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THE EMERGENCE OF EMOTION

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Abstract

Emotion is conscious experience. It is the affective aspect of consciousness. Emotion arises from sensory stimulation and is typically accompanied by physiological and behavioral changes in the body. Hence an emotion is a complex reaction pattern consisting of three components: a physiological component, a behavioral component, and an experiential (conscious) component. The reactions making up an emotion determine what the emotion will be recognized as. Three processes are involved in generating an emotion: (1) identification of the emotional significance of a sensory stimulus, (2) production of an affective state (emotion), and (3) regulation of the affective state. Two opposing systems in the brain (the reward and punishment systems) establish an affective value or valence (stimulus-reinforcement association) for sensory stimulation. This is process (1), the first step in the generation of an emotion. Development of stimulus-reinforcement associations (affective valence) serves as the basis for emotion expression (process 2), conditioned emotion learning acquisition and expression, memory consolidation, reinforcement-expectations, decision-making, coping responses, and social behavior. The amygdala is critical for the representation of stimulus-reinforcement associations (both reward and punishment-based) for these functions. Three distinct and separate architectural and functional areas of the prefrontal cortex (dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex) are involved in the regulation of emotion (process 3). The regulation of emotion by the prefrontal cortex consists of a positive feedback interaction between the prefrontal cortex and the inferior parietal cortex resulting in the nonlinear emergence of emotion. This positive feedback and nonlinear emergence represents a type of working memory (focal attention) by which perception is reorganized and rerepresented, becoming explicit, functional, and conscious. The explicit emotion states arising may be involved in the production of voluntary new or novel intentional (adaptive) behavior, especially social behavior.

Key Words: amygdala, prefrontal, conscious, experience, feedback, nonlinear, explicit

INTRODUCTION

Emotions are self-generated internal explicit states, arise rapidly in recurrent systems, take time to form, have a prolonged duration, are seamless, structurally-complex, ineffable, transparent, bounded, unified and coherent, informative, serial, limited in capacity, subject to interference, new or novel, variable, flexible, project outwards, can gain access to other systems, and arise from attention. All these properties of emotion have also been ascribed to conscious experience (Baars,1988;Koch,1998;Tononi & Edelman,1998). Because emotion has all the same properties as conscious experience, emotion must be conscious experience (Liebnetz's Law). An emotion is defined as a conscious mental reaction subjectively experienced as strong feeling which is usually directed towards a specific object and is typically accompanied by physiological and behavioral changes in the body; emotion is the affective aspect of consciousness (Merriam-Webster). In other words, an emotion is defined as a conscious experience. Emotion therefore is conscious per se. Conscious experiences (including emotions) make up the content of consciousness (what is in consciousness). The conscious experience aspect of emotion, apart from the bodily changes, is known as "affect" (Merriam-Webster) and an emotion may be referred to as an "affective state".

An emotion therefore has three components of variable intensity and prominence: a physiological component (various physiological reactions), a behavioral component (various behavioral reactions), and an experiential (subjective, feeling, conscious) component (LeDoux,1990). Particular combinations of these reactions are recognized as particular kinds of emotion. Fear, for example, may consist of increased sympathetic autonomic discharge, the release of stress hormones (adrenaline, cortisol), breakdown of energy reserves, increase in heart rate and respiratory rate, sweating, flight, cowering, freezing, fearful facial expression, fearful vocal expression, and subjective conscious experience of fear. Other combinations of reactions may also be recognized as fear. Happiness may consist of increased parasympathetic autonomic response, release of energy storage and conservation hormones (insulin, androgens, estrogens, oxytocin, growth hormone), storage of energy reserves, slowing of heart rate and respiration, receptive open approach behavior, smiling, happy facial expression, happy vocal

expression, and subjective conscious experience of happiness. Similarly, other emotions are recognized as particular combinations of physiological, behavioral, and experiential reactions. The reactions making up an emotion can vary in kind, number, intensity, and prominence. Different emotions may share some of the same reactions. Thus we have many varieties and degrees of fear, joy, anger, happiness, or other emotion.

Emotions arise from sensory stimulation. It is often difficult to connect emotions to the sensory stimulation from which they arise, as emotions are not inherent in or in any way part of the sensory stimulation itself (this is true of all conscious experiences). Emotions are composed entirely by the person (they are subjective). We may consciously experience sensory stimulation without emotion. For example, we may consciously experience a rock or a pool of water without emotion. However, anger or fear may become part of the conscious experience of the same rock (if it just hit us) or the same pool of water (if we had almost drowned in it). We may also experience different emotions (fear, anger, love, hate, sympathy, etc.) to the same sensory stimulation (a specific person) at different times. An affective aspect is added to conscious experience to produce emotion.

THE AFFECTIVE ASPECT OF CONSCIOUSNESS

It has been demonstrated by a number of investigators that there are two opposing systems in the brain for the development of an affective value or valence for sensory input (Gray,1991;Heath in 1952,see MacLean,1969;Kissin,1986;LeDoux,1990;Murray et al,2009; Olds & Milner,1954;Paton et al,2005). This affective valence has physiological, behavioral, and experiential components (LeDoux,1990). LeDoux (1990) and Kissin (1986) found that affective valence for sensory stimulation is developed by fixed and plastic neural mechanisms that can facilitate or inhibit further stimulus processing at the brainstem, thalamic, and cortical levels. The two systems are called the reward (pleasure, approach) and punishment (pain, avoidance) systems. Every emotion has an affective valence (it may be regarded as a positive or negative emotion) and appears to arise from affective valence.

REWARD SYSTEM

Activation of the reward system has rewarding pleasurable facilitating approach effects. The reward system of the brain follows the course of the medial forebrain bundle (Kissin,1986).

It has been demonstrated through electrical stimulation of the olfactory bulbs, hippocampus, amygdala, cingulate cortex, substantia nigra, tegmentum, locus coeruleus, raphe nuclei, nucleus basalis of Meynert, caudate, putamen, lateral hypothalamus (had the greatest effect), thalamus, reticular formation, medial forebrain bundle, and orbitofrontal cortex (Best,2010;Kringelbach & Rolls,2004;Olds & Forbes,1981;Rang, 2003;Volz et al,2008;Waraczynski & Stellar,1978). The reward system is activated by rewarding conditioned and unconditioned stimuli, positive reinforcers, brief stimuli, low-intensity stimuli, and the cessation or absence of punishing stimuli or negative reinforcers (Gray,1991;Joseph,1990,2000;Kissin,1986). Base nuclei (ventral tegmentum, substantia nigra, nucleus basalis, locus coeruleus, raphe) which control four major neurotransmitter systems of the brain (dopamine, acetylcholine, noradrenaline, serotonin) are part of the reward system and mediate the effects of the system (Best,2010;Rang,2003). Activation of the reward system has arousal, alerting, facilitating, rewarding effects, which are also the effects obtained through activation of the above four neurotransmitter systems (Bakin & Weinberger,1996;Best,2010;Davis et al,1991; Frank et al,2004;Kapp et al,1990;LeDoux,1990;Rang,2003; Weinberger et al,1990). There is evidence that variations in affective valence have a differential effect on mesocortical, mesolimbic, and nigrostriatal dopaminergic projections (Bertolucci-D'Angio et al,1990;Deutch & Roth,1990). Deutch and Roth (1990) found that this depended on neuropeptide release. Gray (1991) and Maier (1991) found that acetylcholine, noradrenaline, serotonin, and opioid systems may also be influenced by affective valence. The phasic burst firing of ventral tegmental dopamine neurons and the accompanying rise in dopamine release normally occurs in response to primary rewards and reward-predicting stimuli (Schultz,1997). Diminished stimulation of mesolimbic dopamine release may result in the absence of behavioral incentive, apathy, and anhedonia (Drevets et al,1998). Excessive stimulation of mesolimbic dopamine release may result in exaggerated hedonia response and elevated motivational drive (Drevets et al,1998). Dopamine appears to modulate the reward value of stimuli. The reward system is closely associated with the reticular activating system. The reticular activating system essentially originates in the upper brainstem reticular core and projects through the intralaminar thalamic nuclei to the cerebral cortex to mediate arousal, attention, and consciousness, ie. the waking state (Evans,2003; Hannaman,2005;Kinomura et al,1996;Reiner,1995;Steriade,1995, 1996;Young,2009). Bilateral damage to the intralaminar thalamic nuclei produces lethargy or somnolence (Steriade,1996). Damage

to the reticular activating system results in coma (Daltrozzo et al,2009,2010;Liversedge & Hirsch,2010;Young,2009). Partial destruction or inhibition of the reticular activating system results in a partial loss of consciousness known as acute confusion state or delirium (Rull,2011). The reticular activating system and the cerebral cortex are necessary to maintain consciousness (Hannaman,2005;Young,2009). The noradrenergic locus coeruleus, the mesencephalic reticular formation, the cholinergic nucleus basalis of Meynert, the dorsal hypothalamus, the intralaminar thalamic nuclei, and the tegmentum make up the reticular activating system (Afifi & Bergman,1986;Best,2010;Hannaman,2005;Kinomura et al,1996;Rang, 2003;Steriade et al,1990;Thierry et al,1990;Vertes,1990). The function of the reticular activating system is modulated by the adrenergic and cholinergic components (Burlet et al,2002;Evans, 2003;Garcia-Rill,1997;Garcia-Rill et al,2007;Reiner,1995). Hence much of the reticular activating system is also part of the reward system and the two systems are activated together. Following damage to the lateral hypothalamus (the reward system), pleasurable sensation and emotional responsiveness were reduced (Olds & Forbes,1981) and faces became blank and without expression (Joseph,1990). Unilateral lesions may produce neglect and indifference (loss of consciousness and emotion) to all contralateral sensory stimuli (Marshall & Teitelbaum,1974). Activation of the reward system has anabolic and restorative physiological effects, acts to facilitate further stimulus processing, stimulates attention and consciousness, generates positive emotion, produces approach behavior, and the individual works to prolong or maintain the rewarding stimulation (Gray, 1991;Joseph,1990,2000;Kissin,1986). Hence the reward system acts to produce the three components of positive affective valence and emotion.

PUNISHMENT SYSTEM

Activation of the punishment system has punishing inhibiting avoidance negative effects. The punishment, avoidance, or pain system consists of the entorhinal cortex-hippocampus-subiculum-septal nucleus circuit, cingulate cortex, amygdala, medial hypothalamus, parasympathetic nervous system, reticular formation, periaqueductal gray (involved in pain transmission), thalamus, basal ganglia, parietal cortex, and frontal lobe (Afifi & Bergman,1986;Ganong,1988;Gray,1991;Joseph,1990,2000; Kissin,1986;Kringelbach & Rolls,2004;Volz et al,2008). The punishment system is activated by punishing conditioned or unconditioned stimuli, negative reinforcers, long-duration stimuli, high-intensity stimuli, painful stimuli, and the

cessation or absence of rewarding stimuli and positive reinforcers (Gray,1991;Joseph,1990,2000;Kissin,1986). The punishment system consists of a number of limbic structures involved in memory and negative emotion (septal nuclei, amygdala, medial hypothalamus, frontal cortex). Activation of the punishment system also activates the reticular activating system (via the amygdala, hypothalamus, thalamus, reticular formation, septal nuclei). The punishment system has catabolic physiological effects, a negative inhibiting effect on processing, stimulates attention and consciousness, produces avoidance or aggressive behavior, and generates negative emotion. The individual works to attenuate further stimulus processing and response and may enter a state of quiescence and behavioral inhibition or aggression and attack (Gray,1991; Joseph,1990,2000;Kissin,1986). The punishment system acts to produce the three components of negative affective valence and emotion.

AFFECTIVE VALENCE (STIMULUS-REINFORCEMENT ASSOCIATION)

The two affective valence systems appear to work in opposition. The development of affective valence begins early in sensory processing, long before a sensory stimulus is recognized cognitively and consciously in the cerebral cortex (Kissin, 1986;Joseph,1990,2000). The effects of affective valence are relatively primitive and reflexive at subcortical levels but may be refined into higher-order emotional reactions at higher limbic and neocortical levels (Kissin,1986;Joseph,1990,2000). The affective valence systems act in close association with the reticular activating system for the regulation of attention and consciousness. This may be important in the formation of emotion. The reticular activating system is also active during other states of consciousness (inattentive states, meditation, and hypnosis) enabling the production of emotion during these states as well (Svorad,1957). The affective valence systems appear to be instrumental in the development of emotion. Affective valence might be considered a determination of the emotional significance of a sensory stimulus and the first step in the generation of an emotion.

AMYGDALOID NUCLEI

The amygdaloid nuclei are small almond-shaped structures found in the deep medial-temporal region of the brain, just in front of the hippocampus. The basolateral amygdaloid nuclei receive input from sensory pathways (Perryman et al,1987;Schutze et al, 1987;Turner et al,1980;Van Hoesen,1981), reticular formation,

specific and non-specific thalamic nuclei, olfactory pathways, anterior cingulate cortex, medial and lateral hypothalamus, and orbitofrontal cortex (Afifi & Bergman,1986;Blair,2008;John, 2009). The central amygdaloid nuclei project to a large number of structures in the brain that are involved in the behavioral, humoral, autonomic (physiological), and experiential signs of emotional reactions: the central grey for the arrest of ongoing behavior (freezing) or for the production of fight/flight behavior, the dorsal motor nucleus of the vagus nerve for changes in heart rate, the hypothalamus for changes in blood pressure and other autonomic changes, the parabrachial nuclei for changes in respiration (panting, respiratory distress during panic), the nucleus reticularis pontis caudalis for an increase in reflex excitability (startle response) and for the production of orientation and attention, the trigeminal motor nuclei for jaw movements, the facial motor nuclei for facial expressions, and the ventral tegmental area for an increase in dopaminergic, cholinergic, and noradrenergic actions in the cerebral cortex for increased vigilance, alerting, cortical processing, and learning (Davis et al,1991;Kapp et al,1990;LeDoux,1990; Weinberger et al,1990). The amygdala is critical for the representation of stimulus-reinforcement (affective valence) associations (both reward and punishment-based) and the processing of emotional expressions (Blair,2008). Distinct neurons in the amygdalae respond to positive and negative stimuli, but these neurons are not segregated to specific nuclei (Paton et al,2005). The basolateral nucleus does more than simply control the central nucleus. It enables the transmission of stimulus-reinforcement (affective valence) association information forward to the ventromedial prefrontal cortex (areas 10 and 11 of the orbitofrontal cortex, ventral anterior cingulate cortex) for emotion regulation, reinforcement-expectancies for decision-making, and social behavior (Blair, 2008;Budhani et al,2007;Etkin et al,2006;Pezawas et al,2005; Phillips et al,2003a,2003b;Rauch et al,2006;Rushworth et al, 2007;Schoenbaum et Roesch,2005;Volz et al,2008). Blair (2008) found that these critical functions are impaired in psychopathy (marked by emotional dysfunction, antisocial behavior, and poor decision-making) and result in its development.

The amygdala plays a primary role in emotion and memory (Amunts et al,2005;Blair,2008). People shown threatening faces, confronted with frightening situations, or with severe social phobia showed increased activation of the amygdala (Nathan et al,2006). Depressed patients showed left amygdala hyperactivity when interpreting emotions for all faces, especially fearful faces, and this hyperactivity was normalized when the patients

went on antidepressants (Sheline et al,2001). Bipolar patients have considerably smaller amygdala and hippocampal volumes (Blumberg et al,2003). Normal subjects exposed to frightening faces or faces of people from another race showed increased activation of the amygdala, even if the exposure was subliminal (Williams et al,2006). However, the amygdala is not necessary for the processing of fear-related stimuli, since people which have bilateral amygdala damage show a rapid reaction to fearful faces, even in the absence of a functional amygdala (Tsuchiya et al,2009). As early as 1888, Brown and Shafer showed that temporal cortex lesions (including the amygdala) in monkeys produced severe deficits in emotion and social behavior. Kluver and Bucy (1939) showed that large anterior temporal lobe lesions caused overreaction to all objects, hypoemotionality, hypersexuality, hyperorality, and loss of fear (Kluver-Bucy Syndrome). Terzian and Ore (1955) found that amygdala lesions reduce or abolish aggressive or violent behavior while Bunnel (1966) found such lesions produce social-emotional agnosia. In 1981, Appleton and Passingham found that lesions of the whole amygdala caused Kluver-Bucy Syndrome. Emotionally-reflected speech, the ability to sing, and the ability to carry melody may be affected with right-sided amygdala lesions (Joseph,1988a, 1990). Stimulation of the amygdala may produce sustained orientation and attention, fear and/or anger, rage reactions (Egger & Flynn,1963;Gunne & Lewander,1966;Ursin & Kaada,1960; Zybrozyna,1963), intense changes in facial expressions, grimacing, baring the teeth, pupil dilation, licking, chewing, smacking of the lips, tearing (Anand & Dua,1955;Ursin & Kaada, 1960), and emotional sounds (Robinson,1967;Ursin & Kaada,1960). Stimulation of the amygdala increases sexual activity and aggression, while harm to the amygdala causes the opposite effects (Brink,2008). The medial-temporal region of the brain is prone to develop seizure activity (temporal lobe epilepsy-Joseph,1990). Emotional reactions may be distorted or inappropriate in temporal lobe epilepsy (Gloor,1960;Joseph, 1990;Kluver & Bucy,1939).

Much of the wiring system of the amygdala appears to be innate, but it is subject to plastic change and learning (Davis et al, 1991). The amygdala represents the positive or negative value (valence) of sensory stimuli during learning (Blair,2008;Paton et al,2005). The amygdala has been implicated in the acquisition and expression of classical (Pavlovian) fear conditioning (a form of conditioning of emotional responses) and long-term potentiation appears to be involved (Amunts et al, 2005;Blair et al,2001;Helmuth,2003;Marin,1999;Pare et al,2002; Paton et al,2005;Sapolsky,2003). Damage to the amygdala was

shown to impair the acquisition and expression of Pavlovian fear conditioning (Amunts et al,2005). The amygdala is also involved in appetitive (positive) conditioning (Blair,2008;Paton et al, 2005).

The amygdala plays a primary role in the formation and storage of memories associated with emotional events and it appears to be the role of the amygdala to regulate memory consolidation in other brain regions (Marin,1999). The amygdala has many connections with the hippocampus (more going to the hippocampus than received from it-Ben Best,The amygdala and the emotions, *Anatomical Basis Of Mind*,Ch.9,see website) and emotions can facilitate the encoding of memories (Gasbarri & Tomaz,2012). Increased amygdala activation immediately after an event (such as emotional arousal) increases the strength and retention of the memory of that event (Asari et al,2010;Hutcherson et al, 2008;Nathan et al,2006;Pare et al,2002). The more emotionally-arousing the event, the greater is the activation of the amygdala (especially the basolateral nuclei) and the greater the strength and retention of the memory of the event.

The amygdala plays a substantial role in mental states and is related to a number of psychological disorders. Lesions of the medial temporal lobe usually produce in their early stages severe emotional symptoms, fear, aggression, anxiety, irritability, inappropriate sexual activity, over-attention to external stimuli, distractibility, periods of apathy or restlessness, paranoid symptoms, and hallucinations (Bogarts, 1997). The amygdala plays an important role in modulating vigilance, social relations, and generating negative emotional states and is abnormally reactive in mood and emotional disorders (Donegan et al,2003). Donegan and coworkers (2003) found that amygdala hyperreactivity contributes to hypervigilance, emotion dysregulation, and disturbed interpersonal relations in borderline personality disorder. Amygdala connections are more widespread from the left amygdala in heterosexual women and homosexual men, while amygdala connections are more widespread from the right amygdala in heterosexual men and homosexual women (Swaab,2007,2008). The amygdalae of males also tend to be larger than those of females (Caviness et al,1996;Goldstein et al,2001). These differences might account for some of the differences in emotional expression and social behavior of men and women. Amygdala volume correlates positively with both size and complexity of social networks (Bickart et al,2010;Szalavitz et al,2010). People with larger amygdalae have larger and more complex social networks than people with smaller amygdalae. People with larger

amygdalae also make more accurate social judgments about other people's faces (Bzdok et al,2011). Larger amygdalae may allow for greater emotional intelligence, enabling better social integration and cooperation (Buchanan et al,2009). There appear to be functional differences between the two amygdalae. Stimulation of the right amygdala induced negative emotions, while stimulation of the left amygdala induced either positive or negative emotions (Lanteaume et al,2007). Other evidence indicates the left amygdala is part of the reward system (Murray et al,2009).

PREFRONTAL CORTEX

The prefrontal cortex is the central executive of the brain, mediating specific functions carried out by other cortical and subcortical structures (Petrides & Pandya,2007;John,2009). As will be seen, the prefrontal cortex is also involved in focal attention and the formation of conscious experiences, including emotions. The prefrontal cortex consists of three distinct cytoarchitectural and functional areas (John,2009;Petrides & Pandya,1999;Preuss & Goldman-Rakic,1991). The dorsolateral prefrontal cortex is the central executive for cognitive control, while the orbital prefrontal cortex (orbitofrontal cortex) is the central executive for emotion and social control (Stuss & Levine,2002). The medial prefrontal cortex (anterior cingulate cortex) mediates drive or motivation (Licter & Cummings,2001;Stuss et al,1986). Each subdivision of the prefrontal cortex does not produce its functions exclusively. Each is connected to a series of subcortical areas (striatum, pallidum, substantia nigra pars reticulata, subthalamic nucleus, thalamus) which lead back to the same prefrontal subdivision from which it arose to form a functionally-segregrated circuit or loop (Alexander et al,1986;Dolan et al,1999). These prefrontal-subcortical circuits have open connections with other cortical and subcortical areas, of which those with the temporal-limbic cortex (left hippocampus, parahippocampal gyrus, amygdala, pallidal internal segment, superior temporal gyrus) appear to be the most important (Bogarts,1997;Petrides & Pandya, 2007). Specific neurobehavioral syndromes have been linked to preferential involvement of each of these circuits (Duffy & Campbell,2001). These are the dysexecutive syndrome, the disinhibition or pseudopsychopathic syndrome, and the apathetic or pseudodepressive syndrome, linked to preferential involvement of the dorsolateral, orbitofrontal, or anterior cingulate circuits respectively (Duffy & Campbell,2001;Licter & Cummings, 2001). These syndromes, along with the behavioral disorders associated with dysfunction of brain areas connected through

open connections, bear striking resemblance to the symptom dimensions and neurocognitive dysfunctions seen in the group of conditions collectively known as schizophrenia (John,2009).

DORSOLATERAL PREFRONTAL CORTEX (DLPFC)

The DLPFC consists of areas 6 and 8, the lateral part of areas 9-12, and areas 44-47 (Joseph,1990;Philip & Miller,2002). The DLPFC is connected to the orbitofrontal cortex, anterior cingulate cortex, thalamus, basal ganglia, hippocampus, and temporal, parietal, and occipital cortex (Alexander et al,1990; Cavada,1984;Fuster,1989;Goldman-Rakic,1990;Groenewegen et al, 1990;Kolb,1990;Pandya & Yeterian,1990;Philip & Miller,2002; Uylings & Van Eden,1990). The DLPFC is involved in the integration of sensory and memory information, the regulation of intellectual function, and is the highest cortex for motor planning, organization, and regulation (Fuster,1997,2000,2008; Goldman-Rakic,1990;Hale & Fiorello,2004;Philip & Miller,2002). As will be seen, the DLPFC participates in focal attention and the formation of conscious experiences, including emotions.

The DLPFC has been shown to consist of a number of different regions, each region having different inputs and projecting to different target areas (Groenewegen et al,1990;Kolb,1990;Pandya & Yeterian,1990). Each region appears to be quite important in the mediation of a specific type of voluntary intentional action (action produced by choice for some purpose-Sieb,1995,2004,2007, 2011). For example, a region in area 9 receives input from the trunk and limb representations of somatosensory cortex and from association cortex involved in peripheral vision and projects to the supplementary motor cortex, premotor cortex, rostral motor cortex, basal ganglia, and cerebellum (Brooks,1986;Pandya & Yeterian,1990;Sieb,1987,1987,1989,1995). The target areas of this region are involved in the preparation and execution of voluntary intentional skeletomotor movements (Brooks,1986;Sieb, 1987,1987,1989,1995). The supplementary motor cortex is involved in the preparation of voluntary intentional skeletomotor movements and the programming of voluntary intentional motor sequences (Brooks,1986;Hyland et al,1989;Mann et al,1988;Orgogozo & Larsen,1979;Roland et al,1980;Tanji et al, 1988;Wiesendanger et al,1985). The supplementary motor cortex has been shown to be activated by movement intent, even if no movement occurs (Brooks,1986;Roland et al,1980). The supplementary motor cortex drives the execution of voluntary intentional skeletomotor movements via projection to the premotor cortex, rostral motor cortex, basal ganglia, and cerebellum (Afifi & Bergman,1986;Brooks,1986;Porter & Lemon,

1993;Primrose & Strick,1985;Sieb,1987,1987,1989,1995). Similarly, other regions in the DLPFC have been identified (Joseph,1990;Pandya & Yeterian,1990;Sieb,1995) which appear to be involved in the production of other types of voluntary intentional actions (area 44 or Broca's area-speech; part of areas 8 and 9 of the frontal eye fields-scanning eye movements; the border region of areas 6, 8, and 46-Exner's writing area; area 10-memory, thought; areas 10 and 11-emotion, social behavior). The output of DLPFC regions therefore appears to be directed to cortical and subcortical areas involved in the preparation and execution of various types of voluntary intentional actions (skeletomotor movements, oculomotor movements, speech, writing, reading, emotion, social behavior, memory, and thought). Voluntary intentional actions are prepared and directed by focal attention (working memory) and consciousness (Fuster,1997,2008;Sieb,2004,2007,2011). The DLPFC utilizes working memory in the mediation of its motor functions (Fuster,1997,2008;Goldman-Rakic,1990). In the absence of attention and consciousness, no such actions occur (sleep, coma, or anesthesia). The DLPFC therefore appears to be driven by attention and consciousness to produce various voluntary intentional actions. Attention and consciousness also appear to be utilized by the DLPFC to mediate intellectual (cognitive) functions (executive memory, language comprehension and production, grammar, abstract thinking, planning, problem-solving, decision-making, logic, reasoning, calculating).

Damage to the DLPFC produces the dysexecutive syndrome (Duffy & Campbell,2001;John,2009;Licter & Cummings,2001). This syndrome is characterized by problems with attention, affect, social judgment, executive memory, abstract thinking, intentionality, and motivation: difficulty altering set in response to changing contingencies, impaired strategy generation for solving complex problems, memory retrieval deficit, impaired verbal fluency, poor abstraction, concrete and perseverative thinking, impaired reasoning and mental flexibility, and reduced mental control. Such patients are inattentive and easily distractible, may need constant redirection, and may exhibit disorganized behavior.

ORBITOFRONTAL CORTEX (OFC)

The OFC consists of the prefrontal cortex immediately surrounding the orbit of the eye. The OFC consists of areas 10, 11, and 47 (Kringelbach,2005). The OFC is primary for the production of emotion, social behavior, and reinforcement-expectancies for decision-making (Blair,2008;Rushworth et al, 2007;Schoenbaum & Roesch,2005;Volz et al,2008). The OFC is

considered the central executive for emotion and social control (John,2009;Stuss & Levine,2002). Stimulus-reinforcement association (affective valence) input from the amygdala is essential for these functions of the OFC (Blair,2008;Lanteaume et al,2007;Murray et al,2009;Rushworth et al,2007;Schoenbaum & Roesch,2005). The OFC is interconnected with the higher cortical sensory association areas (areas 5,7,18,19,20,21,22), DLPFC, anterior cingulate cortex, superior temporal cortex, inferior parietal cortex, parahippocampal gyrus, hippocampus, thalamus, parts of the basal ganglia, hypothalamus, and amygdala (Alexander et al,1986;Blair,2008;Dolan et al,1999;Fuster,1997; John,2009;Johnson et al,1968;Jones & Powell,1970;Joseph,1989, 1990,2000;Pandya & Kuypers,1969;Rauch et al,2006;Rushworth et al,2007;Van Hoesen et al,1972). The OFC has been found to utilize affective valence to influence emotional reactions (Kringelbach & Rolls,2004;Phillips et al,2003a,b;Sauerland et al,1967;Siegel & Wang,1974;Steriade,1964;Volz et al,2008). Kringelbach and Rolls (2004) found that activity in the medial OFC was associated with the monitoring, learning, and memory of the reward value of reinforcers, while activity in the lateral OFC was associated with the evaluation of punishers. They found that more complex reinforcers (like monetary gain or loss) are represented more anterior in the OFC than less complex reinforcers (taste). Volz and coworkers (2008) also found the OFC involved in evaluation of reinforcement (affective valence) and in emotion. Volz found that the OFC continuously extracts affect-laden recognition patterns or cues (stimulus-reinforcement associations, affective valence) from sensory input, associates these with prior experiences with general categories (perception), and is responsible for a subjective sense of perceptual coherence of environmental information (the formation of emotion). Kringelbach (2005) found that the OFC mediates subjective hedonic experience (a conscious experience of a positive emotion) from reward (a positive affective valence). Hence the OFC appears to mediate the production of emotion from affective valence. The OFC is connected with the inferior parietal cortex. As will be seen, the OFC participates in focal attention and the formation of emotion from perception via its interaction with the inferior parietal cortex. The ability to shift attention (Butter et al,1970;Joseph,1990,2000; Kolb et al,1974;Luria,1980) and the ability to discriminate between relevant and irrelevant stimuli (Bechara et al,1994; Fuster,1990b,1997;Oscar-Berman,1975;Rolls et al,1994) are reduced with OFC damage. Complete destruction of the OFC may produce the complete loss of emotion and social behavior (Kling & Steklis,1976;Myers et al,1973). Severe damage greatly reduces emotional/motivational function, including motor activity

(Butter et al,1970). Less extensive damage produces loss of emotional control, leading to such emotional and social behavioral dysfunctions as disinhibition, hyperactivity, hypersexuality, euphoria, extroversion, lability, verbosity, perseveration (Bechara et al,1994;Rolls et al,1994), excessive swearing, irresponsibility, antisocial behavior, poor social interaction, poor empathizing ability, impulsivity, compulsive gambling, drug use (including alcohol and tobacco), inappropriate laughter, aggression, or a demanding manner (Benson & Geschwind,1971;Blumer & Benson,1975;Butter et al,1970; Kolb et al,1974;Lishman,1973;Luria,1980;Stuss & Benson,1984). Damage to the OFC may produce what is called the disinhibition or pseudopsychopathic syndrome (Duffy & Campbell,2001;Licter & Cummings,2001;John,2009). This syndrome is characterized by disinhibition, diminished self-supervision of behavior, distractibility, impulsiveness, tactlessness and loss of interpersonal sensitivity/empathy, utilization and imitation behavior, inappropriate jocularity, sexual preoccupation and jesting, hypomanic symptoms, neglect of personal care, poor hygiene, decreased social judgment, antisocial acts, and limited insight. Such a syndrome appeared in Phineas Gage, after a 3.5 foot metal rod was blown vertically through his brain passing just behind his left orbit (KSPS TV Production,How Smart Can We Get?*Nova Science Now,*2012). Right OFC damage seems to cause the most severe disruption of mood, emotion, and social behavior (Grafman et al,1986).

The OFC projects to the inhibitory medullary reticular formation and the excitatory pontine reticular formation (Fuster,1997; Sauerland et al,1967;Siegel et al,1977). The OFC may influence physiological and behavioral reactions via this connection. Damage to the OFC releases the autonomic nervous system from inhibitory control, leading to widespread disturbances of autonomic function (Joseph,1990;Kaada,1951,1972). The OFC is connected to the DLPFC. The OFC was found to be involved in planning behavior associated with sensitivity to reward and punishment (Bechara et al,1994). The OFC is interconnected with the medial magnocellular dorsomedial thalamic nuclei (Fuster, 1997;Sauerland et al,1967;Siegel et al,1977). The dorsomedial thalamic nuclei gate input to the neocortex and limbic system (Joseph,1990,2000;Skinner & Yingling,1977;Yingling & Skinner, 1977). The dorsomedial thalamic nuclei are reciprocally and topographically connected with the DLPFC and make up its defining projection (Fuster,1997;Joseph,1990,2000;Kolb,1990; Uylings & Van Eden,1990). Hence the OFC could influence DLPFC function via this connection as well.

ANTERIOR CINGULATE CORTEX (ACC)

The ACC (area 25) is the frontal part of the cingulate cortex and is a distinct cytoarchitectural and functional region of the prefrontal cortex (John,2009;Petrides & Pandya,1999;Preuss & Goldman-Rakic,1991). The DLPFC and supplementary motor cortex may have evolved from the ACC (Sanides,1972) and these areas are massively interconnected (Damasio & Van Hoesen,1980). Electrical stimulation of the ACC produces fragments of movements (Goldberg,1985) and vocalizations (Robinson,1967). The ACC is also connected with the lateral amygdala, septal nuclei, hippocampus, anterior hypothalamus, striatum, dorsomedial thalamic nuclei, inferior parietal cortex, and OFC (Baleydiere & Maguiere,1980;Pandya & Kuypers,1969;Powell,1978; Powell et al,1974). Electrical stimulation of the ACC also produces anxiety, fear, and pleasure (Meyer et al,1973). As will be seen, the ACC appears to be involved in focal attention and the production of emotion via its connections with the inferior parietal cortex. Regulation of autonomic function, detection of errors, monitoring of conflict, expectation of reward or loss (reinforcement-expectancies, anticipation of outcomes), decision-making, attention, motivation, and regulation of emotion are some of the functions that have been attributed to the ACC (Bush et al,2000;Decety & Jackson,2004; Drevets et al,2008;Jackson et al,2006;John,2009;Lictor & Cummings,2001;Nieuwenhuis et al,2001;Posner & Digirolamo,1998; Stuss et al,1986;Weissman et al,2005). The key functions of the ACC appear to revolve around the detection of errors or shortfalls from some standard, anticipation and preparation for action, and regulation of emotion. Lesions of the ACC result in inability to detect errors, severe difficulty resolving conflict in Stroop tasks, emotional instability, inattention, and akinetic mutism (Bush et al,2000;Posner & Digirolamo,1998). Lesions of the ACC may result in the apathetic or pseudodepressive syndrome (Duffy & Campbell,2001;John,2009; Licter & Cummings,2001). This syndrome is characterized by apathy (a loss of motivation or drive): emotional apathy-absence of interest, excitement, and intensity of emotional responsiveness to positive or negative events; affective apathy-flat, unchanging emotional expression; cognitive apathy-decreased generative thinking, curiosity, engagement with usual activities, interest in learning and new experiences, and lack of concern with one’s health, family, or future; motor apathy-lack of effort, productivity, ability to sustain activities, and initiation of new activities, increased dependence on others to structure activities, difficulty in initiating motor acts, hesitation when starting a new movement, and tendency to not

gesture when speaking. Akinetic mutism may present with bilateral ACC damage.

REGULATION OF EMOTION

The dorsal (perigenual) ACC and the ventral (subgenual) ACC have different connections, are part of different circuits, and have different functions. In fact, the ACC has been separated into a ventral emotional component and a dorsal cognitive component (Bush et al,2000;Bush et al,2002). Both components may be involved in attention and the regulation of emotion.

VENTRAL ACC

Neuroimaging, neuropathological, and lesion analysis data indicate an extended neural network formed by the connections of the ventral ACC with the OFC, amygdala, hippocampus, superior and medial temporal gyri, ventral striatum, mid and posterior cingulate cortex, thalamus, hypothalamus, periaqueductal gray, and habenula (Ongur et al,2003). This network has been found to be involved in the regulation of the evaluative, expressive, and experiential aspects of emotion (Alexander et al,1986;Blair, 2008;Bruno et al,2004;Dolan et al,1999;Rauch et al,2006). Abnormal interactions between the ventral ACC and other areas of the network may contribute to disturbances in emotional processing and regulation (Ongur et al,1998). In emotional disorders (depression, panic attacks, phobias, bipolar disorder, posttraumatic stress disorder) increased activation occurs in the ventral ACC and the amygdala (Ressler & Mayberg,2007). This increased activation decreased with resolution of the disorder through treatment (medications, behavioral therapy, electroshock therapy, magnetic stimulation techniques, or deep stimulation techniques). Impaired function within the network appears to give rise to the clinical signs and symptoms of depression or mania (Drevets et al,2008). The ventral ACC was found to undergo a substantial reduction in gray matter volume (mostly due to reduction in glia cells) in the mood disorders of major depression and bipolar (Drevets et al,1997;Drevets et al,2008; Ongur et al,1998). A reduction in gray matter volume was also detected in the brain areas connected to the ventral ACC, indicating that these areas are interconnected and part of a common circuit (Drevets et al,1997;Drevets et al,2008;Ongur et al,1998). This reduction in gray matter volume was accompanied by an increase in ventral ACC activity, which was decreased (the activity) by administration of antidepressants or by remission (Drevets et al,2008). Lithium (a mood stabilizer) has a neurotrophic effect and increased the gray matter volume when

used in the successful treatment of bipolar disorder (Drevets et al,2008). A reduction in gray matter volume in the ACC was also found in obsessive-compulsive disorder (Davidson et al,2003). In mood disorders, ventral ACC activity correlates positively with the severity of depressive symptoms (Osuch et al,2000). Humans with lesions which include the ventral ACC demonstrate abnormal autonomic responses to emotional experiences (understimulation of parasympathetic tone may occur in mood disorders), inability to experience emotion related to concepts that normally evoke emotion, and inability to use information regarding the likelihood of punishment versus reward (reinforcement-expectancies) in guiding social behavior (Bechara et al,1994;Damasio,1995;Drevets et al,2008). Patients with lesions to the ACC have diminished autonomic reactivity and reduced variations of affect to stressors (such as shocks), without affecting the capacity to control cognitive processes (Critchley et al,2003). The ACC appears to be involved in the experience of sadness and anxiety (Barrett,2006;Mauss & Robinson,2009;Phan et al,2002). Studies suggest that the ventral ACC functions in the automatic regulation of emotion (Elliott et al,2000;George et al,1995;Gillath et al,2005;Mayberg et al,1999;Phelps et al,2004;Rauch & Drevets,2008). The ventral ACC receives emotional input (affective valence, stimulus-reinforcement association information) from the amygdala (Budhani et al,2007;Pezawas et al,2005). The ventral ACC may be involved in evaluating the salience of rewards, as it modulates electrophysiological responses of ventral tegmental area dopamine neurons (Drevets et al,1998). The phasic burst firing of these neurons and the accompanying rise in dopamine release normally occurs in response to primary rewards and reward-predicting stimuli (Schultz,1997). In depression, reduced ventral ACC activity is associated with diminished stimulation of mesolimbic dopamine release, resulting in the absence of behavioral incentive, apathy, and anhedonia (Drevets et al, 1998). In mania, increased ventral ACC activity results in excessive stimulation of mesolimbic dopamine release, producing exaggerated hedonia response and elevated motivational drive (Drevets et al,1998). The ventral ACC is critical for the representation of stimulus-reinforcement association (affective valence) information that can be used by other structures for the regulation of emotion, reinforcement-expectancies for decision-making, and behavior (Blair,2008;Rushworth et al,2007; Schoenbaum & Roesch,2005). The precise role played by the ventral ACC and the OFC in reinforcement-expectancies for decision-making is as yet not understood (Rushworth et al,2007).

DORSAL ACC

The dorsal (perigenual) ACC is connected to the prefrontal cortex (DLPFC, OFC, other parts of the ACC), parietal cortex, hippocampus, motor system, and frontal eye fields (Allman et al, 2006;Damasio & Van Hoesen,1980;Posner & Digirolamo,1998). Summarizing the evidence gained from electrical studies, the dorsal ACC appears to receive information about a stimulus, selects an appropriate response, monitors the response, and adapts behavior if there is a violation of expectancy or error (Bush et al,2000;Gehring et al,1993;Holyroyd et al,2004;Luu & Pederson,2004). The rostral ACC (part of the perigenual ACC) was found to be active after error commission (a response function), while the dorsal ACC was found to respond to error and feedback of the error-an evaluative function (Bush et al, 2002;Carter et al,1998;Polli et al,2005). The evaluation function of the dorsal ACC was emotional in nature and indicative of the amount of distress associated with a certain error (Bush et al,2000).

The most basic form of dorsal ACC theory states that the dorsal ACC is involved in error detection (Bush et al,2000). A typical task which activates the dorsal ACC involves eliciting some sort of conflict in an individual that has the potential to result in error (Eriksen flanker task-Botvinik et al,1999;Stroop task-Pardo et al,1990). The dorsal ACC has been found to do more than merely detect error. It was found to be active during correct responses as well (Carter et al,1998). In addition, the more competitive are the stimuli, the greater the activation of the dorsal ACC (Carter et al,1998). Luu and Pederson (2004) and Polli and coworkers (2005) found that the dorsal ACC was active when subjects were not aware of their errors and that awareness of errors increases dorsal ACC activation and results in the production of fewer errors. Brown and Braver (2005) and Bush and coworkers (2002) found the dorsal ACC involved in the evaluation of the magnitude or cost of errors or shortfalls. Damage to the dorsal ACC causes difficulty in evaluating the consequences of social behavior and difficulty in evaluating the outcome of the treatment of mood disorders (Drevets et al,2008). Thus the dorsal ACC appears to detect error, evaluate the degree of error, and selects an appropriate form of action to be implemented by the motor system (Bush et al,2002;Polli et al, 2005;Taylor et al,2006).

Rather than representing errors or shortfalls, the dorsal ACC might be most active when conflicts between competing tendencies arise (Botvinik et al,1999;Carter et al,1998). Trials that produced the most conflict also produced the greatest activation

of the dorsal ACC (Botvinik et al,1999). Patients with ADHD were found to have damage to the dorsal ACC and difficulty evaluating conflict during Stroop tasks (Bush et al,1999). Patients with schizophrenia also were found to have damage to the dorsal ACC and difficulty dealing with conflict in Stroop-like tasks (Holyroyd et al,2004). However, it has been found that lesions to the dorsal ACC do not eliminate the processing of errors and conflicts (Baird et al,2006;Nachev et al,2006) and many executive functions, including the monitoring of errors and conflicts, remain intact after large dorsal ACC lesions (review by Critchley et al,2005). Dorsal ACC activity therefore appears not to be involved in error or conflict processing per se, but is correlated with error or conflict processing occurring somewhere else.

Many of the functions of the dorsal ACC appear to be correlated with awareness (consciousness). As described above, the response of the dorsal ACC to error or conflict is correlated with awareness (the evaluation of error or conflict by the dorsal ACC is therefore correlated with awareness). Lane and coworkers (1998) showed that ACC activity reflected improved recognition of emotional cues and targets in subjects with better emotional awareness. The dorsal ACC seems to be especially active when effort is required for the performance of a task, such as early learning and problem-solving (Allman et al,2001). Allman and coworkers (2001) and Critchley and coworkers (2003) found that the dorsal ACC was most active during effortful context-dependent tasks. Effort is defined as conscious exertion (Merriam-Webster). The ACC is larger on one side of the brain than the other. Morton and Rafto (2010) found that agency or will (a type of conscious experience, awareness) reside in the hemisphere with the larger ACC. ACC activity remains elevated in tasks which involve adherence to sequential decision-making paths (Stroop tasks-Pardo et al,1990). Awareness is central to the performance of these tasks. Rehearsing a task which originally produced spontaneous, novel responses (which requires awareness) to the point of producing stereotyped rigid responses (which are automatic, not requiring awareness) resulted in diminished ACC activation (Raichle et al, 1994). The dorsal ACC might regulate emotion using the same mechanism as it uses for the evaluation of errors (Compton et al,2008). The dorsal ACC might detect and evaluate undesirable emotion or shortfalls from some optimal emotion.

Both the dorsal ACC and the DLPFC have been shown to be involved in emotion regulation (Bouregard et al,2001;Kalisch et al,2005; Ochsner et al,2004). Proficient performance on tasks which

purportedly utilize the dorsal ACC and DLPFC (like the adjustment of performance after errors) tends to correlate with emotional well-being, such as diminished anxiety (Compton et al, 2008;Robinson,2007). Remember, the DLPFC is the central executive for cognitive function. Reward-based learning theory suggests that the dorsal ACC is involved in reward-based decision-making and learning, while the rostral ACC is involved in the emotional responses that arise (Bush et al,2002;Polli et al,2005;Taylor et al,2006;Volz et al,2008). The dorsal ACC might bias response selection with expectations of reward or loss, ie. with reinforcement-expectancies (Holyroyd et al,2004). The DLPFC might be more involved in the implementation of adjustments (Garavan et al,2002). Mayberg and coworkers (2005) found that electrical stimulation of the dorsal ACC curbed depression in some patients. They concluded that the dorsal ACC is a conduit between the cognitive functions of the prefrontal cortex (DLPFC) and the emotional experiences of the medial temporal limbic system and that this system was overactive in depression. The supragenual (dorsal) ACC was found to suppress the amygdala (Etkin et al,2006;Pezawas et al,2005) and hence change emotion generation. The dorsal ACC has been considered an interface between cognition and emotion (Bush et al,2000; Mayberg et al,2005;Volz et al,2008). Hence the dorsal ACC may be involved in what might be called the cognitive regulation of emotion.

LIE-DETECTION

Traditional lie detection depends on various technologies that measure physiological (blood pressure, galvanic skin response, heart rate) or behavioral (facial micro-expressions, eye tracking movements, pupil size, body language such as gestures or postures, and various linguistic indicators like voice analysis, word composition, and word patterns) reactions that accompany deception (Pjister,2011;Preston,2002;Rothwell,2006). Many of these reactions are under autonomic control and are more or less involuntary and very difficult to control consciously. It is thought that the physiological and behavioral reactions that occur during deception are part of an emotional reaction arising from putting the lie together. Utilizing fMRI (Ganguli, 2007;Langleben,2005), it was found that regions in the prefrontal cortex (specifically the superomedial and inferolateral) and parietal cortex are more active during deception than when telling the truth. These are just the areas (dorsal ACC, DLPFC/OFC, and inferior parietal cortex) that one would expect to be active during the cognitive regulation of emotion.

MOOD AND EMOTIONAL DISORDERS

There are therefore two regulatory systems involved in the regulation of emotion: the ventral system and the dorsal system. There are distinct patterns of structural and functional abnormality in the two systems which may be responsible for the specific symptoms of various mood and emotional disorders (John, 2009;Ongur et al,1998;Phillip et al,2003b;Ressler & Mayberg, 2007). The affective valence systems, amygdala, DLPFC, OFC, ACC, and IPC appear to be key structures involved.

POSITIVE FEEDBACK AND NONLINEAR EMERGENCE

Positive feedback and nonlinear emergence is a basic physical mechanism responsible for a vast number of natural phenomena-flames, waves, groups, hurricanes, businesses, organisms, individuals, etc. (Scott,1996,1999,2000,personal communication; Sieb,2004,2007,2011). Positive feedback is excitatory feedback (Scott,1996,1999,2000;Sieb,2004,2007,2011). It occurs when the output of a system feeds back as an additional excitatory input into the system. A positive feedback cycle arises when the output of a system feeds back as an additional excitatory input into the system, which produces more output, producing more positive feedback, producing more output, and so on. If unopposed, such a cycle results in an uncontrolled exponential increase in activation of the system (Scott,1996,1999,2000, personal communication;Sieb,2004,2007,2011) and the system would soon exhaust itself (like seizures). However, this uncontrolled increase in activity is normally counteracted by inhibitory influences which act against the excitatory positive feedback to produce a balanced equilibrium state (otherwise, the system would be suppressed or exhausted). The inhibitory influences are said to be nonlinear because they are variable and have variable effects, which are not entirely predictable (Gershenson & Heylighner,2004). Because the balanced state emerges in a nonlinear manner, it is known as a nonlinear emergent state. Nonlinear emergent states are always new or novel. They have new properties not present in the original external inputs from which they were developed (Newman,1997;Scott,1996,1999,2000, personal communication). Nonlinear emergent states cannot be precisely predicted in advance from the original inputs from which they were developed as linearly-produced states can be (Gershenson & Heylighner,2004). Nonlinear emergent states are fixed for a period of time and are said to be explicit (ie. they are fully realized physically and are considered discreet

physical objects which can be directly utilized-O'Brien & Opie, 1999). Nonlinear emergent states therefore can affect other physical systems and processes. Nonlinear emergent states developed from positive feedback have the following properties (Sieb,2011): they are system-generated, form rapidly, but do take time to form, persist for a period of time, are explicit, can affect other physical systems and processes, are complex, bounded, have content, have limited capacity, are variable, flexible, seamless, ineffable, transparent, serial in production, prone to interference, unified and coherent, and projected outwards. All these properties have also been ascribed to conscious experience, including emotion (Baars,1988; Koch,1998;Tononi & Edelman,1998). This suggests that conscious experiences, including emotions, may arise from positive feedback and nonlinear emergence.

EXAMPLES OF POSITIVE FEEDBACK AND NONLINEAR EMERGENCE

There are many examples of phenomena arising from positive feedback and nonlinear emergence (Scott,1996,2000,personal communication;Sieb,2004,2007,2011). One familiar example is a candle flame. When a candle is lit (input), candle wax melts, producing wax vapor, which burns, dissipating heat and light (output). Some of the dissipated heat feeds back (positive feedback) to melt more candle wax, producing more wax vapor, which burns, producing more heat, some of which feeds back, and so on, producing a positive feedback cycle. Nonlinear inhibitory influences (cross-sectional area of the candle, loss of heat, air movement, air temperature, humidity, etc.) act to inhibit the excitatory influences (melting of candle wax, formation of wax vapor, burning of wax vapor, dissipation and feedback of heat). The candle flame is a balanced equilibrium state that arises between the inhibitory and excitatory influences (a balanced dissipation of heat and light). The candle flame therefore is a nonlinear emergent state and has all the properties mentioned in the preceding section. As everyone knows, a candle flame is explicit (physical) and can affect other physical systems (one's finger, inflammable objects). The candle flame has properties not present in or predictable from the original external inputs from which it was developed (candle wax, wax vapor, match). Waves (water, optical, sound) also may be explained through nonlinear emergence. When a rock is thrown into a pond (input), water molecules are displaced outwards (output). Displaced water molecules push against other adjacent water molecules, some of which rush in to fill the space previously occupied by the displaced water molecules (positive feedback). This excitatory input further displaces water

molecules outwards, which feeds back, producing further displacement, leading to a positive feedback cycle. This positive feedback cycle is counteracted by nonlinear inhibitory influences (weight of water molecules, water temperature, depth, currents, obstacles, etc.) producing a wave (a balanced displacement of water molecules propagating outwards). A wave is certainly explicit with new properties not present in or entirely predictable from the original inputs (rock, water molecules). Any business may be described utilizing nonlinear emergence. Money is utilized to set up an inventory or services (input). Sale of the inventory or services results in the acquisition of more money (output). Some of this money (positive feedback) is used to provide more inventory or services, and so on, resulting in a positive feedback cycle. Nonlinear inhibitory influences (number of employees, number of customers, amounts of money, size and type of the inventory or services, hours of operation, etc.) act to balance the disposition of money resulting in the nonlinear emergence of a specific business (a functioning Walmart store, Imperial Oil company, or Macdonald's restaurant, etc.). Nerve impulses, groups of people (committees, nations, families, etc.), stock markets, and many other phenomena may be described as arising from positive feedback and nonlinear emergence. Conscious experiences (including emotions) may also arise from positive feedback and nonlinear emergence (Sieb,2004,2007,2011).

PERCEPTION

Conscious experiences (including emotions) appear to arise from perception (Mattingley,1999;Rowlands,2002;Velmans,1999). Perception is the interpretation of sensory stimulation in terms of previous experience (Merriam-Webster). Perception appears to occur in the inferior parietal cortex-IPC (Mattingley,1999). The IPC is at the junction of the parietal, temporal, and occipital lobes of the brain and consists of the supramarginal (area 40) and angular (area 39) gyri, part of area 7, and is partly coextensive with the posterior superior temporal gyrus-area 22 (Joseph,1990,2000). The IPC contains neurons which are multimodally responsive, receiving highly processed input from somesthetic, visual, and auditory association cortex, the frontal lobes, and other higher-order assimilation areas throughout the neocortex (Bruce et al,1986;Burton & Jones,1976; Jones & Powell,1970;Joseph,1990,2000;Seltzer & Pandya,1978;Zeki, 1974). Note that the three regions of the prefrontal cortex (DLPFC, OFC, and ACC) connect to the superior temporal gyrus and the IPC (Bogarts,1997;John,2009;Petrides & Pandya,2007). All higher-order sensory processing converges on the IPC. IPC

neurons can simultaneously analyze visual, auditory, and somesthetic information, respond to visual stimuli of any size, shape, or form, and have visual receptive properties that span almost the entire visual field (Bruce et al,1982,1986;Hyvarinen & Shelepin,1979;Joseph,1990,2000). The IPC is involved in the creation and assimilation of cross modal associations (auditory, visual, and somesthetic equivalents of objects, events, ideas, actions, feelings-Joseph,1990,2000). This is perception. Damage to the left IPC may cause anomia, object and finger agnosia, acalculia, most forms of apraxia, agraphia, left-right disorientation, pure word blindness, alexia, conductive aphasia, disruption of sequential-grammatical relationships, disruption of spatial-sequential ability, impairment of visual-spatial functioning, impairment of temporal-sequencing ability, disruption of the performance of skilled temporal-sequential motor acts, and the impairment of logic and grammar (Benson et al,1973;Heilman,1973,1979;Hechaen & Albert,1978;Joseph,1990, 2000;Luria,1980;Sauguet et al,1971;Straub & Geschwind,1983; Vignolo,1983). Dressing apraxia and constructional apraxia may occur with right IPC damage (Hier et al,1983;Mehta et al,1987; Joseph,1988a,1990,2000). Note that impaired formation of conscious experiences could result in all these disruptions. It is interesting to note that the parietal cortex of Albert Einstein's brain was 15% greater in size than in any other human brain on file (KSPS TV Program,How Smart Can We Get?,Nova Science Now,2012). Einstein was noted for his extraordinary perceptual and cognitive skill.

NEGLECT

Neglect syndrome may occur with damage to the IPC (Joseph,1990, 2000;Mattingley,1999;Prinz,Lecture at *Towards A Science Of Consciousness (TASOC)* 2012 Conference). Loss of dreaming (a type of conscious experience) can occur with parietal cortex damage (Solms,M.,In *"What Are Dreams?"*,KSPS Nova TV Program, 2009). Neglect (hemineglect, unilateral neglect, spatial neglect, hemispatial neglect) syndrome is a deficit in awareness (conscious experience) of one side of space that occurs as a result of brain damage (Husain,2008;Unsworth,2007). Neglect is not due to lack of sensation or ability to move, but appears to be caused by an inability to pay proper attention to sensory stimuli (Husain,2008;Unsworth,2007). Even though the person with neglect is unaware of sensory stimuli, appreciable processing of the stimuli still appears to occur, even to the point of identity and meaning (Berti & Rizzolatti,1992;Rees, 2001;Rees et al,2000). The neglect may extend to the visual, auditory, somatosensory, and olfactory domains (Husain,2008;

Unsworth,2007). Neglect is most profound and long-lasting with damage to the right cerebral hemisphere with the awareness deficit to left (contralateral) space (Husain,2008;Unsworth, 2007). Left hemisphere damage may produce a less prolonged and long-lasting neglect of right space (Husain,2008;Kim et al, 1999). Neglect has classically been associated with lesions of the right posterior parietal cortex, particularly in the right temporoparietal junction or inferior parietal lobe (Husain, 2008;Mattingley,1999;Vallar & Perani,1986), in other words, in the right IPC. Isolated lesions of the right frontal lobe have also been associated with neglect of left space (Husain & Kennard,1996). Severe left unilateral inattention and neglect is seen with massive lesions to the right frontal lobe of the brain (Joseph,1988a,1988b,1990,2000). Subcortical lesions (such as of the basal ganglia and thalamus), without involvement of the overlying cortex, can lead to neglect (Hillis et al,2002; Hillis et al,2005). Damage to white matter pathways connecting posterior and frontal cortical areas has also been implicated in neglect (Bartolomeo et al,2007;Thiebaut de Schotten et al,2005). Patients with neglect have different combinations of posterior cortical, frontal cortical, subcortical, and white matter damage and this is thought to underlie the diversity of functional deficits found in such people (Husain,2008). Thus the neglect syndrome suggests that the connection between the IPC and frontal cortex is quite important in attention and the production of conscious experience, including emotion.

POSITIVE FEEDBACK AND NONLINEAR EMERGENCE OF CONSCIOUS EXPERIENCES (INCLUDING EMOTIONS)

Crick and Koch (2003) determined that conscious experience depends on neuronal coalitions spanning the posterior sensory cortices and the executive areas of the prefrontal cortex. Hudetz (Anesthesia:When the front fails to see the back,Talk at *TASOC* 2008 Conference) and Hudetz and coworkers (2006) found that anesthetic drugs produce unconsciousness by disrupting these long range anterior-posterior connections. Laplant and coworkers (2005) demonstrated a reduction in frontal activation in anesthetized animals that was normally present in waking animals. This "hypofrontality" is commonly produced by a number of anesthetics. Laureys (Identifying the brain's awareness system:Lessons from coma and related states,Lecture at *TASOC* 2012 Conference;Demertzi et al,2013;Lapitskaya et al,2013) found that external sensory awareness is an emergent property dependent on cortical-cortical interconnections in a widespread frontal-parietal network. Activity in this network was reduced during anesthesia, coma, unresponsive wakefulness syndrome

(vegetative state), and minimally conscious states. Imas and coworkers (2005) found recurrent feedback from anterior to posterior brain areas was suppressed earlier in anesthetized animals than feed forward information transfer. Mashour (Consciousness in the operating room,Lecture at *TASOC* 2012 Conference;Avidan et al,2011;Ku et al,2011;Lee et al,2009) found that anesthetics suppress mainly parietal cortex feedback connectivity, but not parietal cortex feed forward connectivity. This is reversed when the person awakes. Mashour concludes that the parietal cortex is affected much more than frontal areas by anesthetics, that conscious experience is likely a cortical-cortical feedback interaction, and that conscious experience may be governed by this feedback interaction. A number of other investigations have found that feedback connectivity is required to generate conscious experience (Boehler et al,2008;Crick & Koch,1995;Fahrenfort et al,2007;Lamme & Roelfsema,2000;Ro et al, 2003;Tononi & Koch,2008). A positive feedback interaction between the prefrontal cortex and IPC appears to be involved in the production of conscious experience, including emotion.

POSITIVE FEEDBACK, NONLINEAR EMERGENCE, AND ATTENTION

Perception and focal attention are necessary for the production of conscious experience, including emotion (Rowlands,2002; Velmans,1999). As discussed, the IPC is involved in perception. The frontal lobes, especially the right frontal lobe, and the parietal lobes have great attention capacity (Desmedt,1977; Dimond,1976,1979;Hielman & Van Den Abell,1979,1980;Joseph,1982, 1986a,1988a,b;Tucker,1981). Prinz (2003;Attention as the mechanism of consciousness,Lecture at *TASOC* 2012 Conference) found that awareness (conscious experience) does not arise without attention and develops over a time delay. Prinz suggested this time delay may be the time necessary for attention to act. Joseph (2000) and Prinz found that attention, perception, and working memory occur in the IPC. Working memory also operates in the DLPFC (Fuster,1997,2000,2008;Goldman-Rakic, 1990). Working memory is the provisional retention of perceptual information for prospective action, a type of focal attention whereby perception is reorganized and rerepresented, becoming explicit, functional, and conscious (Luck & Vogel,1997; Todd & Marois,2004;Vogel & Machizawa,2004). Working memory is a cognitive process that is accompanied by consciousness (Fuster, personal communication,2013). Working memory represents the two components (perception, focal attention) necessary for the production of conscious experience and produces conscious experience. Working memory may be formed and maintained by reentry (Fuster,1997;2008). Reentry is a reciprocal interaction

and occurs throughout the brain (Tonelli & Edelman,1998). Consciousness is impaired if the reentry mechanisms of the brain are disrupted (Tonelli & Edelman,1998). Positive feedback is a type of reentry. Positive feedback and nonlinear emergence in prefrontal-IPC connections also occurs over a time delay and results in conscious experience. Positive feedback and nonlinear emergence in prefrontal-IPC connections then may be a type of working memory (focal attention) whereby perception is reorganized and rerepresented, becoming explicit, functional, and conscious. The reorganization and rerepresentation of perception occurring in working memory is not entirely predictable and probably arises in a nonlinear fashion typical for complex systems like the brain (Gershenson & Heylighen, 2004). Positive feedback and nonlinear emergence in prefrontal-IPC connections therefore may be a type of focal attention which produces conscious experience, including emotion. Conscious experience (emotion) is in effect arising from focal attention and perception. Any disruption in the positive feedback interaction between the prefrontal cortex and IPC would be tantamount to a disorder of attention and might produce neglect.

PRODUCTION OF ADAPTIVE ACTION

Working memory has been shown to operate in the DLPFC (Fuster, 1997,2000,2008;Goldman-Rakic,1990). Fuster (1997,2000,2008) found that the DLPFC mediates the ongoing production of voluntary new or novel intentional (adaptive) actions through the integration of three cognitive processes: active short-term memory (working memory), motor set (selection, preparation, and readiness for action), and the inhibition of excess activity (also part of attention). Hence the explicit conscious states (conscious experiences, emotions) arising from positive feedback and nonlinear emergence (working memory, focal attention) may be utilized by the DLPFC for the preparation of voluntary new or novel intentional (adaptive) actions (and in other cognitive functions). Such actions are thus mediated, deliberated, voluntary, and intentional. Emotions are subjective; that is, emotions are developed by processes within the brain and are not directly caused by external sensory stimuli. Subjective states like conscious experiences (including emotions) may be thought of as new internal points of view or perspective (internal contexts) built from previous experience (perception) and external input (Alter,2002;McClamrock,1992;McCrone,2001;Sieb, 2011). Adaptive actions (actions performed in response to changes in the environment, also called voluntary new or novel intentional actions) are always produced in relation to our point of view or perspective (subjective conscious state) at the

time (Alter,2002;McClamrock,1992;McCrone,2001;Sieb,2011). Perception would directly produce only stereotyped, reflexive, or programmed-type actions (Gershension & Heylighen,2004). In order to produce voluntary new or novel intentional (adaptive) actions, perception must be reorganized and rerepresented (Gershension & Heylighen,2004). As just discussed, this may occur through positive feedback and nonlinear emergence in prefrontal-IPC long-range connections (working memory, focal attention). The creation of explicit functional conscious states (emotions, other conscious experiences) allows us to respond to our constantly changing environment in a voluntary, rapid, new or novel, versatile, and purposeful manner; in other words, we respond to change by self-organizing our behavior (McCrone,2001;Sieb,2011). We are not bound by stereotyped, inflexible, reflexive, or programmed-type responses, typical of animals and machines, but can create new or novel responses, as the situation demands (Sieb,2004,2007,2011). Various degree, composition, and type of emotion may be created, depending on the situation. Hence we have a high capacity to produce adaptive behavior based on our emotions. Emotion may be especially important in the production of the adaptive behavior known as social behavior. The ability to create a variety of emotions is necessary for successful social interaction and for competition and survival in our complex societies.

CONCLUSIONS

Emotion is explicit, functional, and conscious. It is the affective aspect of consciousness. Emotion has all the same properties as conscious experience and is defined as conscious experience. Emotion therefore is conscious experience. Emotion is also produced in the same way as conscious experience. Hence emotion is conscious per se. Conscious experience, including emotion, represents the content of consciousness (what is in consciousness). Emotion may have evolved for the production of adaptive behavior, especially social behavior.

Emotions are complex reaction patterns arising to sensory stimulation and consist of three components: a physiological component, a behavioral component, and an experiential (conscious) component. Emotions arise from processes within the brain and are not directly caused by external sensory stimuli (hence they are subjective). The reactions making up an emotion determine what the emotion will be recognized as.

Three processes are involved in producing emotion: (1) identification of the emotional significance of a sensory

stimulus, (2) production of an affective state (emotion), and (3) regulation of the affective state. Process (1) consists of the development of an affective value or valence (stimulus-reinforcement association) for sensory stimulation. Affective valence is developed through the relative activation of two opposing systems in the brain by sensory stimulation: the reward and punishment systems. The development of affective valence begins early in sensory processing, long before the sensory stimulus is recognized cognitively and consciously in the cerebral cortex. The development of affective valence is important as it regulates further stimulus processing (facilitating or inhibiting stimulus processing), regulates arousal, attention, and consciousness (through activation of the reticular activating system), and is the basis for the evaluative, expressive, and experiential aspects of emotion, conditioned emotion learning acquisition and expression (in the amygdala), memory consolidation (via the amygdala and hippocampus), reinforcement-expectancies for decision-making and coping responses (via the amygdala, OFC, and ACC), adaptive behavior (via the dorsal ACC and DLPFC), and social behavior (via the dorsal ACC, OFC, and DLPFC). The effects of affective valence are relatively primitive or reflexive at subcortical levels but may be refined into emotional reactions at higher limbic and neocortical levels. The reward system appears to produce its effects through the modulation of dopamine, acetycholine, noradrenaline, and serotonin neurotransmitter actions in the brain. The punishment system appears to produce its effects via inhibitory limbic structures. All emotions have an affective valence (are seen as positive or negative emotions) and appear to develop from affective valence.

The amygdala is critical for the representation of stimulus-reinforcement (affective valence) associations (both reward and punishment-based) needed for emotion processing and expression (process 2), positive and negative conditioned emotion learning acquisition and expression, memory consolidation, reinforcement-expectancies for decision-making and coping responses, and social behavior. Stimulus-reinforcement associations are passed from the amygdala to the ventromedial prefrontal cortex (OFC, ventral ACC) for emotion expression and regulation, reinforcement-expectancy for decision making, and social behavior. Increased amygdala activity due to emotional arousal increases the strength and retention of long-term memories via connection of the amygdala with the hippocampus and parahippocampus. Amygdala projection to the ventral ACC is utilized for the automatic regulation of emotion. The dorsal ACC suppresses the amygdala and may be involved in the cognitive

regulation of emotion. Differences in the size and connections of the amygdalae may account for some of the differences in emotion and social behavior seen in men and women. People with larger amygdalae are better able to interpret emotions, have larger and more complex social networks, are better able to make social judgments, and may have better emotional intelligence than people with smaller amygdalae.

The prefrontal cortex appears to be essential for the regulation of emotion (process 3). The prefrontal cortex may be separated into three distinct and separate architectural and functional areas. The DLPFC is known as the central executive for cognitive and intentional control. It is the highest cortex for motor planning, preparation, and execution (voluntary new or novel intentional actions). Explicit functional conscious states (including emotions) may participate in the preparation of cognitive functions and voluntary intentional (adaptive) actions (including social behavior and coping responses) by acting on the DLPFC. The OFC is known as the central executive for emotion and social control. The OFC is primary for the production of emotion, social behavior, and reinforcement-expectancies for decision-making. Affective valence information from the amygdala is essential for this function. The ventral ACC may assess affective valence information from the amygdala for the automatic regulation of emotion and works with the OFC in the implementation of reinforcement-expectancies for decision-making. The activity of the dorsal ACC is correlated with consciousness and appears to be involved in the cognitive regulation of emotion and motivation.

A positive feedback interaction between the prefrontal cortex (DLPFC, OFC, and ACC) and the IPC (perception) appears to give rise to the conscious experience of emotion. Positive feedback and nonlinear emergence in prefrontal-IPC connections may represent a type of working memory or focal attention by which perception is reorganized and rerepresented, becoming explicit, functional, and conscious. Explicit functional conscious states (including emotions) arising from this mechanism may be utilized for the preparation of cognitive functions (planning, reasoning, calculating, problem-solving, decision-making, logic, grammar, language comprehension and production), voluntary new or novel intentional (adaptive) actions, social behavior, and motivation.

Emotion has an intimate relationship with social behavior. Disruption at any stage in the development of emotion (affective valence, regulation of attention and consciousness by the reticular activating system, amygdala, OFC, ACC, DLPFC, IPC,

prefrontal-IPC connections) results in disordered social behavior. Emotion appears to control social behavior (our emotions determine how we respond to others). Study of the various structures and interconnections involved in the production of emotion is very important for the understanding, diagnosis, and treatment of various mood and emotional disorders and social dysfunctions.

Essentially, the whole brain is involved in the production of emotion. Affective valence must be developed for sensory input via the reward and punishment systems. The reticular activating system regulates the level of attention and consciousness. Affective valence is represented in the amygdala. Affective valence information is passed from the amygdala to the prefrontal cortex. Sensory input is integrated in the sensory areas of the cerebral cortex and converges for perception in the IPC. A positive feedback interaction between the prefrontal cortex and the IPC results in the nonlinear emergence of an explicit functional emotion.

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