The Neural Basis of the Dynamic Unconscious

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A great deal of complex cognitive processing occurs at the unconscious level and affects how humans behave, think, and feel. Scientists are only now beginning to understand how this occurs on the neural level. Understanding the neural basis of consciousness requires an account of the neural mechanisms that underlie both conscious and unconscious thought, and their dynamic interaction. For example, how do *conscious* impulses, thoughts, or desires become *unconscious* (e.g., repression) or, conversely, how do *unconscious* impulses, desires, or motives become *conscious* (e.g., Freudian slips)? Research taking advantage of advances in technologies, like functional magnetic resonance imaging, has led to a revival and re-conceptualization of some of the key concepts of psychoanalytic theory, but steps toward understanding their neural basis have only just commenced. According to psychoanalytic theory, unconscious dynamic processes defensively remove anxiety-provoking thoughts and impulses from consciousness in response to one's conflicting attitudes. The processes that keep unwanted thoughts from entering consciousness include repression, suppression, and dissociation. In this literature review, studies from psychology and cognitive neuroscience in both healthy and patient populations that are beginning to elucidate the neural basis of these phenomena are discussed and organized within a conceptual framework. Further studies in this emerging field at the intersection of psychoanalytic theory and neuroscience are needed.

Keywords: unconscious; psychodynamic; repression; suppression; dissociation; neural

"Nothing is so difficult as *not* deceiving oneself."

Ludwig Wittgenstein [1889–1951]

Cognitive unconscious processing

The intricate relationship between conscious and unconscious processes is one of the many mysteries that continue to perplex our understanding of ourselves.[1] How much of our subjective conscious experience is influenced by unconscious processes? There is a distinction, however, between unconscious processes, which neuroscience is more likely to explore, and the unconscious mind with its psychoanalytic contents (Kihlstrom, 1994, 1999; Macmillan, 1996; Westen, 1998a). Early psychodynamic theorists attempted to explain phenomena observed in the clinic, but later cognitive scientists used computational models of the mind to explain empirical data. By using models based mostly on nonclinical data, cognitive science (in branches like neuroscience, cognitive psychology, neural modeling, and neural linguistics) departed from the older psychoanalytic theories, heading into new areas involving neural processes (Ekstrom, 2004). For example, recent imaging, psychophysical, and neuropsychological findings suggest that unconscious processes take place hundreds of milliseconds before conscious awareness.

It is largely accepted that lower levels of processing (e.g., motor reflexes, sensory analysis) can operate outside of perceptual awareness (implicitly) (e.g., Castiello, Paulignan, & Jeannerod, 1991). And although the existence of nonconscious computations at higher levels (e.g., semantic or inferential processing) has been controversial (Dixon, 1971; Eriksen, 1960; Greenwald, 1992; Holender, 1986), a range of empirical find-

[1] The terms consciousness and awareness (or conscious and aware) are used in this article synonymously and anything outside of awareness/consciousness is referred to as nonconscious (a term used more in cognitive psychology that emphasizes the descriptive and empirical nature of the phenomenon) or unconscious (traditionally used in the psychoanalytic tradition to reflect more dynamic unconscious processes).

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ings on the unconscious over the last several decades has led most cognitive neuroscientists today to believe that mental activity can occur outside of conscious awareness (Hassin, Uleman, & Bargh, 2005). Some have argued that all information processing can, at least in principle, operate without conscious experience, and that consciousness (C) may thus be of a different nature (Chalmers, 1996). This view goes along with the hypothesis that nonconscious processes can achieve the highest levels of representation (Marcel, 1983). A large amount of complex cognitive processing appears to occur at the unconscious level in both healthy and psychiatric and neurological populations. For example, evidence from patients with blindsight (Goebel, Muckli, Zanella, Singer, & Stoerig, 2001; Weiskrantz, 1986), prosopagnosia (Renault, Signoret, Debruille, Breton, & Bolgert, 1989), implicit awareness in hemineglect (Cappelletti & Cipolotti, 2006; Marshall & Halligan, 1988; Vuilleumier et al., 2002), nondeclarative learning even in amnesia (Knowlton, Mangels, & Squire, 1996; Knowlton, Squire & Gluck, 1994; Turnbull & Evans, 2006), and the "split-brain" syndrome (Gazzaniga, 1995) supports the idea that unconsciously processed stimuli can activate high-level cortical regions.

Subliminal perception

Kouider and Dehaene (2007) suggest that in order to reach C, a stimulus must have sufficient strength (which can be hindered by masking)[2] and receive top-down attention (which can be thwarted by drawing attention to another task or stimulus). Subliminal perception (aka perception without awareness) occurs when stimuli are processed by our sensory systems, but do not reach the "threshold" of entering into C because they are presented below the limen for conscious perception. This is usually demonstrated by presenting stimuli that are "masked" or presented in a subtle form or too briefly to be consciously perceived, but are sufficient to prime or bias a subject's performance in tasks like lexical decision-making. Subliminal perception studies have shown that unconscious processing can influence awareness. Subliminal priming can occur in a range of sensory modalities and with a range of different stimuli (visual, verbal, auditory etc.) and is inferred when a stimulus is not perceived, yet still influences actions, thoughts, feelings, learning, or memory.

Evidence shows that subliminal stimuli can still be highly processed and can even activate motor responses (e.g., Dehaene et al., 1998, 2001, 2004; Marcel, 1983; Naccache et al., 2005; Nakamura, Dehaene, Jobert, Le Bihan, & Kouider, 2005; Nakamura et al., 2007). Subliminal priming studies indicate that a masked word or digit can have an influence on perceptual, lexical, and semantic levels (Allport, 1977; Kouider & Dehaene, 2007; Marcel, 1974, 1980, 1983; Nisbett & Wilson, 1977). These studies suggest that the subliminal words activate cognitive processes associated with the meanings of words, even though there was no conscious awareness of such an effect. Semantic priming from masked stimuli has been shown not only with words (Balota, 1983; Fowler, Wolford, Slade, & Tassinary, 1981) but also with auditory stimuli (Holender, 1986; Nisbett & Wilson, 1977; Schacter, 1992) and pictures (Carr, McCauley, Sperber, & Parmelee, 1982; McCauley, Parmelee, Sperber, & Carr, 1980; Nisbett & Ross, 1980). Even associative learning, as measured by event-related brain potentials, can occur without awareness (Wong, Bernat, Bunce, & Shevrin, 1997). Thus, it seems as though some stimuli that are sensed by our sensory organs, but do not reach the "threshold" of conscious awareness, are still processed by our neural network and can influence higher level cognitive processing and behavior.

Neuroimaging studies show that subliminal priming evokes activation in several cortical areas (see Kouider & Dehaene, 2007). Compared to supraliminal stimuli, cortical activation to subliminal stimuli is often weaker, but there are many exceptions (e.g., attentional blink) that show that high activation is not a sufficient condition for conscious access (Kouider & Dehaene, 2007). Studies using intracranial recordings with electrodes in humans provide the first direct evidence that subliminal words perceived unconsciously can have long-lasting effects on neuronal signals and can trigger long-lasting cerebral processes (Gaillard et al., 2007; Naccache et al., 2005). Nakamura et al. (2006) show that the subliminal priming effects in lexical decision and pronunciation tasks can be selectively disrupted by transcranial magnetic stimulation (TMS) to distinct sites, suggesting that task set influences subliminal processing. And evidence from event-related potentials (ERPs) shows that goal-driven, task-set dependent attention can be captured by visual stimuli that are not consciously perceived (Ansorge, Kiss, & Eimer, 2009).

Based on studies that show that inhibition is present when stimuli are presented superluminally but not when

[2] When presenting a visual stimulus (the "mask") directly before and/or after another briefly presented (≤ 50 ms) visual stimulus (the "target") leads to a failure to consciously perceive the target stimulus (Breitmeyer & Ogmen, 2007).

presented subliminally (e.g., Allport, Tipper, & Chmiel, 1985; Marcel, 1980; McCormick, 1997; Merikle, Joordens, & Stolz, 1995; Neill, Valdes, & Terry, 1995; Tsushima, Sasaki, & Watanabe, 2006), some contend that inhibitory control is restricted to stimuli that are accessible to C. They assert that while subliminal stimuli can trigger passive activation, only supraliminal stimuli can elicit active inhibitory control. However, Eimer and Schlaghecken (2003) review studies that challenge this view. These experiments show that inhibitory processes can take place even when response predispositions are activated by subliminal stimuli. Results from subliminal priming experiments have shown that masked stimuli, which are not perceived consciously, can still trigger response activations, and that these response activations can subsequently be inhibited (Eimer, 1999; Eimer & Schlaghecken, 2002), perhaps to prevent behavior from being controlled by extraneous stimuli (Eimer, 1999). Early response facilitation produced by consciously perceived information may in fact counteract the automatic effects of self-inhibitory motor control (Eimer & Schlaghecken, 2002).

Schlaghecken, Münchau, Bloem, Rothwell, and Eimer (2003) found that repetitive TMS (rTMS) over left premotor or motor cortex during a masked prime task did not affect reaction times triggered by subliminal primes. So subliminal priming effects do not appear to be caused by activation of premotor or motor cortex. Subsequent data suggest that motor control in a masked prime task is influenced by low-level, automatic processes mediated by subcortical (presumably basal ganglia–thalamic) control circuits (Schlaghecken, Bowman, & Eimer, 2006). Thus, inhibitory motor control processes can be decomposed into separate mechanisms that operate at different levels within the motor response system (Schlaghecken et al., 2006). "*Endogenous*" inhibition, which occurs when stimuli are presented supraliminally, is voluntary, optional, dependent on the conscious detection of task-relevant signals, and thought to be controlled by executive mechanisms mediated by the prefrontal cortex (PFC) (Band & van Boxtel, 1999; Berlin, Rolls, & Iversen, 2005; Berlin, Rolls, & Kischka, 2004; Konishi, Nakajima, Uchida, Sekihara, & Miyashita, 1998; Liddle, Kiehl, & Smith, 2001; Menon, Adleman, White, Glover, & Reiss, 2001; Rubia et al., 2001; Tsushima, Sasaki, & Watanabe, 2006). In contrast, "*exogenous*" inhibition (i.e., inhibitory response control to subliminally presented stimuli) appears to be reflexive and nondependent on the conscious detection of task-relevant signals and is believed to be mediated by corticostriate circuits (involving subcortical structures like the thalamus and caudate nucleus, and perhaps posterior parietal cortex) (Eimer & Schlaghecken, 2003) and may not engage the PFC at all (Aron et al., 2003).

Affective and motivational unconscious processing

Despite the surge of empirical studies of unconsciousness and cognitive processes (e.g., see Greenwald, 1992; Hassin, Uleman, & Bargh, 2005; Kihlstrom, 1987; Schacter & Buckner, 1998), few cite current psychodynamic work or theories (Robins & Craik, 1994). The unconscious of cognitive scientists is automatic, cold, and cognitive, and many are skeptical of extending the notion of unconscious processes to affect and motivation and of the idea that affect can bias how thought is constructed outside of awareness ("defense"). Phenomena of the kind described above, where sophisticated *cognitive* processes can occur without subjective experience of them, support Sigmund Freud's [1856–1939] general claim of omnipresent unconscious mental activity (Turnbull, 2001). But they do not support the more specific facets of his model, described by psychodynamic theorists and clinicians for a century—for example, that unconscious *emotional* and *motivational* factors can mold the conscious mind (Turnbull & Solms, 2007). Attention to the affective and motivational aspects of the unconscious would give a more comprehensive, balanced, and valid depiction of the workings of the human mind (Westen, 1998a).

A vast amount of data supports the proposition that much of mental life, including thoughts, feelings, and motives, is unconscious (Westen, 1998b). Researchers are beginning to discover that the same principles that apply to cognition operate with unconscious (implicit) affective and motivational processes as well. So the *cognitive unconscious* (Kihlstrom, 1987, 1990) is now becoming the *cognitive–affective–motivational unconscious* (Brenner, 1982; Sandler, 1987; Westen, 1998a). Due in part to advances in functional imaging, we now have incomparable access to the neurobiological bases of instinctual drives and basic emotions (Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Etkin et al., 2004; Jackson et al., 2003; Sander, Roth, & Scheich, 2003; Yoshino, Kimura, Yoshida, Takahashi, & Nomura, 2005), and evidence for their importance in mental life (Damasio, 1994, 1999; LeDoux, 1998a; Panksepp, 1998; Rolls, 1995). Recent findings support Freud's claim that mental activity is rooted in phylogenetically old emotion and motivation systems that influence early mental development (LeDoux, 1998a; Panksepp, 1998; Pfaff, 1999).

Unconscious emotional processes

Studies on unconscious affect provide persuasive evidence that people can feel things without knowing they feel them and can act on feelings of which they are unaware (e.g., see Westen, 1998a, 1998b)—an idea that has guided psychoanalytic clinical practice for a century. The studies presented thus far on unconscious affect provide particularly compelling evidence for a central hypothesis that has been propounded only by psychoanalytic theory and has guided psychoanalytic clinical work for a century: People can feel things without knowing they feel them, and they can act on feelings of which they are unaware—for example, subtly hostile, indifferent, or defensive treatment of members of ethnic minority groups. A cognitive–affective neuroscience of the unconscious has recently emerged, focusing on laboratory paradigms like subliminal perception, implicit cognition, and directed forgetting and proving new insights into the neural basis of unconsciousness and cognition and affect (Stein, Solms, & van Honk, 2006). Evidence suggests that emotion processing is initiated and can proceed without conscious awareness (Balconi & Lucchiari, 2008; Bunce, Bernat, Wong, & Shevrin, 1999; LeDoux, 1998a; Phelps et al., 2000; Wiens, 2006; Wong et al., 1994,). This makes sense since emotional input is highly adaptive and thus preferentially processed with or without capacity-limited C. Behavioral and physiological measures reveal that unconscious stimulation is sensitive to the emotional content of the stimuli (Lang et al., 1998).

Craig's (2002, 2009) theory of the neural basis of interoceptive conscious perception ties emotions to body states. Consistent with the theories of William James (1890) and Antonio Damasio (1994), Craig (2002, 2009) suggests that subjective human emotion is based on an abstracted meta-representation of the physiological state of the body in the right anterior insular cortex, which provides the foundation for the volitional modulation of feelings, emotion, and efferent activity affecting the state of the body. So feelings may have their basis in body representations, but we do not have conscious access to the neuronal processes that underlie bodily homeostasis and emotion states (Craig, 2002, 2009).

Tsuchiya and Adolphs (2007) review the evidence for unconscious emotions. Emotional responses can occur without awareness of the stimuli that triggers them—for example, in studies of fear conditioning to subliminal stimuli (Wong, Bernat, Snodgrass, & Shevrin, 2004). "Invisible" visual stimuli can affect judgments of visible stimuli (Murphy, Monahan, & Zajonc, 1995; Murphy & Zajonc, 1993; Tamietto & de Gelder, 2008), and emotional visual stimuli can elicit affective somatic responses even when cortical processing of the stimuli is diminished by backward masking (Macknik & Livingstone, 1998). Evidence for the unconscious perception of masked faces in humans has been shown in studies using subjective reports (Esteves, Parra, Dimberg, & Öhman, 1994), autonomic reaction (Morris, Buchel, & Dolan, 2001a), ERPs (Kiefer & Spitzer, 2000), and brain imaging (Whalen et al., 1998). Subjects show increased skin-conductance responses to masked fear-conditioned visual stimuli (Esteves et al., 1994) and covert facial mimicry to masked fearful faces (Dimberg, Thunberg, & Elmehed, 2000). ERPs also show subliminal processing of fearful faces, providing further evidence of emotional processing without conscious awareness (Kiss & Eimer, 2008).

Brain lesion patients also provide evidence that nonconscious stimuli can, in fact, elicit emotion states. In a phenomenon known as "affective blindsight,"[3] patients with lesions in the primary visual cortex (V1) can have affective responses to emotional visual stimuli presented in their blind visual field, without early cortical processing (e.g., in V1) or conscious awareness (i.e., they deny consciously seeing anything in the blind field) of the stimuli. These responses include behavioral responses (e.g., above chance discrimination of gestures and emotional facial expressions in forced-choice paradigms) (de Gelder & Hadjikhani, 2006; de Gelder, Vroomen, Pourtois, & Weiskrantz, 1999; Pegna, Khateb, Lazeyras, & Seghier, 2005), judgments of visible stimuli presented simultaneously (de Gelder, Morris, & Dolan, 2005; de Gelder, Pourtois, van Raamsdonk, Vroomen, & Weiskrantz, 2001), and somatic responses (e.g., startle reflex potentiation) (Anders et al., 2004; Hamm et al., 2003). Some patients with V1 lesions can reliably discriminate the affective valence of facial expressions presented to their blind fields by guessing, or by using techniques like reaction times, despite having no conscious awareness of the stimuli (Anders et al., 2004; de Gelder, Haan, & Heywood, 2001; de Gelder, Pourtois, & Weiskrantz, 2002; de Gelder, Vroomen, Pourtois, & Weiskrantz, 2000; de

[3] Blindsight (Cowey & Stoerig, 1991; Weiskrantz, 1986) is a phenomenon where patients with primary visual cortex (V1) lesions, but intact retina and retino-tectal projections, maintain that they are blind, but have accurate (above chance) behavioral responses to visual tracking and other select visual tasks, which are thought to be mediated by extrageniculostriate retinofugal pathways (Cowey & Stoerig, 1991). Thus, they can perceive visual stimuli in some way even though they are not conscious of doing so. Similar phenomena have been observed in other sensory modalities, such as blindsmell (Schwartz, 1996; Schwartz et al., 1994) and blindtouch (Paillard, Michel, & Stelmach, 1983), where patients are not consciously aware of the stimuli due to lesions in or near the corresponding primary sensory cortex but have appropriate behavioral responses to them.

Gelder et al., 1999, 2001; Hamm et al., 2003; Pegna et al., 2005; Tamietto & de Gelder, 2008).

These "unconscious emotion" effects (e.g., affective somatic responses to visual stimuli presented in a V1 lesion patient's blind visual field) are thought to be mediated by a subcortical visual pathway that includes the superior colliculus, pulvinar thalamus, and amygdala (aka a subcortical retino–tecto–thalamic route to the amygdala) (e.g., Berman & Wurtz, 2010; Diamond & Hall, 1969; Lyon, Nassi, & Callaway, 2010). However, recent work by Schmid et al. (2010) shows that in the monkey, the thalamic lateral geniculate nucleus (LGN) is critical in the processing of visual information independent of V1 (i.e., blindsight), via direct LGN projections to extrastriate cortex (e.g.V2, V3, V4, and V5). In either case, there appears to be an "alternative" pathway that bypasses neocortical processing routes thought to be necessary for conscious detection, discrimination, and identification of stimuli (Andino, Menendez, Khateb, Landis, & Pegna, 2009; Johnson, 2005; Linke, De Lima, Schwegler, & Pape, 1999; Morris, Buchel, C., & Dolan, 2001; Morris, Friston, & Dolan, 1997; Morris, Ohman, & Dolan, 1999; Pegna et al., 2005; Rosen et al., 1992).

Emotional influences on conscious perception may be related to automatic activation of emotional circuits including, but not limited to, the amygdala. Animal studies suggest that fear-related responses occur via a direct subcortical pathway from the thalamus to the amygdala, allowing emotional (specifically threatening) stimuli to be processed automatically and outside awareness (LeDoux, 1998a). Imaging studies reveal that while implicit cognitive learning is mediated by regions including the striatum (Rauch et al., 1997), unconscious emotional responses are mediated by regions including somatosensory association areas (Anders et al., 2004) and the amygdala (de Gelder, Morris, & Dolan, 2005; Morris, Ohman, & Dolan, 1998; Stein, Solms, & van Honk, 2006; Vuilleumier et al., 2002; Whalen et al., 1998). For example, individual differences in trait anxiety predict basolateral amygdala response to unconsciously processed fearful faces (Etkin et al., 2004), and amygdala activation correlates with indirect/unconscious measures of race evaluation (Implicit Association Test and potentiated startle), but not with the direct/conscious expression of race attitudes (Phelps et al., 2000). Naccache et al. (2005), using intracranial electrodes, recorded brain potentials in three epileptic patients. Emotional words presented subliminally modulated amygdala activity at a long latency (>800 ms), implying that subliminal words can trigger long-lasting cerebral processes, like semantic access to emotional valence.

Threatening (fearful, angry) as well as nonthreatening (happy) emotional pictures and faces result in increased amygdala activity even when they are unattended (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Vuilleumier et al., 2001), presented briefly, masked from awareness (Morris, Ohman, & Dolan, 1998; Whalen et al., 1998), or suppressed during binocular rivalry (Pasley, Mayes, & Schultz, 2004; Williams, Morris, McGlone, Abbott, & Mattingley, 2004). In accordance with this, blindsight patients show modulation of amygdala activity in response to the emotional meaning of stimuli that they cannot see consciously (Andino et al., 2009; Morris, de Gelder, Weiskrantz, & Dolan, 2001; Penga et al., 2005). Other neuroimaging studies have found substantial activation in the amygdala (as well as the fusiform gyrus and superior temporal sulcus) and emotional responses to objectively invisible emotional stimuli (see Tsuchiya & Adolphs, 2007). For example, Jiang and He (2006) found that bilateral amygdala responses to fearful faces occurred independent of objective visibility, but the responses to neutral faces were modulated by visibility. The increased amygdala activity for suppressed affective faces, regardless of valence, may be driven by inputs via the rapid, phylogenetically older, subcortical pathway that assists in prompt detection of potential danger (Vuilleumier, Mohr, Valenza, Wetzel, & Landis, 2003; Williams et al., 2004). Back projections linking the amygdala to the visual cortex via the thalamus (Amaral & Price, 1984; Amaral, Price, Pitkanen, & Carmichael, 1992) may provide a route by which emotion can influence perceptual dominance of rivaling images during visual cortex processing (Alpers, Ruhleder, Walz, Mühlberger, & Pauli, 2005). This "low road" of visual processing may prime and modulate the visual cortex for preferential processing of emotional material (especially fearful) (Davis & Whalen, 2001; LeDoux, 1998b, 2000). However, although the amygdala is believed to process fear-related stimuli nonconsciously and rapidly, a woman (SM) with complete bilateral amygdala lesions,[4] who could not recognize fear from faces, still showed normal nonconscious processing and rapid detection of those same fearful faces (Tsuchiya, Moradi, Felsen, Yamazaki, & Adolphs, 2009). Thus, the authors suggest that the amygdala may not be essential for early stages of fear processing, but may modulate social judgment and recognition.

[4] Note that this was not a conventional lesion that took place suddenly. SM suffers from Urbach–Wiethe disease, a rare recessive genetic disorder that causes bilateral symmetrical calcification of the amygdala, which most likely took place very early in her life.

Visual stimuli presented to fully sighted people, and in the sighted visual field of blindsight patients, are thought to be processed via the subcortical/"alternative" pathway described above (i.e., the retino–tecto–thalamic route, or via direct LGN projections to extrastriate cortex (Schmid et al., 2010), and simultaneously by the retino–geniculo–cortical pathway directly to V1 involved with in-depth, conscious cortical visual processing. And some studies suggest that the level of this parallel cortical processing determines the degree to which information from subcortical processing modulates emotional responses and reaches awareness. For example, Jolij and Lamme (2005) induced affective blindsight in healthy people by applying TMS to their visual cortex. Interestingly, subjects could report the valence of the affective face only when TMS interfered with cortical processing. Access to the affective content of the stimuli disappeared after prolonged task training or when the stimulus visibility increased. Thus, it seems that conscious processing of information can actually *repress* unconsciously processed information, lending credence to the idea that conscious processes can repress unconscious tendencies.

In line with this, using functional magnetic resonance imaging (fMRI) in 9 cortically blind patients, Anders et al. (2009) found that despite similar startle reflex potentiation in their blind and sighted visual fields in response to a threatening visual stimulus, patients reported significantly *less* negative affect when the stimulus was presented to their sighted visual field. In other words, when the affective visual stimulus was visible and received full cortical processing, the patients' conscious phenomenal experience of affect was reduced and did not reflect their unconscious somatic response. The results also implied that this "decoupling" of somatic responses and consciously experienced affect might occur via left ventrolateral PFC activity inhibiting affect-related somatosensory cortex, resulting in the reduction of negative phenomenal experience when the negative stimulus is consciously seen. However, this "repression" mechanism may be bypassed when the stimulus is not consciously seen, and in such cases the subjective negative affective experiences may counterintuitively be enhanced. Thus, the left PFC appears to play a role in the passive control of negative affect. In accordance with this, Tsushima, Sasaki, and Watanabe (2006) using fMRI and a very well controlled paradigm found that supraliminal inhibition is mediated by dorsolateral PFC activity.

It is also interesting to note that studies using pharmacological administration together with cognitive–affective paradigms or fMRI (Harmer, Hill, Taylor, Cowen, & Goodwin, 2003; Harmer, Shelley, Cowen, & Goodwin, 2004) have suggested that monoamine neurotransmitters and steroid hormones (Hermans, Putman, Baas, Koppeschaar, & van Honk, 2006; van Honk, Peper, & Schutter, 2005) play a key role in mediating implicit cognitive–affective processes as well (Stein, Solms, & van Honk, 2006).

In sum, studies in both healthy and brain lesion subjects have demonstrated that, under certain circumstances, stimuli that are not experienced consciously still can modulate neural activity and generate emotional responses. Further evidence demonstrates that subliminally presented stimuli, if sufficiently weak, can lead to autonomic responses, without the subject experiencing the emotional responses themselves—that is, when subjects are completely unaware of their own emotional reaction (Dimberg, Thunberg, & Elmehed, 2000; Tsuchiya & Adolphs, 2007). For example, two studies show that emotional states that are not experienced consciously at all can still motivate behavior (Adolphs, Tranel, Koenigs, & Damasio, 2005; Winkielman, Berridge, & Wilbarger, 2005). Winkielman, Berridge, and Wilbarger (2005) found that subliminally presented (masked) happy or angry faces, for which participants reported no subjective change in affect, could still influence their subsequent drinking behavior. Subjects placed more value on beverages (via pleasantness ratings and willingness to pay) and consumed more of the beverage after subliminally presented happy faces, while their beverage value and consumption decreased after subliminally presented angry faces. So nonconscious stimuli can influence motivation, value judgment, and goal-directed behavior without affecting conscious feeling. Further support comes from a bilateral insula lesion patient who could not perceive taste (Adolphs et al., 2005). He described solutions of lime juice, saline, and sugar as all tasting "like pop" and drank them arbitrarily. But he preferred the sugar solution when given a choice between solutions presented simultaneously, showing a motivational preference based on the affective value of the taste, without an emotional response to, or conscious experience of, the tastes. These studies demonstrate that the affective value of stimuli that are not consciously perceived and do not produce any conscious affective feelings can still motivate behavior.

Unconscious motivational processes and decision-making

Motives, like skills, may be activated unconsciously. Some claim that the majority of the motives that drive our behavior occur outside of awareness (e.g., Bargh

& Chartrand, 1999; Solms, 1997), so a person may be unable to report the goals or rewards that underlie their behavior (Bargh, 1997). A recent review paper by Custers and Aarts (2010) summarizes studies that demonstrate how the pursuit of goals can operate outside of conscious awareness, a phenomenon they call "unconscious will." Studies show that under certain circumstances, actions can be initiated without conscious awareness of the goals to be attained or their motivating effect on one's behavior. However, we still do not understand exactly how unconscious goals control behavior at the neural level, and as such, this should be explored in future research.

There are many examples that show that people are often not aware of the countless different things that affect their decisions about what they do and say (Bargh & Ferguson, 2000; Ferguson & Bargh, 2004; Hassin, Ferguson, Shidlovski, & Gross, 2007). Nisbett and Wilson (1977) review evidence suggesting that people have little or no direct introspective access, and have only *inferential* access, to their higher order cognitive processes and causal links of their mental states. Studies show that when people act on the basis of motives or preferences for which they cannot access reasons, they tend to make up sensible, often incorrect, explanations about their behaviors after the fact, based on intuitive theories about themselves and psychological causality.

Unconscious motivation in humans is often inferred but is rarely demonstrated empirically. However, Bargh (1997; Bargh & Barndollar, 1996) produced research showing the existence of unconscious motivational processes. Extending findings on automatization of cognitive processes (Anderson, 1995) to motives, Bargh claims that well-learned goals can be activated by environmental stimuli, and related behaviors can occur without conscious awareness. Disagreeing with the idea of a simple, irrational unconscious (e.g., Greenwald, 1992), he thinks an individual's history of learning in a given situation is embodied in automatic and habitual motives, which are often better guides to behavior than a conscious, seemingly rational analysis of a single event, which may be ignorant of base rates and previous automatic actions (Westen, 1998a). Accordingly, "gut" feelings are often better guides to action and produce more postdecisional satisfaction than reasoned thoughts, which may interfere with emotion-based judgments (Damasio, 1996; Wilson & Schooler, 1991; Wilson et al., 1993). Furthermore, ventromedial PFC lesion patients, whose reasoning processes are mostly intact, cannot use prior affective associative learning to adaptively guide their behavior, and consequently they make poor decisions (Bechara, Tranel, Damasio, & Damasio, 1996).

According to the "theory of unconscious thought" (Dijksterhuis & Nordgren, 2006; see also Dijksterhuis, Bos, Nordgren, & van Baaren, 2006), conscious thought, due to its precision (it can strictly follow self-generated rules), may lead to good choices in simple matters, but to worse choices in more complex matters because of its limited capacity. Unconscious thought ("deliberation without attention") can conform to rules but has a higher capacity, but due to its relative imprecision it may lead to lower quality choices. However, quality of choice does not deteriorate with increased complexity, so unconscious thought may lead to better choices under complex conditions, since large amounts of information can be integrated into the evaluation. Dijksterhuis et al. (2006) confirmed in four studies on consumer choice, in the laboratory and in actual shoppers, that purchases of complex products were viewed more favorably when decisions were made without attentive deliberation, while choices about simple products produced better and more favorable results after conscious deliberation. This suggests that complex cognitive processes like decision-making occur at the unconscious level and that it may be better to think consciously about simple matters, and unconsciously about complex ones.

However, in contrast to the predictions made by the "unconscious thought theory" (i.e., that complex decisions are best made following an interval of distraction presumed to generate "unconscious thought"), the findings of Waroquier, Marchiori, Klein, and Cleeremans (2009, 2010) suggest that decisions made after distraction are better because conscious deliberation/rumination can deteriorate decisions that have already been made on first impressions that were formed "online" during information acquisition. But conscious deliberation can improve decisions when a high-quality first impression is not available, because conscious thinking can help improve performance when alternatives have not been properly compared and a decision has not yet been made. In sum, they suggest that rather than "thinking unconsciously" about a decision, "if you have a clear first impression, stick with it; if not, keep thinking" (Waroquier et al., 2010). Waroquier et al. (2010) do not, however, assert that decisions are always best when made consciously, or that decision-making involves only conscious processes, but simply that certain types of information processing, in particular those that involve symbol manipulation and propositional reasoning, can only take place in conscious thought.

Still, substantial evidence from recent studies suggests that conscious thought does not always lead to the best choices and that, in accordance with Benjamin

Libet's classic studies (Libet, 1985; Libet, Gleason, Wright, & Pearl, 1983) and Wegner's (2003) theory that "conscious will is an illusion," simple decisions can be predicted by brain activation well before a person becomes consciously aware of his or her intent to take a certain course of action. For example, using probabilistic population codes for Bayesian decision-making, Beck et al.'s (2008) evidence suggests that our "unconscious" brain makes the best decisions; similarly, using fMRI, Soon, Brass, Heinze, and Haynes (2008) found that unconscious brain activity in prefrontal and parietal cortices predicts decisions made by as much as 7 seconds before the subject is consciously aware of his or her decision (although some argue that these studies do not adequately manipulate C (e.g., by masking) or test for explicit conscious awareness (C. Koch, personal communication). Furthermore, by recording electroencephalography (EEG) signals while participants solved verbal puzzles, Sheth, Sandkühler, and Bhattacharya (2009) found that unconscious brain activity (posterior beta and anterior gamma oscillations) predicts the moment of cognitive insight. Finally, Zhong, Dijksterhuis, and Galinsky (2008) showed that distractions facilitate creative problem-solving, demonstrating the importance of unconscious thought in creativity, and Zhaoping and Guyader. (2007) found that people performed feature-detection tasks better when they simply "trusted their instincts."

The term "defense" describes processes whereby people adjust their cognitions to avoid aversive feelings like guilt and anxiety (Freud, 1933). Emotion systems (and their governing drives) may distort cognitive representations of reality by seizing executive resources via "defenses." Freud argued that humans are often irrational, holding false beliefs, because their consequences are subjectively advantageous (Turnbull & Solms, 2007). Emotion-biased, or motivated, reasoning, biased to produce emotionally preferable conclusions, is a form of implicit affect regulation where the brain comes to solutions that simultaneously satisfy cognitive constraints that maximize goodness of fit to the data and emotional constraints that maximize positive and minimize negative affect states associated with threat to or attainment of motives (Thagard, 2003; Westen, 1994, 1998a; Westen & Blagov, 2007). Research has begun to examine explicit (conscious) processes used to regulate emotion (e.g., suppression and distraction; Anderson et al., 2004; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Ochsner, Bunge, Gross, & Gabrieli, 2002), but studies examining the neural basis of motivated reasoning or implicit affect regulation are rare.

In the first study to describe the neural correlates of motivated reasoning (and related concepts of psychological defense, implicit affect regulation, confirmatory biases, and cognitive dissonance; Westen, 1994), during the 2004 U.S. Presidential election, Westen, Blagov, Harenski, Kilts, and Hamann (2006) gave 30 committed partisans reasoning tasks involving judgments about information threatening to their own candidate, the opposing candidate, or neutral targets. Motivated reasoning, measured during fMRI, was associated with activation of ventromedial PFC, lateral orbitofrontal cortex, anterior and posterior cingulate, and insular cortices, and not with activation in regions (dorsolateral PFC) previously linked to conscious/explicit emotion regulation (e.g., suppression) and "unemotional" reasoning. Thus, motivated reasoning appears to be qualitatively different from reasoning when there is no strong emotional investment in the outcome. But the extent to which motivated reasoning engages neural circuits involved in "unemotional" reasoning and conscious emotion regulation is unknown.

Pessiglione (2007) imaged unconscious motivational processes in a paradigm where the tighter subjects squeezed a handgrip when an image of money was presented, the more of it they could win. The presentation duration, and thus reportability, of the images varied from 17 and 50 ms, which were determined to be subliminal from a preliminary behavioral test, to 100 ms, which was consistently associated with conscious perception. Subjects squeezed harder when larger sums of money appeared, regardless of whether they were consciously perceived or not. The ventral pallidum (of the basal ganglia) was activated whenever participants responded, and it may be part of a circuit underlying both unconscious and conscious motivation, enabling expected rewards to invigorate behavior. The results suggest a "bottom-up" decision-making process, where the ventral pallidum is part of a circuit that first weighs the reward and decides, and then interacts with the higher level, conscious regions, like the PFC, if at all. In line with this, experiments by Libet and colleagues (Libet, 1985; Libet et al., 1983,) suggest that cerebral activity (readiness potentials) precede the conscious intent to perform a motor act by as much as 500 ms, implicating unconscious processes in decision-making. It appears as if our self-sufficient brains can evaluate a situation and select adaptive action before they (i.e., we) are aware of it or of the initial input, if at all (Kinsbourne, 1998). Thus, although decisions probably involve a complicated mix of unconscious and conscious processes, evidence suggests that they are largely predetermined and biased by unconscious processes, perhaps much more than we would like to believe.

Brain lesion patients with disorders of awareness such as anosognosia (apparent unawareness of their disorder) provide further support for "cognition beyond conscious awareness" and a unique window into the nature of self-deception (Trivers, 2000). Evidence suggests that patients with anosognosia (in particular for hemiplegia) have "implicit" awareness of their deficit, and that their lack of explicit awareness is driven by the emotionally aversive consequences of bringing deficit-related thoughts into C—that is, they appear to be engaged in a "defensive" emotion-based denial of their deficit (Bisiach & Geminiani, 1991; Fotopoulou, Pernigo, Maeda, Rudd, & Kopelman, 2010; Fotopoulou, Rudd, Holmes, & Kopelman, 2009; Fotopoulou et al., 2008; Kaplan-Solms & Solms, 2000; Nardone, Ward, Fotopoulou, & Turnbull, 2007; Ramachandran, 1996a; Turnbull, Jones, & Reed-Screen, 2002; Turnbull, Owen, & Evans, 2005; Vuilleumier, 2004). It has been suggested that anosognosia might result from a lesion of a right-lateralized emotion-regulation system, such that these patients are less able to tolerate aversive stimuli (Kaplan-Solms & Solms, 2000; Nardone et al., 2007; Turnbull, Jones, & Reed-Screen, 2002; Turnbull, Owen, & Evans, 2005). In line with this and with Craig's (2002, 2009) theory (described above), findings from Fotopoulou et al. (2010) suggest that the delusional features of anosognosia for hemiplegia can be explained by a failure to "re-represent" sensorimotor information in the right insular cortex (and possibly limbic areas and basal ganglia circuits), which is thought to be required for explicit, affectively personalized sensorimotor awareness.

Using an attentional-capture paradigm with hemiplegia-deficit-related words, Nardone et al. (2007) found that non-anosognosics showed reduced latencies (i.e., facilitation) for emotionally threatening words, while anosognosics (most with hemiplegia) showed increased latencies (i.e., interference). This indicates some degree of "implicit" knowledge of their deficit, which may be kept outside of C by a process akin to repression, in that they seem to be avoiding thoughts related to their deficits (i.e., despite explicit indifference to their motor impairment, anosognosic patients show interference for disability-related words). Along similar lines, nonlesion individuals classified as repressors show slowed responses to threatening objects, while highly anxious participants show speeded-up responses to the same objects (Calvo & Eysenck, 2000).

Interestingly, anosognosic patients can temporarily acquire conscious awareness of their disability subsequent to certain psychological manipulations (Kaplan-Solms & Solms, 2000; Ramachandran, 1995; Weinstein & Kahn, 1953), such as interventions that change the affective consequences of their motor disability, manipulate a first- versus third-person perspective (Fotopolou et al., 2009; Marcel, Tegner, & Nimmo-Smith, 2004), or offer a nonaversive explanation for their paralysis (Ramachandran, 1996b). These occasional episodes of transient awareness, when knowledge of their deficit reaches C, often cause the patient a great deal of distress and negative affect such as sadness (Kaplan-Solms & Solms, 2000; Moss & Turnbull, 1996; Turnbull, Jones, & Reed-Screen, 2002; Turnbull, Owen, & Evans, 2005). These findings exemplify the importance of motivation and emotion in the generation and maintenance of self-deception.

The neural basis of unconscious dynamic processes

There has been recent interest in scientific data relevant to analytic theory (Bilder & LeFever, 1998; Solms & Turnbull, 2002; Westen, 1999) and in the reformulation of its concepts using advances in cognitive science (Erdelyi, 1985; Horowitz, 1988; Kihlstrom, 1987; Stein, 1992, 1997; Stein, Solms, & van Honk, 2006; Turnbull & Solms, 2007). Psychodynamic theories emphasize unconscious dynamic processes, which are mental processes and contents that are defensively removed from C as a result of conflicting attitudes. Empirical studies in healthy and patient populations are beginning to elucidate the neural basis of the classical psychodynamic concepts of repression, suppression, and dissociation.

Repression

Freud (1892–93) proposed that much of human behavior is influenced by unconscious processes, and that the unconscious contains socially unacceptable ideas, motives, desires, and memories associated with conflict, anxiety, and emotional pain, which are put out of mind, so as to not be easily retrieved, to protect the person from distress. Defense mechanisms are unconscious mental strategies used to protect the mind from conflict and distress. One such mechanism proposed by Freud (1915) is repression—the *unconscious* process of pulling thoughts into the unconscious, to keep unwanted, anxiety-provoking, painful memories, thoughts, desires, and impulses from entering C. But these "forgotten" thoughts, memories, and urges can still influence conscious thoughts and feelings and express themselves as symptoms. Freud believed that mental illness

arises when these unconscious forces, wishes, and motives, which influence behavior, are in conflict.

Research suggests a link between physical illness and people with repressive personality style (usually measured by questionnaires and/or psychological tests), who tend to avoid feeling emotions to manage distress and defensively renounce their affects, particularly anger (Jensen, 1987; Schwartz, 1990; Weinberger, 1992, 1995). The inhibition of conscious access to emotions puts the body, especially the heart and immune system, under significant stress (Westen, 1998a). These memories and emotions do not just disappear; they continue to influence behavior (e.g., a person with repressed memories of childhood abuse may later have difficulty forming relationships). Repression may express itself through symptoms (e.g., a repressed sexual desire may resurface as a nervous cough or slip of the tongue; Freud, 1895). So, the body can articulate unconscious desires via symptoms that one cannot verbalize. This information can also leak into C via a Freudian slip (accidentally revealing a hidden motive), free association, or dreams.

The majority of studies show that while people who repress report healthy coping and adaptation, objective physiological or cognitive measures indicate that they are hypersensitive to anxiety-provoking information, especially when it is personally relevant (Furnham, Petrides, Sisterson, & Baluch, 2003). One study found that while heterosexual men exhibited increases in penile circumference to heterosexual and female homosexual videos, only the homophobic men showed an increase to male homosexual stimuli (Adams, Wright, & Lohr, 1996). Homophobia was associated with homosexual arousal that the homophobic individual was unaware of or denied. Homophobia may thus be a response to a threat to an individual's own homosexual impulses causing repression, denial, or reaction formation to such impulses (West, 1977).

The neural mechanisms underlying repression are unknown. People with a repressive personality style were found to have smaller evoked potentials to subliminal stimuli and gave significantly fewer verbal associations to the stimuli (Shevrin, 1973; Shevrin, Smith, & Fritzler, 1969, 1970). Repressiveness was also related to the presence of unconscious conflict reflected in differential brain responses to subliminal- and supraliminal-conflict-related words (Shevrin, Bond, Brakel, Hertel, & Williams, 1996). There is some evidence that subliminal conflicts are resolved without a significant contribution from the anterior cingulate cortex, which instead participates, along with the PFC, in a distributed network for conscious self-regulation (Dehaene et al., 2003).

Although some have technical objections to his account (e.g., see Koch, 2004), Libet (Libet, 1966, 1973, 1978; Libet et al., 1964) found that a critical time period for neural activation is needed for a stimulus to become conscious. During neurosurgical treatment for dyskinesias, primary somatosensory cortex (S1) was stimulated with an electrode and elicited a sensation in a portion of the contralateral hand, wrist, or forearm. A train of repetitive 0.5-ms pulses of liminal intensity had to persist for about 500 ms to elicit a sensation. This was known as the minimum "utilization train duration" (UTD). UTD values varied little over time within subjects, but they varied between subjects from 200–750 ms. Subjects with a longer UTD exhibited a greater tendency to repression, as measured by a battery of psychological tests (Shevrin, Ghannam, & Libet, 2002). So, people who need a longer time period of neural activation in order to develop a conscious experience of a stimulus may be prone to develop repression as a defense against unacceptable unconscious wishes (for instance, people with high intelligence may be prone to develop intellectualization as a defense). This suggests that this neurophysiological time factor is necessary, but not sufficient, for the development of repression and that it may be possible to explore the neurophysiological processes involved in repression itself.

Using a very clever paradigm and technique called "continuous flash suppression" (Tsuchiya & Koch, 2005; Tsuchiya, Koch, Gilroy, & Blake, 2006), Jiang, Costello, Fang, Huang, and He (2006) demonstrated that interocularly suppressed ("invisible") images of naked men and women, which do not enter the subjects' C, can attract or repel subjects' spatial attention based on their gender and sexual orientation. Despite being unaware of the suppressed images, heterosexual males' attention was attracted to invisible female nudes, heterosexual females' and homosexual males' attention was attracted to invisible male nudes, and homosexual/bisexual females performed in-between heterosexual males and females. What was particularly interesting was that *heterosexual* males were actually *repelled* by pictures of naked men in that their attention was diverted away from areas of their visual field where invisible naked men were presented. None of the other groups showed this repulsion effect. This appears to be an example of the Freudian concept of repression—that is, the unconscious prevention of anxiety-provoking thoughts or desires (in this case, perhaps latent homosexual desires in heterosexual men) from entering C. Another controversial implication of this experiment is that it suggests that an individuals' sexual orientation can be statistically inferred from their unconscious at-

tentional biases (Koch, 2008). Although these results are only behavioral and do not uncover the neural pathways that enable such unconscious attentional modulation, the authors suggest that because the stimuli were arousing erotic images, the amygdala is likely to play a critical role.

Despite the evidence described above, the existence of repression remains contentious, due in part to its association with trauma and to the practical and ethical problems of studying it in controlled animal and human experiments. Therefore, creative paradigms with which to study the mechanism underlying repression in the laboratory are needed.

Suppression

Suppression—the voluntary form of repression proposed by Freud (1892–93)[5]—is the *conscious* process of pushing unwanted information (thoughts, emotions) out of awareness, and it is thus more amenable to controlled experiments than repression. While some claim that memory repression or suppression is a clinical myth with no scientific support (Kihlstrom, 2002), others have provided initial evidence for memory suppression (Anderson & Green, 2001; Anderson et al., 2004). Memory suppression requires people to override or stop the retrieval process of an unwanted memory, and this impairs its later retention (Anderson & Green, 2001). Executive control processes can be recruited to prevent unwanted declarative memories (provoked by cues) from entering awareness, and this cognitive operation makes later recall of the rejected memory harder (Anderson & Green, 2001). If suppression by executive control processes becomes habitual over time, inhibition may be maintained without any intention of avoiding the unwanted memory, evolving from an intentional to an unintentional process (i.e., repression).

Anderson et al. (2004) used a "think/no-think paradigm" where participants first learned word pairs (e.g., Ordeal–Roach), and then, during fMRI, were shown one member of a pair (e.g., Ordeal) and told to recall and think about the associated response (e.g., Roach) (respond condition) or to prevent the associated word from entering C for the entire 4-s stimulus presentation (suppression condition). Suppression impaired memory. After scanning, cued recall for Suppression items, when given the originally trained cue, was inferior to recall of Baseline items that did not appear during scanning. So, suppression during scanning made subjects unable to recollect memories that had been formed prescanning, and this memory deficit was beyond what was measured for simple forgetting over time. Furthermore, controlling unwanted memories (suppression) was associated with increased dorsolateral PFC activation and reduced hippocampal activation. Also, the magnitude of forgetting was predicted by both PFC and right hippocampal activations. So people can actively suppress unwanted memories by recruiting dorsolateral PFC involved in executive control (e.g., stopping prepotent motor responses [inhibition], switching task sets, overcoming interference in cognitive tasks) to disengage hippocampal processing (important for declarative memory formation and retrieval). These results establish a neurobiological model for guiding research on motivated forgetting (suppression) and integrate it with fundamental and widely accepted mechanisms of behavior control.

Depue, Curran, and Banich (2007) employed Anderson's think/no-think paradigm (Anderson & Green, 2001; Anderson et al., 2004), but instead used neutral faces as cues and negative pictures as targets. The behavioral evidence showed that subjects effectively suppressed memory. Using fMRI, they found that emotional memories are suppressed by two neural mechanisms: (1) initial suppression by the right inferior frontal gyrus over areas that support sensory elements of the memory representation (e.g., thalamus, visual cortex), preceded by (2) right medial frontal gyrus control over areas that support emotional and multimodal elements of the memory representation (e.g., amygdala, hippocampus), both of which are influenced by frontopolar areas (Figure 1). This implies that memory suppression does in fact occur and is under the control of prefrontal regions, at least in healthy populations.

Another form of "suppression" worth mentioning here is visual perceptual suppression (Blake & Logothetis, 2002; Kim & Blake, 2005; Tsuchiya & Koch, 2005), which occurs when an image—or part of one—is not accessible to conscious perception (i.e., not seen), even though the stimulus is present on the retina. Various paradigms that elicit this type of perceptual suppression are used widely, as they allow the experimental manipulation of the relationship between physical, objective stimuli and subjective, conscious content and therefore the isolation of the neuronal correlates of C.

The best-known form of perceptual rivalry is "binocular rivalry" (BR) (Alais & Blake, 2004), where perceptual content (conscious experience) oscillates, despite constant, if ambiguous, sensory input. In BR,

[5] Some (Erdelyi, 2001) suggest that this distinction in terminology is a distortion of Freud's view by Anna Freud, and that Freud used the term "repression" to refer to both conscious and nonconscious acts (Anderson et al., 2004).

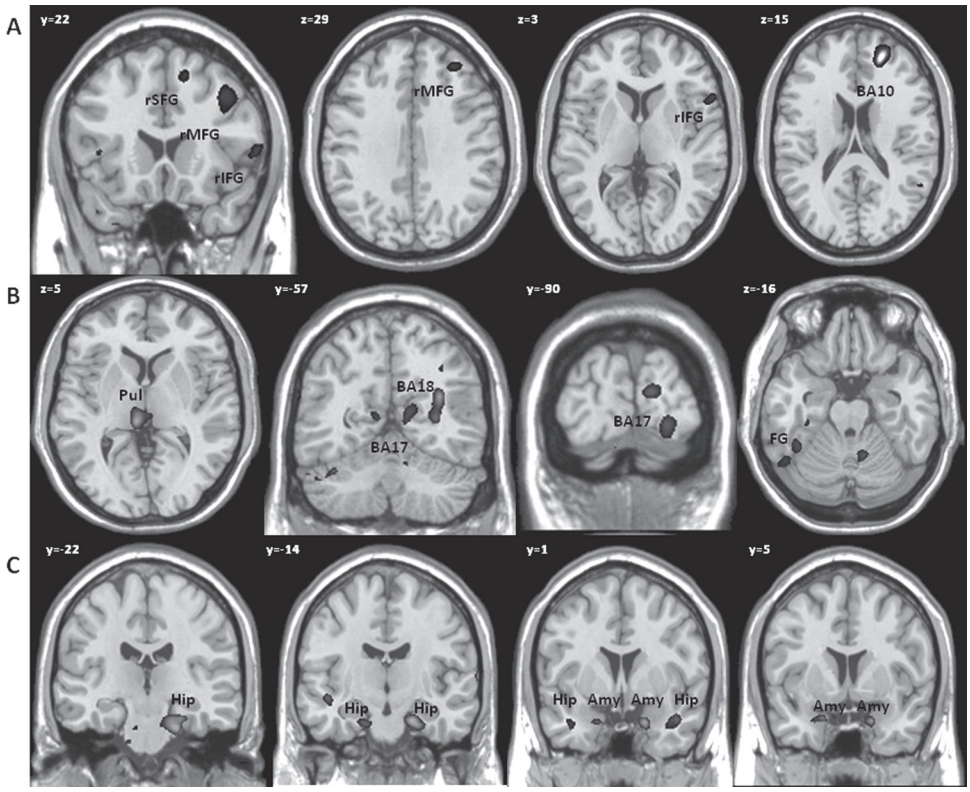

Figure 1. Functional activation of brain areas involved in (A) cognitive control, (B) sensory representations of memory, and (C) memory processes and emotional components of memory (rSFG, right superior frontal gyrus; rMFG, right middle frontal gyrus; rIFG, right inferior frontal gyrus; Pul, pulvinar; FG, fusiform gyrus; Hip, hippocampus; Amy, amygdala) (courtesy of Brendan E. Depue).

two different images are presented simultaneously, one to each eye; rather than perceiving a binocularly fused image, perception alternates between the two images, usually every few seconds, in a seemingly random way, indefinitely. Each "rivaling" image (monocular view) undergoes a period of dominance and of suppression from awareness. The proportion of time each dominates depends on attributes of both images (i.e., their contrast, spatial frequency, content, size, etc.) as well as characteristics of the individual viewer. Selective attention can influence the dominance duration of an image, but whether BR can be controlled by attention is debated. Voluntary, "endogenous" attention appears to be effective only during dominance, but not during suppression (Blake & Logothetis, 2002). Therefore, a more apt description of "perceptual suppression" would be "perceptual repression" due to the lack of conscious control over which stimulus enters awareness and for how long. However, remains to be determined whether the neural mechanisms underlying this form of suppression are related to those underlying psychodynamic suppression and repression.

Dissociation

The concept of "dissociation" was originally put forward by the French psychiatrist Pierre Janet [1859–1947] to describe the "dual consciousness" characteristic of hysteria (Ellenberger, 1970). Dissociation is currently described as a psychological state in which certain thoughts, emotions, sensations, or memories are separated from the rest of the psyche (aka "splitting"), which is not inherently pathological but is more prevalent in people with mental illness (APA, 2000). The DSM-IV-TR (APA, 2000) defines dissociation as "a disruption in the usually integrated functions of consciousness, memory, identity or perception," and specifies five dissociative disorders: dissociative amnesia, dissociative fugue, depersonalization disorder (DPD; Simeon & Abugel, 2006), dissociative identity disorder (DID; formerly multiple personality disorder), and dissociative disorder not otherwise specified (Kihlstrom, 2005). Dissociation may also present as a symptom in other psychiatric disorders (Sar & Ross, 2006).

DPD is a dissociative disorder characterized by a

persistent or recurrent feeling of being detached from one's mental processes or body, accompanied by a sense of unfamiliarity/unreality and hypoemotionality, but with intact reality testing (APA, 2000). People with DPD have difficulties with information processing in relation to the dissociative detachment feature of depersonalization, especially in early perceptual and attentional processes, and with effortful control of the focus of attention (Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007; Guralnick, Schmeidler, & Simeon, 2000; Stein & Simeon, 2009). They have also been shown to have attenuated emotional perception, disrupted emotional memory, and a difficulty in identifying feelings (Medford et al., 2006; Montagne et al., 2007; Simeon, Giesbrecht, Knutelska, Smith, & Smith, 2009).

Sierra and Berrios (1998) put forward a "corticolimbic disconnection hypothesis," which is supported by functional neuroimaging and psychophysiological studies. The hypothesis suggests that depersonalization occurs via a fronto-limbic suppressive mechanism, which is mediated by attention, and generates a state of subjective emotional numbing and disables the process by which perception (including that of one's own body) and cognition become emotionally colored. This emotional "decoloring" results in a qualitative change of conscious awareness and feelings of "unreality" or detachment, which become persistent and dysfunctional in people with DPD (Sierra, 2009; Sierra & Berrios, 1998). More specifically, the authors suggest that hyperactivity of the right PFC (in particular the right dorsolateral PFC) increases alertness, while left PFC activation inhibits the amygdala and other limbic structures (in particular the anterior insula), causing chronic hypoemotionality in DPD (Phillips & Sierra, 2003; Sierra, 2009; Sierra & Berrios, 1998). Understanding the neural basis of C requires an account of the neurocognitive and neurobiological mechanisms that underlie distortions of self-perception such as those seen in the context of DPD.

To further examine the neural basis of dissociation, the next section focuses on DID since it is the most complex, chronic, and severe of the dissociative disorders, and it presents as a symptom in the other dissociative disorders. Challenging the notion of a unitary self-consciousness, DID is characterized by identity fragmentation, rather than proliferation, and is usually associated with a history of severe childhood trauma (Putnam, 1997). DID involves the presence of two or more distinct dissociative identity states, characterized by different emotional responses, cognitions, moods, and perceived self-images, that recurrently and alternately take control of one's behavior and C. Clinical data suggest that the "traumatic identity state" (TIS) has access to traumatic autobiographical memories and intense emotional responses to them. But when in the "neutral identity states" (NIS), patients claim amnesia for traumatic memories (coinciding with the notion of suppression) too extensive to be explained by normal forgetfulness. In the NIS they appear to inhibit access and responses to traumatic memories, processing and responding to trauma-related information as if it pertains to neutral and/or nonautobiographical information, thus enabling daily life function.

Neurobiological studies support the validity of the diagnosis of DID and provide clues to the neural basis of dissociation. In the first controlled structural MRI study of DID, Vermetten, Schmahl, Lindner, Loewenstein, and Bremner (2006) found that compared to healthy controls, DID patients had 19.2% smaller hippocampal and 31.6% smaller amygdalar volumes. Ehling, Nijenhuis, and Krikke (2008) also found that DID patients had smaller hippocampal (25–26%) and amygdala (10–12%) volumes than healthy controls, and those who recovered from DID had more hippocampal volume than those who did not. Stress acting via *N*-methyl-D-aspartic acid (NMDA) receptors in the hippocampus may mediate symptoms of dissociation (Chambers et al., 1999). Early life exposure to elevated glucocorticoid levels, released during stress, may result in progressive hippocampal (a target for glucocorticoids) atrophy (Bremner et al., 2003; Stein, Koverola, Hanna, Torchia, & McClarty, 1997). However, stress may not cause hippocampus damage; rather, those born with a small hippocampus and/or amygdala, perhaps owing to genetics, may be at greater risk for DID. In fact, abused subjects without DID had larger hippocampal and amygdalar volumes than nonabused subjects without DID (Vermetten et al., 2006), perhaps helping protect against early trauma. Psycho- and/or pharmacotherapy for dissociative disorders may increase hippocampal volume (Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003), but longitudinal studies are needed. Coincidently, electrical stimulation of the hippocampus in epilepsy patients resulted in dissociative-like symptoms, including feelings of *déjà vu*, depersonalization, derealization, and memory alterations (Halgren, Walter, Cherlow, & Crandall, 1978; Penfield & Perot, 1963). And ketamine, an NMDA receptor (concentrated in the hippocampus) antagonist, resulted in dissociative symptoms in healthy subjects, including feelings of being out of body, of time standing still, perceptions of body distortions, and amnesia (Krystal et al., 1994).

In relation to an orbitofrontal hypothesis of DID (Forrest, 2001), using single photon emission com-

puted tomography, Sar et al. (Sar, Unal, Kiziltan, Kundakci, & Ozturk, 2001; Sar, Unal, & Ozturk, 2007) found that compared to healthy controls, DID patients had decreased perfusion (regional cerebral blood flow [rCBF] ratio) in the orbitofrontal cortex bilaterally, and increased perfusion in median and superior frontal and occipital regions bilaterally, and in the left lateral temporal region. Dysfunctional interaction between anterior and posterior brain areas may contribute to the neurophysiology of dissociation. Reinders et al. (2003) found specific changes in localized brain activity (via positron emission tomography [PET]) consistent with DID patients' ability to generate at least two distinct mental states of self-awareness, each with its own access to trauma-related memories. The rCBF patterns showed involvement of medial PFC and posterior associative cortices (including parietal areas) in the representation of the different states of C. Based on findings with other "disorders" of C (e.g., see Laureys, 2005; Laureys, Lemaire, Maquet, Phillips, & Franck, 1999; Laureys, Owen, & Schiff, 2004; Laureys et al., 1999, 2000), these highly connected areas have been suggested to be part of the neural network for C.

Data suggest that one brain can generate at least two distinct states of self-awareness, each with its own pattern of perception, reaction, and cognition (Dorahy, 2001; Nijenhuis, van der Hart, & Steele, 2002) and displaying different psychobiological traits that are generally not reproducible in DID-simulating controls (e.g., Miller & Triggiano, 1992; Putnam, 1997). Differential responses in DID patients have been reported in electrodermal activity (Larmore, Ludwig, & Cain, 1977; Ludwig, Brandsma, Wilbur, Bendfeldt, & Jameson, 1972), autonomic nervous system variables (Putnam, Zahn, & Post, 1990), arousal (Putnam, Zahn, & Post, 1990), EEG (Coons, Milstein, & Marley, 1982; Hughes, Kuhlman, Fichtner, & Gruenfeld, 1990; Mesulam, 1981; Putnam, 1993), visual evoked potentials (Putnam, 1992), and rCBF (Mathew, Jack, & West, 1985; Saxe, Vasile, Hill, Bloomingdale, & Van der Kolk, 1992; Tsai, Condie, Wu, & Chang, 1999). Brain areas directly or indirectly involved in emotional and memory processing are most consistently reported as being affected in DID (Dorahy, 2001; Nijenhuis, van der Hart, & Steele, 2002).

Physiologic differences across identity states in DID also include differences in dominant handedness (which may indicate opposing hemispheric control of different identity states), response to the same medication, allergic sensitivities, endocrine function, and optical variables such as variability in visual acuity, refraction, oculomotor status, visual field, color vision, corneal curvature, pupil size, and intraocular pressure in the various DID identity states, compared to healthy controls (Birnbaum & Thomann, 1996). One patient (BT) with DID in response to trauma, gradually regained sight during psychotherapy, after 15 years of diagnosed cortical blindness by neuro-ophthalmic examination (Waldvogel, Ullrich, & Strasburger, 2007). Initially only a few personality states regained vision, while others remained blind. Amazingly, visual evoked potentials were absent in the blind personality states, but normal and stable in the sighted ones. This case shows that, in response to personality changes, the brain has the ability to prevent early visual processing and consequently obstruct conscious visual processing at the cortical level. The neural basis of this ability is being explored (Strasburger et al., 2010). Top-down modulation/suppression of activity in the early stages of visual processing, perhaps at the level of the thalamus or primary visual cortex, may be the neural basis of psychogenic blindness (Berlin & Koch, 2009).

Reinders et al. (2006) were the first to compare the response to trauma-related stimuli in the same DID patients in different dissociative identity states. Differences were found between the NIS and TIS, in response to a trauma-related versus neutral memory, in subjective reactions (emotional and sensorimotor ratings), cardiovascular responses (heart rate, blood pressure, heart-rate variability), and cerebral activation patterns (rCBF via PET). When exposed to identical trauma-related stimuli, the two dissociative identity states exhibited different autonomic and subjective reactions and rCBF patterns, implicating different neural networks. This extends findings in healthy subjects (Anderson et al., 2004) that memory suppression can be transferred to unrelated memories, which Reinders et al. (2006) suggests may result in psychopathology.

So there seems to be a type of "splitting" of C in DID patients. But how does this relate to the neural correlates of C? By what mechanism can multiple selves coexist or alternate in the same brain? There is remarkable similarity between psychiatric and neurological dissociation syndromes, but the main difference is that the former are conceived as a disconnection between psychic functions such as seeing and acting, while the latter are defined in terms of physical disconnection between specialized brain regions such as vision and motor areas. But both types of disorders can be considered disorders of integration, the former because of a "functional" or dynamic impairment of connectivity and the latter because of a neuroanatomical lesion.

Thus, what appears to be altered in both neurological disconnection syndromes and dissociative disorders is not so much the degree of *activity* of a brain area or psychic function, but the degree of *interactiv-*

ity between such areas or functions. Integration of various cortical and subcortical areas appears to be necessary for cohesive conscious experience (Laureys et al., 1999a, 1999b, 2000; Tononi, 2004, 2005). Dissociation may involve disruption of cortico–cortical, thalamo–cortical, amygdalo–cortical, or hippocampo–cortical connectivity (Krystal, Bremner, Southwick, & Charney, 1998). Many of these connections are excitatory NMDA receptor mediated and are blocked by the NMDA antagonist ketamine, which results in dissociative symptoms in healthy subjects. Psychopathologies, like dissociative disorders, that defy the apparent unity of the self, may be failures of coordination or integration of the distributed neural circuitry that represents subjective self-awareness (Kinsbourne, 1998).

The French neurologist Jean-Martin Charcot [1825–1893] believed that the transient effects of hypnosis and the inexplicable neurological symptoms of "hysteria," currently known as "dissociative (conversion) disorder," involved similar brain mechanisms. In line with this, recent studies in cognitive neuroscience reveal that the brain processes involved in symptoms of "hysteria" are in fact similar to those seen in hypnotic phenomena (see Bell, Oakley, Halligan, & Deeley, 2010). Studies also indicate that hypnotizability is associated with a tendency to develop dissociative symptoms, particularly in the area of sensorimotor function, and that suggestions in highly hypnotizable people can replicate dissociative symptoms (Bell et al., 2010). Interestingly, converging evidence indicates that dissociative "symptoms," whether simulated through hypnosis or diagnosed clinically, are linked to increased PFC activation. This implies that interference by the prefrontal/executive system in voluntary and automatic cognitive processes is a shared neural feature of both dissociation and hypnosis. However, systematic, well-controlled, and well-designed experiments investigating the neurocognitive basis of dissociation and hypnosis are needed.

The neural basis of conscious vs. unconscious processes

The evidence described thus far suggests that complex cognition can proceed in the absence of C and that the unconscious brain is active, purposeful, and independent and can selectively access and activate implicit goals and motives. However, exactly how unconscious emotions and evaluations help shape the dynamics of the neural coalitions that give rise to conscious perception is still unknown. Studies suggest that subliminal stimuli produce enough neural activity at a relatively high level of complexity to trigger an appropriate behavioral response. But something in this neural activation is inadequate for conscious experience to arise. So, what is missing?

One possibility is suggested by experiments that show that various cognitive tasks that require awareness are accompanied by short-term temporal correlations among distributed populations of neurons in the thalamocortical system. A coalition of neurons is a collection of mono- or polysynaptically coupled forebrain neurons that dynamically assemble over a fraction of a second to encode a percept, memory, or thought (Koch, 2004). Coalition members reinforce each other and suppress competing coalition members. These competitive interactions can be biased by attention (Koch, 2004). Oscillatory and synchronized neural firing may play a key role in strengthening one coalition over others and in determining which percept enters C (Cosmelli et al., 2004; Engle & Singer, 2001; Gross et al., 2004; Koch, 2004; Rodriguez et al., 1999; Srinivasan, Russell, Edelman, & Tononi, 1999; Swindale, 2003; Thompson & Varela, 2001; Varela, Lachaux, Rodriguez, & Martinerie, 2001). When we become conscious of an event, there is evidence of synchronized activity between widely separated brain regions, particularly within the thalamocortical system (Rodriguez et al., 1999; Srinivasan et al., 1999; Tononi, 2004, 2005). Brief periods of synchronization of oscillating neuronal firing in the gamma range (30–80 Hz) may be an integrative mechanism that brings together a widely distributed group of neurons into a coherent assembly that underlies a cognitive act (Balconi & Lucchiari, 2008; Engle & Singer, 2001; Gross et al., 2004; Meador, Ray, Echauz, Loring, & Vachtsevanos, 2002; Melloni et al., 2007; Nakatani, Ito, Nikolaev, Gong, & van Leeuwen, 2005; Palva, Linkenkaer-Hansen, Naatanen, & Palva, 2005; Rodriguez et al., 1999) and correlates with conscious perception (Doesburg, Kitajo, & Ward, 2005; Fries, Roelfsema, Engel, Konig, & Singer, 1997; Fries, Schroeder, Roelfsema, Singer, & Engel, 2002; Srinivasan et al., 1999).

So, rather than activation of specific brain regions, conscious perception appears to depend on coordinated dynamic states of the cortical network and on transient synchronization of widely distributed neural assemblies (Engel, Fries, Konig, Brecht, & Singer, 1999; Engel & Singer, 2001; Fries et al., 1997, 2002; Lamme, 2006; Melloni et al., 2007; Singer, 2002; Thompson & Varela, 2001). Some evidence suggests the need for a critical level of activation and complexity of widely distributed neuronal assemblies (Greenfield & Collins, 2005) to enable them to be included in the "dominant focus" of C, where information is integrated into the

currently dominant pattern of neuronal activity (Kinsbourne, 1988, 1993, 1997, 1998, 2006).

In general, some (e.g., Greenfield & Collins, 2005; Singer, 2002) argue for more holistic/global properties where activation of many neurons are needed for C. They believe that neurons across the brain synchronize into coordinated assemblies, and then disband, for each conscious experience. So, C is generated by a quantitative increase in holistic brain functioning (e.g., the more neuronal activity the more C) and is not a qualitatively distinct property of the brain. Others argue for more specific local properties of a very specific subset of neurons interacting in a very specific way (e.g., Crick & Koch, 2003; Koch, 2004). They believe that a unique set of neurons in particular brain regions fire in a specific manner for each conscious experience. So, qualitative, not quantitative, differences in neuronal activity give rise to C. Although this is not a theory, it implicates specific mechanisms in space or time or in the brain (e.g., 40-Hz oscillations, temporal synchrony, the PFC, the claustrum, not V1, etc.) that are testable. It implies that the neural basis for specific forms of C perception (e.g., color, motion, faces, familiarity) is restricted to part of the cerebral cortex; so a particular region is an essential node for the particular perceptual trait. However, a combination of both qualitative and quantitative properties of neural firing may be required for conscious experience to arise. For a summary of this debate, see Koch and Greenfield (2007).

Koch (2004; Crick & Koch, 2003) suggests that for conscious visual perception to emerge, neurons at the essential areas in the back of the cortex must receive reciprocal feedback from the planning centers in the front of the brain. He proposes that unless a visual area directly projects into the frontal cortex, activity in that region cannot enter awareness directly, because frontal activity is needed to help establish the dominant coalition of cortical neurons needed for conscious visual perception. Sustained spiking activity that circulates between select neurons in inferotemporal and/or medial temporal cortex and the PFC may constitute the neural basis for object perception (Quiroga, Mukamel, Isham, Malach, & Fried, 2008; Quiroga, Reddy, Kreiman, Koch, & Fried, 2005). The PFC may modulate the competition between sensory networks in the temporal lobe related to conscious perception (Kreiman, Fried, & Koch, 2002). Studies implicate the PFC in top-down control of visual processing in extrastriate cortex and of perceptual transitions during perceptual rivalry (Leopold & Logothetis, 1999; Lumer, Friston, & Rees, 1998). Using ERPs, Del Cul et al. (2007) found that subliminal processing can occur early on in the occipito–temporal pathway (<250 ms poststimulus), but that conscious perception of masked stimuli corresponds to later activity (~300 ms poststimulus) in a broadly distributed fronto–parieto–temporal network. They suggest that this late and highly distributed fronto–parieto–temporal activation may be a marker of C.

Awareness appears to take place hundreds of milliseconds after stimulus presentation, and after the cortical processing that determines the significance and nature of the stimulus (Velmans, 1991). But both conscious and unconscious mental processes are thought to be widespread in, or coextensive with, forebrain function and thus must represent different functional states of that same substrate (Kinsbourne, 1998). Unconscious processes may reflect the neural network in its modular state—that is, relatively isolated loops of action and reaction (Kinsbourne, 1998)—and/or local coordination of neural activity and propagation along sensory processing pathways (Dehaene, Changeux, Naccache, Sackur, & Sergent, 2006). Conscious processes may be the same processes but in some form of global coordination of widely distributed neural activity by long-distance synchronization (Dehaene et al., 2006; Kinsbourne, 1988). Unconscious activity may be mediated by a rapid, feedforward netwave of activity that can trigger neurons, and ultimately behavior, but that is not sufficient to establish a robust coalition for the 500 ms or longer that is necessary for conscious awareness (Koch, 2004).[6]

Conclusion

Since a large part of our mental lives occurs outside of C, with a great deal of it being exceedingly adaptive and advanced, it impels one to question what function (if any) does C actually serve. Unconscious processes appear capable of doing many things previously thought to require deliberation, intention, and conscious awareness, such as processing complex information and emotions, goal pursuit, self-regulation, and cognitive control (Hassin, Uleman, & Bargh, 2005). There have been significant advances from cognitive, neuroscientific, and social perspectives in the empirical study of unconscious mental processes (cognitive, emotional, and motivational), and in understanding their structural and functional neural correlates. This research reveals a new vision of the mind and questions traditional concepts of the self, control of action, and free will.

[6] There has also been some progress in studies on the molecular pathways involved in mediating unconscious processes, e.g. exploring the neurochemistry underlying explicit vs. implicit memory (Nissen, Knopman, & Schacter, 1987; Rammsayer, Rodewald, S., & Groh, 2000).

It is not known how much control an individual (i.e., his or her brain) has over the intricate interaction between unconscious and conscious thought, and how this relates to our concept of free will (Wegner, 2003). We still do not understand exactly how or when *conscious* drives suddenly become *unconscious* (e.g., repression), or *unconscious* drives suddenly become *conscious* (e.g., Freudian slips), or how or when people are able to override hidden urges by force of will (e.g., not acting impulsively; Berlin, Rolls, & Kischka, 2004, 2005; Hollander & Berlin, 2008). To better understand the neural basis of C (Crick & Koch, 2003; Dehaene et al., 2006; Dennett & Kinsbourne, 1992; Humphrey, 2000; Tononi, 2004, 2005), we need to account for the complex, high-level dynamics that occur between unconscious and conscious thought and the neural mechanisms that underlie and distinguish these processes.

Many secrets of the human mind and brain can be revealed when we look to the "disordered" mind and brain for answers and integrate this information with results from animal, single-cell recording, genetic, and imaging studies. Freud had the foresight to look to the brain for answers (Figure 2), but his efforts were limited by the mechanistic understanding and technologies available at the time. New advances in neuroscience and technology are now enabling the neurobiology of the dynamic unconscious that Freud envisioned to come to fruition (e.g., Berti et al., 2005; de Gelder, Morris, & Dolan, 2005; Ramachandran, 1996a; Solms, 1995; Vuilleumier, 2004, 2005; Vuilleumier et al., 2001, 2003). In the process, a good deal of what Freud originally put forth based solely on clinical observations has been revised, refined, and enhanced (Guterl, 2002). But this is to be expected, as the initial insights of every discipline in its early stages require modification over time (Turnbull & Solms, 2007). Only by studying precisely how the human brain processes information will we fully comprehend the true nature of the dynamic unconscious (Tallis, 2002). Devising novel ways, using modern technology, to empirically test dynamic unconscious processes such as repression, suppression, and dissociation will help unveil their neural basis and ultimately lead to more effective treatment options for psychiatric patients, completing the task that Freud began over a century ago.

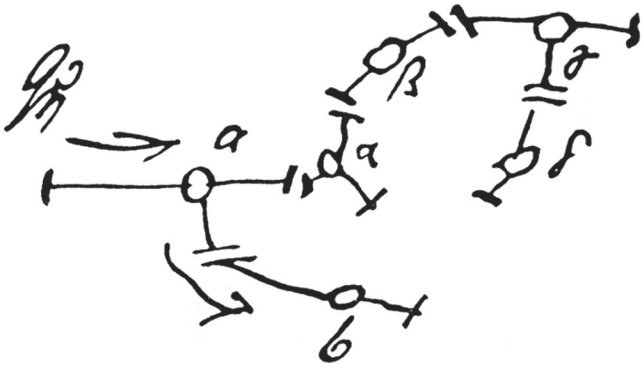

Figure 2. Freud's (1950 [1895]) own sketch of neurons and the flow of neural energy, illustrating his concept of diversion of neural energy via a "side-cathexis." The normal flow of energy (arrow on left labeled *Q'*η) is from neuron "a" to "b." Freud proposed that a side-cathexis of neuron "α" would attract the *Q'*η and divert the flow from neuron "b." He believed this postsynaptic attraction of energy or side-cathexis was the neuronal mechanism underlying repression of forbidden wishes in both waking and dreaming (from McCarley, 1998).

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