate renal and gastrointestinal function
- proper ventilation

(3) hygiene - general; skin, hair, nails
- oral care

(4) equilibrium -

social
↑
mental ← activity ⟷ rest →← sleep
↓
physical

Specific Treatment

The following therapeutic measures are available. No mention will be made of particular regimens which are tailored for specific depressions. Suffice to say that treatment may range from therapeutic nihilism to lobotomy depending on the severity of the illness and the stage of the depression.

(1) Simple supportive therapy including explanation and reassurance.

(2) Psychotherapy: usually supportive, though occasionally analytic is indicated.

(3) Chemical therapy:

(a) hypnotics: Long acting barbiturates for early wakers; short acting barbiturates for those having difficulty getting to sleep.

(b) stimulants: Amphetamine group for psychomotor retardation. Amphetamines are of little use when there is a depression of any severity.

(c) psychic energizers - iminodibenzyl derivatives - Tofranil, Elavil
Monoamine oxidaze inhibitors - Parnate, Marplan, Nardil

(d) tranquilizers - of special use in agitated depression
- Chlorpromazine.

(e) hormones - useful in agitated depressions secondary to menopausal syndrone, or birth control pills
- Estrogen, Progesterone, Testosterone

- ECT or electroconvulsive therapy

- definitive 'in-hospital programme.

- sub-coma insulin therapy - usually only used when the "anxiety component" of depression cannot be ameliorated by tranquilizers.

- bilateral Stellate Ganglion Blockade.

- Lobotomy.

Acknowledgement:

Appreciation is extended to E. J. L. Deinum, M.D.. F.R.C.P.(C), Chief of the Male Division, Ontario Hospital, London, for her interest and her suggestions.

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FOOTNOTES

[1] *A Psychiatric Glossary*: 2nd Ed., Wash., D.C., 1964.
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[6] *Ibid.*, 3, p. 23.
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ADOLESCENT BEHAVIORAL DISORDERS

By BRIAN GAMBLE '67

INTRODUCTION

Behavioral and mood disorders in adolescents form a group of perplexing and difficult psychiatric problems confronting the general practitioner who is often the first called upon for aid. Some of the difficulty stems from the fact that the problems cover several fields; general medicine, psychology and psychiatry. The busy general practitioner should have the ability to recognize into which specialty the presenting problem falls, and readily refer when the situation dictates. Medical education presents opportunity to deal with the major psychotic problems afflicting adolescents but it provides little insight into the purely "behavioral disorders" in adolescence which are commonly considered the domain of the psychologists. Unfortunately there are not enough psychologists to counsel for every physician who is presented with an adolescent behavioral problem or mood disorder by a distraught parent. It is hoped that this article will serve as a stimulus to further reading into these problems.

DEFINITION:

The term adolescence can roughly be called the teenage period but consensus puts it as the period beginning with pubescence and ending with the achievement of adulthood - obviously a highly individualized period, and a most critical one.

It is of the utmost importance in considering problems encountered during this period to remember that the problems encountered during earlier periods which were either not properly solved or completely repressed, become a prominent feature of the next developmental period. When dealing with problems of the behavioral nature, keep in mind the dynamic nature of human development.

SCOPE OF THE SUBJECT

Abnormal behavior begs the question of normal behavior, the delineation of which one would be hard pressed to set forth; rather let the discussion deal with the most common problems which cross the physician's desk and leave it to the personal judgment concerned as to whether the behavior displayed is abnormal. From the outset, he is obliged to classify the type of behavior presented in order to arrive at his diagnosis - a careful history and a complete physical examination should rule out organic causes and place the problem in the functional category. The psychotic reactions should be considered first, and if suspected a psychiatric consultation should be sought. The physician often comes up with a group of symptoms which he knows does not constitute a psychotic reaction or perhaps not even a neurotic reaction rather some sort of social maladjustment yet inability in handling these symptoms tempts the physician to dismiss the problem with the platitudinous reassurance that no pathology exists and the child will grow out of this phase. A discussion of these symptoms and the several theories of their origin may be of some assistance in dealing with them when they occur in practice.

Behavior in the adolescent essentially is the manner in which he responds to the

environmental stimulation after it has been assimilated and coloured by emotion - in attempting to understand behavior one must consider the many facets that go into the response such as perceptual input including sensory input and interpretation; emotional response to stimulus and modes of response to stimulation.

Three of the more common symptoms of "behavior problems" are discussed below as an example of approach.

1. **Aggressive Behavior.**

Various psychiatrists observing children have noted diametrically opposed responses directed at the same person such as the mother; Bleuler in 1924 applied the name 'Ambivalence'. Some other researchers consider these emotional swings do not represent a form of emotional conflict grounded in uncertainty, not a vascillation between feelings of love and hate, but rather the ability to respond in either direction as the occasion or provocation arises. This ambivalence in feeling although common and considered benign can on occasion form the basis for a neurosis in adolescent periods when one is unable to reconcile these contradictory emotions directed especially toward parents. In some cases, ambivalence has a flat quality (without emotion) or inappropriate (strange emotion): this vagueness - a failure of decision-making may be regarded as potentially schizophrenic until proven otherwise; coexistent depression or dysphoria does not exclude it.

2. **Emotional Insecurity.**

The adolescent suffering with emotional insecurity usually assumes the aloof attitude of the schizoid or he shows evidence of excessive bidding for the attention of adults as a result of his being acutely aware of his non-acceptance. He may drive maniacally to achieve this acceptance or he may withdraw from those he needs. Adolescents displaying these tendencies disturb their peers extremely and they often confuse the physician because he considers them schizoid. The physician does well to recall that this condition commonly develops far back in childhood - the child who is rejected by his parents; the child who is coping poorly with sibling jealousy. The fear, aggression and insecurity was manifested early but then the parents considered it was "normal" but as the child continued into adolescence his insecurity followed him and the behavior associated with the insecurity assumes a grander scale which is commensurate with the adolescent's age and abilities. The parent now approaches the physician but the proper treatment - prevention - is not possible.

3. **Sex Behavior.**

Many consider the adolescent period, which begins with the pubarche, as the activator of the sexual urge. Again the development of emotional behavior begins at a much younger age. Tumescence and detumescence are frequently observed in infants and, although there is no estimate in the normal population, many preadolescent children engage in exploratory manipulation and sex play. By adulthood some 90% of males and 50% of females have engaged in masturbation at one time for varying duration. Such endeavours are accompanied by varying degrees of placidity and tension release but, expectedly, by reason of cultural counter-pressures, these pursuits are also accompanied by varying degrees of fear and guilt.

Studies have consistently pointed out that children and adolescents are intensely curious and perplexed about sex and that this curiosity is far more prevalent than opportunities for frank discussion with adults. I need hardly point out that, if the parents will not or cannot explain sex problems, then the family physician is the next best choice. It appears desirable from the point of mental health, that pleasure incident to activities involving sex happen to be socially acceptable to his elders; at the moment dating, mixed parties, dances,

etc. seem to be in order and the exhilaration he may feel when he engages in petting and other forms of play will probably be congenial to his conscience.

TREATMENT

The treatment of serious behavior problems is usually the perogative of the child psychiatrists but there are many areas the general physician can participate in, mainly in the matter of warding off serious problems before they develop.

DEVELOPING EMOTIONAL CONTROL IN CHILDREN

1. Good General Health.

The fretful fearful adolescent or the one easily pushed into a rage is often merely the one who is under-nourished, tired, or ill.

2. Wholesome Parental Attitudes.

The parents who muster as much calmness and poise as the exigencies of life and the limits of the parents' own emotional constitution permit are very significant for the child's and adolescent's emotional development.

3. Inhibition of Expression.

The adolescent from infancy is trained by society to express his emotions only in socially approved ways. The problem arises in the field of sexual behavior; here society brings some of its most forceful restrictions on the developing adolescent. In the majority of cases, however, the development of control of the outward expression occurs at the same time that experience or training is building up suitable modes of emotional reaction. On the whole, forcing a child to inhibit the expression of the strong emotional responses of fear, anger, sexual urgings seems far less useful than either avoidance of highly exciting events or placing emphasis upon substitute activities of positive value.

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THE PLASTIC PERSONALITY

— A FREUDIAN APPROACH TO PEDIATRIC EMOTIONAL DISORDERS —

By RON CASSLEMAN '67

The medical practitioner occupies a strategic position in the treatment of emotional illness, for it is he who sees the patient first. His patients come with confidence for the alleviation of the suffering of minor emotional disturbances causing anxiety. Frequently they are anxious to talk about their problems even though they are fearful of taking this initiative. On dealing with emotional disturbances of childhood, many problems arise for the child is in no position to intelligently discuss his or her anxieties with the physician who may be left with the feeling of inadequacy. What the practitioner does when confronted by such a problem may well determine the tragic outcome of personality maldevelopment in that child, a more or less permanent disability, for "all children do not grow out of their problems, their maladjustment and misbehaviour".

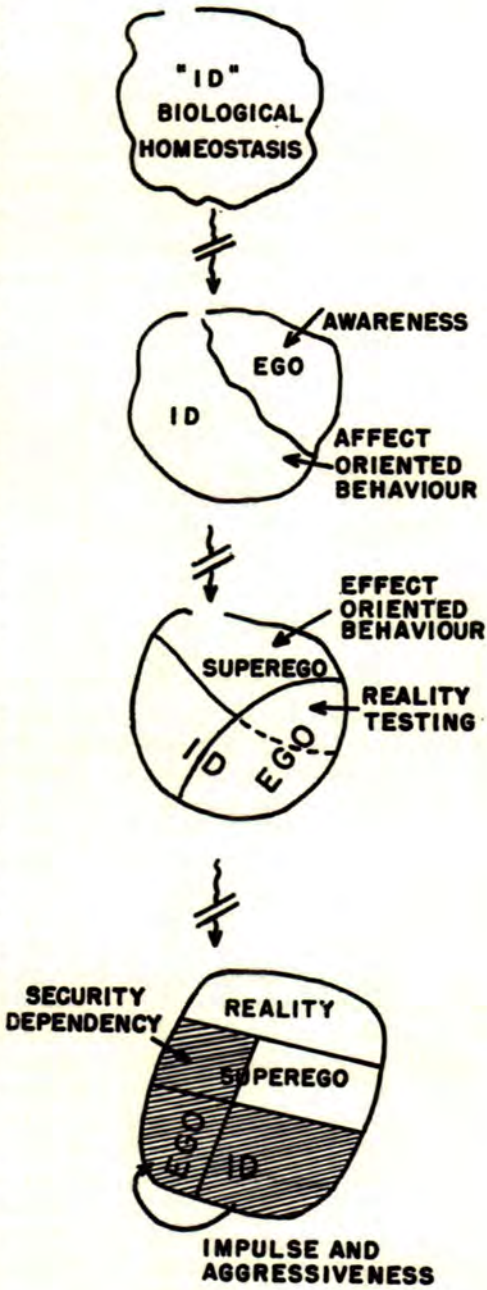
DEVELOPMENT OF IDEAS

In assessing a child who "isn't quite right", it is helpful to have in mind the following model. Some characterize the child's mind by the term **lability.** A more useful epithet is **plasticity** by which is meant the capacity of the developing mind to be moulded during its development and maturation. There are 3 psychological categories entering into the final product: these are the Freudian triad of id, ego, superego, much quoted etiological agents or causative factors in personality maldevelopment. Most forget that originally they were seen as three different predominant categories of the mind both in adult or adolescent patients suffering from mental illness and in Freud himself. The classical "oedipus complex" is also a pathological entity which only becomes manifest in abnormal development. Therefore, such terms represent observations and conclusions by the examiner, of the ongoing dynamics in the regulation of the patient's thinking, feeling, and behaving rather than causative agents in themselves for which they have been misinterpreted. The sequence of development may be represented as follows.

Plastic blob presents at birth. It is composed of "ID" material, also known as the "instinctual drives" within the individual which strive for expression and satisfaction. This is also regarded as a quantity of psychic stimulation or activation, referred to by some as **reactivity** or **mental energy.** Infants differ in their reactivity, i.e. the placid infant vs the hypertonic bilious infant. Organic sources of discomfort, i.e. infections, food allergies make the infant more irritable or reactive.

As the brain matures, it becomes capable of perception. Ego represents the earliest awareness of the self, or self perception: this is composed of the awareness of the id within, the drives and tensions it sets up, and of the immediate environment providing satisfaction or not for these drives. Later, awareness of the potential of the environment to refuse satisfaction occurs, i.e. frustration. The final stage in ego evolution is the awareness of one's separateness from the outer world: this provides the impetus for goal directed activity, for now the mind sees that the environment is not merely an extension of itself but a place from which one can actively obtain what one "needs".

The superego is a quality developing out of the ego. The ego represents awareness of "self" and the superego represents the sum total of the pleasurable, painful, tensing and relaxing experiences of the ego in its active "getting-on" in the outer world: it is "effect" oriented. These "results" of behavior are internalized as memories and serve as an error control mechanism helping the ego to restrict behaviour to the most productive in terms of emotional needs, i.e. security and satisfaction. With advancing development, the blunt desires for satisfaction and security become refined so as to be represented by subtleties of emotional tone. Similarly the superego becomes more refined, developing into a "conscience". Thus conscience has a strong semblance to the values of the parental and other authority figures in the surrounding milieu. Strangely, through its original association with powerful affective responses, it becomes capable of menacing or flattering the "self" in an attempt to direct the ego towards "acceptable behaviour". In the adult this is almost entirely confined to the abstract or intellectual level of functioning, and the biological, emotional substrate becomes obscured by the needs of the ego for satisfaction and security as for value conflicts resulting in anxiety. By our original model we see that it is always the ego which suffers the painful or pleasurable experiences of the mind and so it is the ego that complains to the doctor and it is the ego that comes to be treated for its emotional illness. For the doctor to effectively treat his patient (ego), he must recognize the delicate balance between the 3 qualities of mind, id, ego, superego, and his opportunity to deal with each separately.



This psychic system becomes directly involved with behaviour through the ego, as it alone is the executor of decision. The ego has means and methods of discharging tension that will simultaneously satisfy the superego and the id. By observing the behaviour of the individual child, one may

demonstrate exactly how the ego responds to its drives. The means and methods employed by the ego are known as "mental mechanisms". When they fail to maintain homeostasis, symptoms develop or defence mechanisms are introduced into the fray. For example, "reaction formation" represents a means of restoring order when that order is threatened by a conflict between conscience and a powerful impulse of opposite intent to what conscience directs: hence the ego may direct behaviour that leads in the direction opposite to the impulse. The list of such defences is lengthy and includes sublimation, projection, identification, introjection, regression, denial, isolation, rationalization.

In general, the field of child psychiatry suffers even more than that of adult psychiatry from still undefined diagnostic criteria and a vagueness which pervades attempts to classify and analyse emotional disturbances. The developing mental apparatus of the child tends to meet the stresses of living in a labile and variegated manner, making attempts to classify mental state and capabilities difficult. The child's "neurosis" is rarely confined to a reaction that can be accurately assigned to one of the described types of adult neuroses, but is something more complex and multiform. The child's growing ego shows both fragility and resilience in response to the moulding effect of experience. Relatively minor trauma by adult standards may cause a breakdown of ego-functioning while even severe regressive breakdowns may pass over quickly due to the great recuperative potential of the developing ego, i.e. schizophrenic children (autistic) if treated early may be restored to more or less complete integrity depending on the recuperative potential of that mind. It is most difficult to forecast the prognosis in an individual case showing outstanding symptoms of disordered functioning.

CLINICAL APPROACH

What is one to do when presented with a disturbed child?

Firstly one must let the parents give a history supplemented by a detailed inquiry as to the symptoms shown. Secondly, one must do a "mental physical". In this regard, traditional 'ABC' analysis by appearance, behaviour, conversation go by the board. Conversation as used to determine the state of the sensorium, memory, affect, thinking, intelligence, is less applicable to the child. The important determinant in the child is the **level of ego function** as a clue to the nature of the disorder in the delicate homeostasis of the developing mind. This concept is extremely significant since it permits an estimation of the ability of the child to function in his life situation and thereby indicates the degree of pathology. Observation is important and may be the preferred approach in the use of such tests as the draw a man test, telling stories about pictures, and in the use of play activity to evaluate object-person relationships. Recently, some authors have postulated an addition to the usual diagnostic categories of adult psychiatry, namely "the borderline states of childhood". These states show how disturbances in self-perception and organization may be involved in producing a disturbance of both emotional and behavioural integration. This syndrome is seen with some frequency in residential treatment centres. It seems to represent a framework from which diagnostic appraisal of the difficult case may be built. This concept of borderline states may also aid in the formulation of other diagnoses at variants along a continuum of increasing ego malfunction. The entire syndrome differs from psychotic states with their even more extensive ego dysfunctioning and from character disorders in which disturbances are concentrated in the areas of social and interpersonal relations without accompanying ego disintegration.

Two types of children are described:

(1) In this group, behaviour was characterized by lack of frustration tolerance, emotional immaturity, uncontrollable id impulses, lack of social

adaptation, and a multitude of neurotic symptoms. Although these children were not delusional and were in contact with reality, when alone, they withdrew into fantasy. They also had temper tantrums taking them temporarily out of contact with the adults about them. When rebuffed in a relationship, they would show withdrawal, hostility, or aggressiveness. They could not function in a group.

(2) In this group, children were similar to the above and showed lack of emotional differential from the mother (symbiotic psychoses). These were designated as benign psychoses. Generally, the poor patterning in their development and makeup is more fluctuating and widespread than seen in neurotic disturbances.

The clinical picture may be itemized in 3 categories.

(1) **Social Adaptation:**
- excessively antagonizing
- withdrawn and aloof
- need to be omnipotent
- paranoid tendencies
- great emotional ambivalence (love and hate)
- gains control by negativism and tantrums
- tenuous relations even with parents

(2) **Manageability:**
- generally overimpulsive
- normal negativism of the growing child excessive and unresolved
- not amenable to the usual persuasive and diversionary methods used to control and socialize other children

(3) **"Neurotic-like symptoms"**
- great multiplicity, interchangeability, frequent sudden appearance and disappearance
- overload of anxiety and tension with driven restlessness, insomnia, lack of concentration
- phobias, fears, compulsive rituals, habits and mannerisms, tics, obsessional preoccupations, autoerotic habits of fetishism, hysterical and hypochondriacal traits

Abnormal ego functioning may be evaluated under six headings.

(1) **Relation to reality:**
- **normal:** attention, preception, concept organization produce good reality testing
- **borderline:** withdrawal into fantasy, poor contact or mild disorientation, poor judgment

(2) **Regulation and control internally motivated by affect:**
- **normal:** socially acceptable appropriate responses
- **borderline:** immediate gratification of internal drives; direct, erotic or aggressive expression of impulse, greediness, sloppiness

(3) **Object relations**
- **normal:** energetic investment in a loved object that is differentiated from self
- **borderline:** symbiotic object poorly differentiated from self. Tenuous, demanding and indiscriminate relationships.

(4) **Thought processes:**
- **normal:** reality oriented objectivity leads to logical thought
- **borderline:** subjective independent inner emotions life leads to magical thinking

(5) **Defence functions**
- **normal:** ego-syntonic, realistically oriented, productive activity. (self can deal with inner impulses and demands of superego)
- **borderline:** guilt, anxiety and tension, suspiciousness.

(6) **Autonomous functions**

normal: goal directed coordinated responses

borderline: awkward, excessive, non-directed, incoordinate motor discharge

(7) **Synthetic functions**

normal: sense of causality and understanding

borderline: confusion

LONG TERM RESULTS:

Some of these children develop in various directions to become overly psychotic when entering the stressful pubescent period. Most grow into immature non-neurotic personalities and may be diagnosed as adult borderline cases.

Some workers have proposed several factors in the etiology and psychopathology of these disturbances.

(1) **Congenital or constitutional factors.**

(a) Children are not necessarily born with diseased constitutions: they may just have the potential to do so if mismanaged. Recent studies suggest that certain aspects of temperament and personality are present at birth. Constitutional factors makes some more vulnerable to emotional stress and retards their ability to develop healthy egos. These hypoactive or hyperactive affective constitutions may have a profound influence on the attitudes of the parents to the child and their ability to give him the proper emotional environment for dampening his excessive responses.

(b) The second area in which heredity plays a part is in the rate of maturation of the perceptual apparatus and the ability to organize a strong self or ego. There is some probability that constitutional weakness of the ego may be important in etiology. This may have some relation to intelligence. Functional disorganization of certain areas of the intellect may result from emotional disturbances and be accompanied by variable is distractable even though he may be superior in verbal functions.

There is a parallel between growth of the intellect and growth of the ego, for, by our theory, the ego is organized by the perceptual or intellectual apparatus.

(2) **Developmental factors.**

Psychic traumata in early life are probably decisive in their influence in maldevelopment of the ego. Before consolidation of the ego is firmly established; stress had a decidedly bad influence. Chronic stress of any type, even in the mature personality may lead to secondary perceptual distortion and elaboration. For such to occur while the foundation of the building is being laid means that the whole building will suffer.

Early distresses seen in the borderline children centred about maternal inadequacy or rejection, pathology in the mother-infant relationships and pathology in the symbiotic relationship between mother and infant, parent and child. This latter pathology results in an inability to progress towards separation and individualization. The child fails to develop confidence in forthcoming gratification and remains intolerant to frustration by the mother from whom is still undifferentiated. He experiences the outer world as dangerous and clings to his omnipotence and magic as the only means of adaptation. Basically, the ego remains defenceless against anxiety and impulse, and the disciplining effect of the superego fails to develop.

Reaction to stress, in children, is anxiety. It may be unpleasant or menacing producing dysphoria (painful apprehension) and it must be dealt with since it threatens the integrity of the ego.

CONCLUSION

The application of analysis of ego structure carries over adult psychiatry. Three types of ego structure are recognized as reflecting the end product of ego development in the child.

1) ego - enhancement = ego syntonic.

This form of ego was an adaptive advantage in a competitive society and many normal well-adjusted people have this structure. The extreme is the psychopathic personality who has no internal conflicts, acts out all impulses and whims, has lost the ability to experience deep emotional responses both with respect to others and with respect to himself. He has no consistent purpose or goal in life.

2) ego-depreciation = ego alien

These are the "neurotics". The ego cannot deal with the emotional disruption and conflicts reality brings to it. Anxiety is the common denominator of disorders in this group.

3) ego-disintegration = ego isolation

The ego withers away because it loses contact with emotions giving it orientation towards reality on the outside and towards its "self reality" on the inside. Perceptual distortion or breakdown is seen and this classes it with the psychoses.

In summary, analysis of personality reveals the fundamental defect in the delicate balance from which symptoms arise. Medical management is aimed at modifying the strengths of the various components to re-establish balance, thus favouring continuing development in a more normal manner. Antianxiety drugs, tranquilizers and other drugs may be necessary to decisively arrest pathologic processes and to reestablish control. Early and careful management of the family unit is likely to be successful in producing a "normal" adult. The recuperative self-healing properties of the child's malleable mind enables such an approach to be successful.

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Alumni News

Class of 1910

Dr. William A. Burgess - Deceased.

Dr. George E. Butterwick - Deceased.

Dr. James D. Collins - Deceased.

Dr. W. E. Fraser - Deceased.

Dr. Gordon L. Jepson, 195 Dufferin Ave., London, Ontario, interned at Victoria Hospital. He did postgraduate work in London, England and served with the C.A.M.C. in both the first and second World Wars. He was awarded the Military Cross after the First World War. He is a specialist in Anaesthesia and was Head of the Department of Anaesthesia at Victoria. He is married and has one daughter who graduated from Western (Arts '38).

Dr. Percy O. King - Deceased.

Dr. William A. Marshall, 115 Walnut St., Fairbury, Illinois, U.S.A., interned at St. Joseph's Hospital, London and did postgraduate work in London, England. Since then he has been in General Practice. He has two children.

Dr. Calvin T. McCallum - Deceased.

Dr. Alex S. McCormick - Deceased.

Dr. William H. McFarlane - Deceased.

Dr. William H. McGriffin - Deceased.

Dr. Bernard R. Mooney, 1965 W. Eighth Ave., Apt. 204, Vancouver, B.C., was a radiologist in Edmonton, Alberta but is now retired.

Dr. James A. O'Brien - Deceased.

Dr. George A. Ramsay - Deceased.

Dr. Charles F. Riley - Deceased.

Dr. Hadley V. Robinson - Deceased.

Dr. Thomas Sawdon - Deceased.

Dr. Alfred E. Short, 815 Durham Ave., Calgary, Alberta, served overseas with the C.A.M.C. during the First World War. After doing postgraduate study in Eye, Ear, Nose, and Throat in New York City, he returned to Calgary where he practised his specialty. He was head of the Department of E.E.N.T. and has been very active in the establishment of eye banks.

Dr. William H. Taylor - Deceased.

Dr. Herbert W. Wall - Deceased.

Dr. J. Cameron Wilson - Deceased.

Editor's Note: The four surviving members of Meds 1910 attended the 55 year reunion at Western last fall.

Class of 1925

Dr. Douglas V. Auld - Deceased.

Dr. James D. Balfour - Deceased.

Dr. Thomas I. Barnby, 1909 Verdun Ave., Windsor, Ontario, interned at Victoria Hospital, London. He did postgraduate training at Ann Arbor, Philadelphia, Chicago, and Buffalo. He is in General Practice in Windsor at the present time. He is married to the former Effie Wilson a graduate of Victoria Hospital Nursing, and they have two children, both graduates of U.W.O.

Dr. F. R. Varcoe Bateson - Deceased.

Dr. Harry A. Cave - Deceased.

Dr. John A. Ferguson, Delaware State Hospital, New Castle, Delaware, is a Pathologist. He was associated with Harvard Medical School, Department of Pathology and has written many articles for various Medical Journals.

Dr. George G. Gibson, 1010 Center St., Pittsburgh 21, Pennsylvania, interned at

Pittsburgh Hospital and was awarded a fellowship in the American College of Physicians.

Dr. H. H. Gilbert, 918 Lavoisier, Arvida, Quebec, did postgraduate work in radiology at the University of Toronto. He then did missionary work in China and received his Diploma in Tropical Medicine and Hygiene from the London School of Tropical Medicine. He then practised for some time in India and again in China. He has also received a diploma in Surgery from the University of Edinburgh. He was Chief Medical Consultant for Arvida District Aluminum Co. of Canada.

Dr. Angus S. Graham - Deceased.

Dr. John C. Jose - Deceased.

Dr. Fred S. Kipp - Deceased.

Dr. H. E. MacMahon, 136 Harrison Ave., Boston 1, Massachusetts, interned at Montreal General Hospital and did postgraduate training in Pathology at Boston City Hospital and in Berlin. He is a Fellow of the Royal College of Physicians and is presently at Tufts Medical School in Boston. He received an Honorary D.Sc. from Western in 1948.

Dr. R. Ross MacNeil, Hollisdayburgh, Pennsylvania.

Dr. Harold S. Magee, New Jersey State Hospital, New Jersey, is a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He is superintendent at the New Jersey State Hospital.

Dr. Wilfred L. Maguire, 95 Main St., St. Catharines, Ontario.

Dr. Donald J. McIntosh, 29 Simcoe Blvd., Simcoe, Ontario, is a General Practitioner.

Dr. W. Gordon Morris - Deceased.

Dr. Arthur J. Read - Deceased.

Dr. Lloyd H. Spencer, 26039 Huntingdon Rd., Huntingdon Woods, Michigan, interned at Grace Hospital, Detroit.

Dr. George A. Wainwright - Deceased.

Dr. Reg. J. Wride, 625 Fort St., Victoria, B.C.

Class of 1930

Dr. N. A. C. Andrews, 310 East Main St.. Flushing, Michigan, interned at St. Joseph's Hospital in London, Ontario.

Dr. H. E. Appleyard, 1 Belvedere Ave., Hamilton, Ontario, did postgraduate work in Vancouver; Manchester; and London, England, and received his M.Sc. (Columbia) in Hospital Administration. In 1960 he was named a Fellow of the American College of Hospital Administration. At present, he is associated with the Ontario Hospital Services.

Dr. W. Kenneth Bice, 65½ Hincks, St. Thomas, Ontario, interned at Victoria Hospital, London. At present he is employed at the Ontario Hospital, St. Thomas.

Dr. G. Angus Black - Address not available.

Dr. R. R. Burnett of Durham, Ontario is presently retired. He interned in Pathology and Bacteriology at Western and did postgraduate work in Hamilton and New Jersey.

Dr. A. G. Calder, 321 Dufferin Ave., London, Ontario, interned at Royal Victoria Hospital, in Montreal. He is a specialist in allergy and arthritis and has a private practice in London.

Dr. H. B. Grace, 493 Adelaide St., London, Ontario, is a Paediatrician in private practice in London, Ontario.

Dr. John Humphries - Deceased.

Major General K. A. Hunter, 66 Rollingwood Circle, Orchard Park, London, Ontario, retired as Surgeon-General in December, 1959. He has a son who is also a Doctor.

Dr. William A. Irwin, 2130 Iroquois, Detroit, Michigan, interned at Royal Victoria Hospital in Montreal. He did post-graduate work there and in Ann Arbor, Michigan. At present he is Director of the Department of Radiology at Providence Hospital, Southfield, Michigan. He has three children.

Dr. B. Kelly - Deceased.

Dr. D. M. Lawrason, 503 Fifth St., Medicine Hat, Alberta, is a radiologist in the same city. His wife graduated from Western in 1931.

Dr. H. J. Loughlin, 340 St. James St., London, interned at Harper Hospital in Detroit. He is a Paediatrician in private practice in London.

Dr. A. B. McCallum, 122 Myrtle St., St. Thomas, Ontario, interned at St. Joseph's Hospital in London and then entered medical research.

Dr. J. T. McCullough resides at Elmira, Ontario.

Dr. H. McGuffin - Deceased.

Dr. G. P. Murphy resides at 654 Kingston Rd., Toronto, Canada.

Dr. J. H. O'Brien is at present in Elora, Ontario.

Dr. H. C. Peco, 148 Llydican Extension, Chatham, Ontario, interned at Brantford General Hospital and subsequently set up practice in Chatham, Ontario.

Class of 1950

Dr. John Aldis, 3318 Lakeshore Hwy. E., Burlington, interned at Hamilton General Hospital. He is presently Executive Editor of Modern Medicine of Canada and Coordinating Editor of Applied Therapeutics.

Dr. Craig Arnold, 1877 W. 63rd Ave., Vancouver 14, B.C. interned at Vancouver General Hospital. He is now a specialist in Internal Medicine and is consultant in Medicine at Shaughnessy Hospital. He is married with four children, three girls and one boy.

Dr. Robert R. Austin, 186 Broadway St., Orangeville, interned at Orange Memorial Hospital, Orlando, Florida. He is a member of the College of General Practice of Canada.

Dr. H. M. Barker, 11010 Jasper Ave., Edmonton, Alta., interned at Toronto General Hospital. He did two years post graduate study in child psychiatry at the Children's Service Centre at Wilkes-Barre, Pa., U.S.A. He was assistant professor of psychiatry at the University of Alberta, Edmonton, and director of the Department of Psychiatry's new unit for emotionally disturbed children. He now has a private practice in child psychiatry. He is married with three children.

Dr. Blake Barlow, 143 Thames St., Chatham, is a member of the College of General Practice of Canada.

Dr. James M. R. Beveridge, Ph.D., F.R.S.C., was Dean of Graduate Studies at Queen's University following his association with the Department of Biochemistry at the same University. He has just recently been made President of Acadia University, Wolfville, N.S.

Dr. James Brown, M.D., F.R.C.S.(C), M.R.C.O.G., interned at Toronto Western Hospital and did post-graduate training in London, Ontario; Toronto, and London, England. He is presently a specialist in Obstetrics and Gynecology and is on the active staff of the Hamilton Civic Hospital. He is married and has four children, three girls and one boy.

Dr. Archie Bull, 2130 Gary Cres., Burlington, interned at Royal Jubilee Hospital, Victoria, B.C. He is Director and M.O.H. of the Halton County Health Unit at Milton.

Dr. Frank Butson, 216 Wharncliffe Rd. N., interned at Hamilton General Hospital. He is in General Practice at the same address. He is married with two children.

Dr. Howard S. Cameron, 450 Central Ave., London, Ontario, interned at Ottawa Civic Hospital. He did post-graduate work at Victoria Hospital, Westminster Hospital, Dept. of Anatomy, U.W.O. and Massachusetts General Hospital in Boston. He is an Orthopaedic Surgeon and an instructor in Surgery in the Faculty of Medicine, U.W.O. He is married with five children, three girls and two boys.

Dr. Harold Carry, deceased.

Dr. Gordon Cavell. Last known address was 266 Harbor View Lane, Largo, Florida. He married Corine Jones (Arts '51).

Dr. David A. Clarke, 1935 MacDougall St., Kelowna, B.C., spent a year with the U.B.C. Department of Preventive Medicine. He received his MPH from Harvard. He is presently Director of Public Health for the South Okanagen Health Unit, Kelowna, British Columbia.

Dr. Robert Cowan, 280 E. Thousand Oakes Blvd., Thousand Oaks, California, interned at Hamilton General Hospital and did post-graduate training in opthalmology at the University of Toronto Teaching Hospitals. He received his Diplomate, American Board of Ophthalmology in 1964. He moved to California in July, 1965, after ten years in Guelph, Ontario. He now has a private practice in California. He is married with three children.

Dr. James Cranston, 165 8th St. N.E., Medicine Hat, Alta., interned at Regina General Hospital. He is in General Practice in the Medical Arts Bldg. at Medicine Hat. He is married with two children.

Dr. Wm. Crawford, 4 Field Ambulance, Camp Borden, Ontario, is a Lieutenant-Colonel and Commanding Officer of #4 Field Ambulance, Royal Canadian Army Medical Corps at Camp Borden. He served with the U.N. Emergency Force in the Middle East for a year. He served with the RCAF during the 2nd world war and since then has served in Korea, London, Regina, Winnipeg, Rivers, and Camp Petawawa.

Dr. George Cruickshank, P.O. Box 40, Corunna, Ontario, is a member of the College of General Practice of Canada.

Dr. Robert Detwiler, FRCP & S(C) Surgery (Orthopaedics) lives at 1 Rosecliffe Drive, Hamilton. He interned at St. Paul's Hospital, Vancouver, B.C.

Dr. H. A. DeLuca - Deceased.

Dr. Wm. Downe, 1053 Talbot St., London, interned at St. Michael's Hospital, Toronto. He is a specialist in Psychiatry. He married Susan Thompson (Arts '52)

Dr. Gerald Duck. Last known address - Purvis, Mississippi.

Dr. Donald Duffin, 11 Viola Court, Stratford, Ontario, interned at St. Paul's Hospital, Vancouver. He took further training in internal medicine at Westminster Hospital and in obstetrics and surgery at St. Joseph's Hospital in London before setting up a general practice in London. In 1958 he started training in diagnostic radiology in Kingston and is presently Assistant Radiologist at Stratford General Hospital. He has two boys and a girl.

Dr. Donald Duncanson resides at 2 Princess St., St. Thomas. He won the DFC in World War II and has one daughter.

Dr. H. W. Elder of 170 Woodside Drive, St. Catharines, married Margaret Chattoe (BScN, 1947). They have two children, both sons.

Dr. John Foderick. His last known address was Board of Trade Building, Superior, Wisconsin. He interned at Toronto East General Hospital and did post-graduate training in surgery in the U.S.A. He is married and has three children.

Dr. James Frid, 707 Charlotte Street, Peterboro, Ontario, interned at Victoria Hospital, London and did postgraduate training at the Hospital for Sick Children in Toronto and at Cincinatti Children's Hospital. At present he is a pediatrician in Peterboro. He has three girls and two boys.

Dr. Clair Galbraith of 120 Brian Ave., London interned at Victoria Hospital, London and did postgraduate training in the U.W.O. department of Psychiatry. He is assistant superintendent of the Ontario Hospital, London. He has two boys and a girl.

Dr. Peter Gaskell of 826 Campbell Street, Winnipeg 9, Manitoba, interned at Victoria Hospital, London and received his Ph.D. in Physiology from the University of London. At present he is Associate Professor, Dept. of Physiology, University of Manitoba. His wife, Kathleen Elizabeth (Milligan) graduated from U.W.O. in Honours French and German in 1936. They have two girls and a boy.

Dr. Donald Gillen, 2 South Drive, St. Catharines, interned at Victoria Hospital, London. He lectured in the Dept. of Bacteriology at McGill University and is now in private practice.

Dr. Donald Good, 13 Sterling St., London, Ontario, interned at Waterbury Hospital, Waterbury, Conn. He did postgraduate training at the University of Michigan and the University of London. He served at the Canadian Colombo Hospital in West Pakistan.

Dr. Elliot Goodman of 508 Latmer Road, Merion, Pa., studied clinical cardiology and cardio-pulmonary diseases at Jefferson Hospital, Philadelphia, Pa. According to our last reports he has two daughters.

Dr. Alexander Graham, 85 Stanley Pl., St. Thomas, Ontario. He is on the staff of St. Elgin Hospital. He is Medical Officer for Elgin County Home for the Aged and the NYC Railway.

Dr. Robert Haggar, 877 Kingsway Drive, Burlington, is Director of Laboratories, St. Joseph's Hospital, Hamilton, Ontario. He has two boys and two girls.

Dr. Russell Hall, Main Street, Norwich, Ontario is in general practice. He has four boys.

Dr. Kenneth Hampson, 13 Breadner Place, Trenton, interned at Victoria Hospital, London. He joined the RCAF and studied at the Institute of Aviation Medicine in Toronto. He received his M.P.H. from Harvard.

Dr. John Jewell, 201 Barrington Place, Dearborn, Mich., interned at Grace Hospital, Windsor, Ontario and did postgraduate training at Henery Ford Hospital, Detroit. He is a specialist in obstetrics and gynecology. He has four children.

Dr. Thomas Johnston, 149½ Mitton St., Sarnia, Ontario, married Betty Ramona (Cathcart) (Nurs. '48). They have two boys. He is a urologist in Sarnia.

Dr. Thomas Kane, 1425 Cuthbutson Place, Fort William, Ontario, interned at St. Michael's Hospital, Toronto. He did further study at Sunnybrook Hospital, Toronto. He is a certified internist and has four girls and three boys.

Dr. William Keech of 899 McLaren St., North Bay has three children .

Dr. James Laidlaw, FRCS(C), F.A.C.S., F.A.C.O.G., c/o Christie Clinic, Champaign, Illinois, interned at Royal Victoria Hospital, Montreal. He did further studies in Dept. of Anatomy, U.W.O., Westminster and Victoria Hospitals, London, and Hamilton General Hospital. His wife Corinne received a BScN from Western in 1947. They have four children.

Dr. James Lindon, deceased 1958.

Dr. Peter Marr, 6226 Gilpin St., Burnaby, B.C., interned at Vancouver General Hospital, Vancouver, B.C. In 1957 he received his FRCP & S(C) in Urology. He has a girl and two boys.

Dr. John McCurlie, 2 St. Margaret's Road, Ancaster, Ontario.

Dr. Francis MacDonald, Central Hospital, Kumasi, Ghana, interned at Regina General Hospital and spent several years as pathologist at Toronto Western Hospital. He is presently a pathologist at Central Hospital in Kumasi, Ghana.

Dr. Moncrieff MacDonald, Thedford, Ontario, interned at St. Joseph's Hospital, London.

Dr. Roy McGregor, Box 23, Essondale, B.C., interned at St. Joseph's Hospital in London. After taking a residency in psychiatry, he became Medical Officer in Summerside, P.E.I. at the R.C.A.F. station.

Dr. Gerald MacKichan, 823 Clearview Ave., London, interned at St. Joseph's Hospital, London. He is presently in general practice and is married with two sons and a daughter.

Dr. Douglas MacKinlay, 350 Wellington Street, Sarnia.

Dr. Ian McLaren, 4301 Matthews Lane, Kensington, Maryland, interned at Baylor Hospital, Dallas, Texas. He took his Master of Public Health at University of Pittsburgh and in 1957 became a County Health Commissioner in New York. He is presently the Chief of TB Control for the Health Department in Washington, D.C.

Dr. Donald F. MacLeod, 136 Black Hawk Lane, West Lafayette, Indiana. In 1956 he associated himself with the Student Health Service at Purdue University in Indiana as a specialist in chest diseases.

Dr. Malcolm McNabb, Kapliolani Maternity and Gynecologic Hospital, 164 Bingham Street, Honolulu, Hawaii, interned at Millard Fillmore Hospital in Buffalo, New York. He received post-graduate training in the U.S. army corps and at Toronto Western Hospital. After practising in Aldershot, Ontario, he became assistant resident at Kaliolani Hospital in 1963.

Dr. Joseph Meehan, 293 Winona Drive, Toronto, Ontario, interned at St. Michael's in Toronto and is presently in general practice in Toronto.

Dr. David Meltzer, 1054 Brough Street, London, interned at Victoria Hospital, London. He studied Hematology at Victoria Hospital and Boston, Massachusetts. He received his F.R.C.P. in 1955. He is presently staff hematologist at St. Joseph's Hospital and Westminster Hospital in London and is Assistant Professor in Medicine and Pathological Chemistry at the U.W.O. Medical School. He is married with a son and a daughter.

Dr. Jule J. Merritt, 36640 South Gratiot Avenue, Mt. Clements, Michigan, interned and was a resident in urology at Grace Hospital in Detroit. He did further post-graduate work at the University of Pennsylvania, in St. John's, New Brunswick and at Sinai Hospital in Baltimore. He is presently a practicing urologist in Michigan.

Dr. Geoffrey Miller, 10 Beechwood Place, London, served for a period of time as an officer of the Alumni Executive. He has a daughter and a son.

Dr. Arthur A. Moyen, 867 A Waterloo Street, London.

Dr. Dennis Morey, 8 Westmoreland Avenue, Richmond, Virginia.

Dr. James Murray (W/C), #30658, 1 Air Division, RCAF, Canadian Armed Forces, Europe.

Dr. Geoffery L. Nanson, 6111 MacKenzie St., Vancouver 13, B.C.

Dr. Sigmund Nielson, 2057 Caroline St. E., Burlington, Ont.

Dr. Melvin Pastorius, 5047 Middlesex, Dearborn 1, Michigan, interned at Cook Memorial Hospital, Hartford, Conneticut, and then did a year of surgical residency at the Lutheran Hospital in Cleveland, Ohio and three years of surgical residency

at the Henry Ford Hospital in Detroit. Along with his wife, a nurse, he spent some time in medical missionary work at the Umri Mission Hospital in India and returned to Detroit in 1962.

Dr. Keith Phillips, 441 Chemberlain Lane, Oakville spent two years interning at Hamilton General Hospital. Besides his general practice he is also coroner for Halton County. He has four children.

Dr. Norman Price, 227 Queensdale Avenue E., Hamilton interned at Hamilton General Hospital.

Dr. Raymond Prince, 4280 Western Ave., Apt. 3 Montreal, interned at Toronto General Hospital. He later obtained his MSc in Neurocytology at U.W.O. and his Certification in Psychiatry. At present he is Assistant Professor in the Department of Psychiatry at McGill University. He is married to Mary Grace Keenan and they now have eight children.

Dr. Ruby Raikov - deceased.

Dr. Lorne Rhamey, 69 Arcade Cr. Hamilton, interned at St. Joseph's Hospital in Hamilton and took his Diploma - Psychiatry at the University of Toronto. He is now in private practice. He is married with two sons and one daughter.

Dr. Doug. Richardson, 145 Wellington St., Sarnia is married with one daughter and a son.

Dr. Wm. Richardson, 14 George St. N., Galt, interned at St. Joseph's Hospital in London after which he took two years of post-graduate training in psychiatry. He is now in general practice and has three children.

Dr. Ken Ritchie, 31 Marlboro Rd., Port Arthur, is Medical Officer of Health for Dorion Township.

Dr. Elgin Roberts, 1041 Comox St., Vancouver 5, B.C., did his senior internship at Esperanza General Hospital, Esperanza B.C. and at Shaughnessy Hospital. In September, 1965, he became a Research Fellow at the Faculty of Medicine at the University of Manitoba.

Dr. James Roberts, c/o St. Thomas Elgin General Hospital, St. Thomas, Ontario, interned at St. Joseph's Hospital in Hamilton. After doing post-graduate work in the Department of Anatomy at U.W.O. in 1951 he went to the Vancouver General Hospital. In 1953 he returned to London and Westminster Hospital then in 1955 did some more work at U.W.O. Medical School. In 1962 he did a post-graduate course in radiology at Peter Bent Brighan Hospital in Boston, Mass.

Dr. Peter Roberts, 170 Douglas Street, Stratford, interned at Victoria Hospital in London followed by post-graduate training at Elizabeth Hospital, Prairie Grove, Arkansas. His wife graduated from U.W.O. in Home Economics and they now have three children.

Dr. Harold Robinson, 6 Amberly Pl., R.R.1, Box 442, Ottawa, is now Wing Commander, 3(F) Wing R.C.A.F.

Dr. Edward Roemmele, 433 Askin Blvd., Windsor, is in general practice in Windsor. He interned at the Hôtel Dieu Hospital in Windsor. He is married with two daughters.

Dr. Earl Russell, 86 MacDonnell St., Kingston, Ont., is at present working in the Dept. of Anaesthesia at the Kingston General Hospital. In 1962 he accepted a teaching post with the University of Lagos Medical School in Nigeria where he was instrumental in opening a new teaching hospital and establishing a new medical faculty. He married Marjorie Mills and they now have four daughters.

Dr. Gerald St. Pierre, 712 Devonshire Rd., Walkerville P.O., Windsor, married Rosemary Traynor, a U.W.O. graduate in Public Health and they have two daughters. He is in private practice.

Dr. Ralph Schenck, 603 W. Arch St., Portland, Indiana, interned at Methodist Hospital, Gary, Indiana. He took his residency at Methodist Hospital, Indianapolis, Indiana and is at present a general surgeon. In addition he is Coroner for Jay County, Indiana. He has four children.

Dr. Gordon Skelhorne, 43791 West 6th Avenue, Vancouver, B.C.

Dr. Donald Steepe, 278 Wellington Heights, Sudbury, Ont., did post-graduate training in pediatrics at the Henry Ford Hospital in Detroit.

Dr. Thomas Stewart, 267 O'Connor St., Ottawa 4, entered private practice in Ottawa after doing his internship at the Royal Victoria Hospital in Montreal.

Dr. Chelsea Thiel Capo, 5050 Canadian Armed Forces, served as medical officer for the 3rd Battalion of the Royal Canadian Regiment during the Korean War. He obtained a degree in anesthesiology and is married with one daughter.

Dr. Murray Thompson, 18 Hill Crescent, Scarboro, Ontario, interned at Hamilton General Hospital. He did post-graduate training at the Mayo Clinic and is presently Chief of the Department of Anaesthesia at Scarboro General Hospital. He is married and has five children.

Dr. Roger John Thompson, 92 Henderson Dr., Penfield N.Y., interned at Hamilton General Hospital and did his post-graduate training in Anaesthesia at Montreal General Hospital. He is now in the Department of Anaesthesia, Highland Hospital, Rochester, N.Y. He married the former Dorothy Selena, BScN '47.

Dr. Wm. Tomlinson, Box 250, resides in Pickering, Ontario. He is a member of the active staff of Ajax, Pickering, Oshawa General and Toronto Eastern General Hospitals and is the Industrial Physician for Canadian Automotive Trim, Ajax, and is a district coroner.

Dr. Howard Vernon resides at 1265 Clarkson Rd., Clarkson, Ontario. He married the former Elizabeth Andrews, Meds '51.

Dr. M. James Warden resides at 2120 Mendocino Lane, Altadens, California (ZIP 91001). He is presently Head of Pediatric Surgery, Kaiser Foundation Hospital, Los Angeles, California. He married the former Jane Charlotte Carlson.

Dr. Maxwell Weaver. Last known address is Box 398, Smithers, B.C.

Dr. Rollin Leonard Weston, 105 Anndale Dr., Willowdale, interned at St. Joseph's Hospital, London and did his post-graduate training in Toronto. He is now an anaesthetist on the Staff of Toronto Western Hospital.

Dr. John G. White, Box 308, 42 Judith Cres., Ancaster, is Assistant Superintendent of the Ontario Hospital at Hamilton.

Dr. Hugh J. Williams resides at 5 Sunset Lane, North Oaks, St. Paul 10, Minnesota. From 1951-56, Hugh was in General Practice in Morocco, Indiana. He then proceeded to obtain his Fellowship in Radiology at the Mayo Clinic and his M.Sc. (Radiology) from the University of Minnesota. At present, he is Radiologist at Children's Hospital, St. Paul and C. T. Miller Hospital also in St. Paul, and Clinical Instructor in Radiology at the University of Minnesota.

Dr. Paul Yates, Tungan Magajiya, via Kontagora, Nigeria, West Africa, is Medical Superintendent of the Nigeria Field of the United Missionary Society. He is married to the former Marie Clendenen and has four children.

BOOK REVIEWS

LEOPOLD'S PRINCIPLES AND METHODS OF PHYSICAL DIAGNOSIS: Henry U. Hopkins, 503 pp., Illust., Third Edition, W. B. Saunders Company, Philadelphia and London. $9.20.

The revision for this Third Edition was begun by Dr. Leopold. After his death it was completed by Dr. Hopkins who had worked closely with Dr. Leopold for many years. Consequently those familiar with Editions One and Two will find the same basic style in the Third Edition.

Some changes are in evidence. In this new Edition a whole chapter has been devoted to "The Examination of the Skin". In previous editions much of this material was scattered throughout other areas of the text.

This book begins with a chapter on "The History". Succeeding chapters deal with the psychiatric survey of the patient, vital signs, examination of the head, neck, and breasts. Several chapters are devoted to the examination of the thorax and lungs. Later chapters cover the examination of the heart, abdomen, genitalia, musculoskeletal and neurological systems. The concluding chapter of the book deals with the special problems of the pediatric examination.

The index and table of contents are very clear and complete so that the discussion relative to a specific condition can be readily found. The individual sections of each chapter are clearly indicated by bold face type for easy reading. The discussion in each section is brief and pointed. This is excellent for the student with some experience in physical diagnosis, but for the less experienced student the precise presentation may be somewhat unsatisfactory. This is especially true regarding abnormal findings, their meaning, and the range of normals that can be expected. However it was not the author's purpose to be exhaustive, as the text would undoubtedly suffer in terms of utility from an attempt to achieve completeness.

The photographs and charts are in black and white only. On the whole these demonstrate adequately the case they are representing. This is especially helpful to the student.

This book would be a valuable adjunct to the library of medical students who are beginning to learn the art and science of physical diagnosis.

Reviewed by Elizabeth Musclow '66

BOOKS RECEIVED

The receipt of the following books is acknowledged; the books that appear to be of particular interest will be reviewed as space permits.

SYNOPSIS OF CARDIOLOGY: by William J. Gefter, Bernard H. Paster, Ralph M. Myerson. 4° cloth. 12+839+37 pp. with 240 illustrations. St. Louis: The C. V. Mosby Company, 1965. Price $9.85.

FUNDAMENTALS OF ACID-BASE REGULATION: by James R. Robinson M.D., PhD., Professor of Physiology, University of Otago Medical School, New Zealand. 2nd Ed. 10+75+5 pp. Paperback. Oxford: Blackwell Scientific Publications available in Canada from the Ryerson Press, Toronto, 1965. Price $2.50.

LECTURE NOTES ON GENERAL SURGERY: by H. Ellis and R. Y. Caline, Dept. of Surgery, Westminster Hospital, London. 10+369+12 pp. 4° cloth. Oxford: Blackwell Scientific Publications available in Canada from The Ryerson Press, Toronto, 1965. $9.25.

SYNOPSIS OF CARDIOLOGY: by W. I. Gefter, M.D., B.H. Pastor, M.D., and R. M. Myerson, M.D., 877 pages, 240 illustrations, The C. V. Mosby Company, 1965, $9.85.

This excellent synopsis presents clinical cardiology in a most clear and concise fashion. The authors have attempted to summarize the voluminous literature in this field into a simple readable text without sacrificing completeness.

The book most appropriately begins with a section on the "Cardiovascular Examination" followed by sections on "Circulatory Failure", "Arrhythmias", "Types and Causes of Heart Disease", "Diseases of the Vascular System", and "Special Problems". The last section includes such up to date problems as anticoagulant therapy, prognosis and insurability of the cardiac patient, and air travel and the cardiac patient.

The style of writing is most brief, but not to the point of being staccatto and difficult to follow. The book is arranged in note-book fashion with heavy typed headings and subheadings. The majority of topics are well illustrated with numerous x-rays, electrocardiograms and simple line diagrams; however, no photos are used.

The above points make this book especially suitable for a student text as well as a ready-reference for the practitioner. The authors have successfully incorporated a voluminous amount of material into a relatively small and inexpensive basic cardiology synopsis.

Reviewed by Kelly Jones '66.

LECTURE NOTES ON GENERAL SURGERY: by Harold Ellis and Roy Yorke Calne, 382 pages, Blackwell Scientific Publications, A. R. Mowberry and Co. Ltd., Publishers, Oxford, 1965, $9.25, (Ryerson Press).

This textbook attempts to present a concise summary of general surgery and is aimed at the final year medical student. It fulfills its purpose as a general surgery summary well for the students who have studied surgery in some detail previously.

Each topic is organized well under various headings which leaves the student with a grasp of the important facts. Where applicable. a note on embryology is present. The relatively important and common facets of surgery eg. appendicitis, have adequate coverage.

This book does not cover orthopedics; however a companion text on orthopedics is being planned. Although the text has 35 illustrations it suffers from a lack of additional schematic drawings in certain chapters. As previously mentioned there is a good embryological orientation with each organ system but a review of the gross anatomy of the various systems is lacking. A short anatomical review with each topic would be an asset to this text.

Conclusion: This is a well written medical text in the usual English tradition. This book would lend itself to reproduction in a cheaper paperback edition which would make it more attractive to the average financially embarrassed medical student.

Reviewed by E. Franczak '66.

FUNDAMENTALS OF ACID-BASE REGULATION: by James R. Robinson M.D. Ph.D., 2nd printing, Scientific Publications, Oxford, 1963. 80 pages, $2.25.

This book is a useful addition to any medical student's collection. It explains simply and clearly the subject of acid-base balance which has always been a difficult

problem for most to understand. The author begins by outlining basic principles and then expands to a more detailed and complex discussion. The functions of the respiratory and renal systems with regard to pH are particularly well outlined.

It is beneficial to have a brief review of this subject available in order to understand the physiological basis of many medical emergencies, (e.g. diabetic acidosis, salicylate poisoning) and to carry out their treatment intelligently.

A similar book outlining the principles of fluid and electrolyte balance has been published by the same author and would also be helpful to the student.

Reviewed by D. V. Edwards '66