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Fear and the Defense Cascade: Clinical Implications and Management

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Abstract: Evolution has endowed all humans with a continuum of innate, hard-wired, automatically activated defense behaviors, termed the *defense cascade*. *Arousal* is the first step in activating the defense cascade; *flight or fight* is an active defense response for dealing with threat; *freezing* is a *flight-or-fight* response put on hold; *tonic immobility* and *collapsed immobility* are responses of last resort to inescapable threat, when active defense responses have failed; and *quiescent immobility* is a state of quiescence that promotes rest and healing. Each of these defense reactions has a distinctive neural pattern mediated by a common neural pathway: activation and inhibition of particular functional components in the amygdala, hypothalamus, periaqueductal gray, and sympathetic and vagal nuclei. Unlike animals, which generally are able to restore their standard mode of functioning once the danger is past, humans often are not, and they may find themselves locked into the same, recurring pattern of response tied in with the original danger or trauma. Understanding the signature patterns of these innate responses—the particular components that combine to yield the given pattern of defense—is important for developing treatment interventions. Effective interventions aim to activate or deactivate one or more components of the signature neural pattern, thereby producing a shift in the neural pattern and, with it, in mind-body state. The process of shifting the neural pattern is the necessary first step in unlocking the patient’s trauma response, in breaking the cycle of suffering, and in helping the patient to adapt to, and overcome, past trauma.

Keywords: collapsed immobility, defense cascade, defense responses, fear behaviors, fight, flaccid immobility, flight, freeze, freezing, fright, quiescent immobility, threat-induced fainting, tonic immobility

In *The Expression of the Emotions in Man and Animals* (1872), Darwin¹ argued that human expressions of emotion resembled those of lower animals and that emotions

are adaptive because they prompt action responses that are beneficial to the organism. Positive emotions promote social-engagement behaviors, whereas negative emotions, many of which are activated by threat, invoke defense responses.^{2,3} Writing in 1908, McDougall⁴ described the various instinctual behaviors that accompanied the emotions of fear, anger, and disgust. Building on McDougall’s ideas, Cannon (1915)⁵ wrote his landmark book, *Bodily Changes in Pain, Hunger, Fear and Rage*, describing the bodily changes that occurred in the context of emotional excitement. That work is best remembered for elaborating the concept of *fight or flight*.^{*} In 1920, Rivers⁷ (a physician working with officers suffering from shell shock during the First World War) described five danger instincts: flight, aggression, manipulative activity, immobility, and

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* The concept of “fight or flight” was firmly established in the first, 1915 edition of *Bodily Changes in Pain, Hunger, Fear and Rage*, where Cannon wrote that “the emotion of fear is associated with the instinct for flight, and the emotion of anger or rage with the instinct for fighting or attack.”^{5(p 187)} In both the first and second (1929)⁶ editions, Cannon referred to these two instincts in a variety of ways: “struggle or flight,”^{5(p 202)} “flight or conflict,”^{5(p 205)} “fighting or flight,”^{5(p 211)} and “fighting or escape that accompany or follow distress or fear or rage.”^{5(p 202)} The authors are unsure when and by whom the catchy term *fight or flight* was introduced when referring to Cannon’s earlier work. We (the authors) use the term *fight or flight* because it better captures the tendency of most mammals to flee, rather than to fight.

collapse.[†] Subsequent research with animals determined that, depending on the degree of threat and the distance between the predator and prey, distinct responses—freezing, flight or fight, tonic immobility, and quiescent immobility—proceed sequentially along a continuum, termed the *defense cascade*.^{‡,2,8,9,17} Researchers likewise began to use the defense cascade to define the progressive defense/fear responses in humans.^{2,15,18,19}

In evolutionary terms the responses that make up the defense cascade are primitive emotional states—coordinated patterns of motor-autonomic-sensory response—that are available to be automatically activated in the context of danger. Emotions are played out “in the theatre of the body.”^{20(p 28)} For humans, the activation of defense responses—the sudden change in motor and physiological state—may be experienced as overwhelming, and beyond conscious control. In clinical practice these phenomena are common, and they occur across a broad range of disorders and clinical presentations: posttraumatic stress disorder (PTSD), peritraumatic reactions (as in physical or sexual assaults, or following accidents or natural disasters), complex trauma, borderline personality disorder, and states of intense distress potentially leading to self-harm.²¹ As every clinician knows, these different states are difficult to understand (what is the underlying dynamic?), difficult to identify and differentiate (what exactly is this state, and how does it differ from other states?), and difficult to manage and treat.

The first goal of this article is to examine the defense responses through the lens of neuroscience and to elaborate a model that explains their brain and body mechanisms. For this purpose we conducted wide-ranging searches for relevant literature on PubMed; identified research from, and sometimes communicated with, laboratories and clinical groups worldwide conducting relevant research; and retrieved and tracked references to seminal articles in the

† Rivers’s term *aggression* is equivalent to fight; *manipulative activity* referred to complex behaviors that functioned to overcome or avoid danger; his term *immobility* is equivalent to freezing; and his term *collapse* encompassed both tonic and collapsed immobility (see his examples of a seal in tonic immobility and a human in collapsed immobility).⁷

‡ William Halse Rivers,⁷ in 1920, was the first to propose a continuum of the instincts of self-preservation, at the end of which were instincts pertaining to the protection of the animal or person from danger—flight, aggression, manipulative activity, immobility, or collapse (including both tonic immobility and collapsed immobility). Subsequently, in 1967, Stanley Ratner⁸ proposed a continuum of innate fear reactions that included startle, watchfulness (freezing), and, at the extreme end of the continuum, prolonged immobility (tonic immobility). He did not include flight or fight—described as such by Cannon in 1915⁶—in his continuum. Subsequently, a number of researchers noted that defense behaviors changed in a patterned manner as a predator approached (sometimes referred to as *predatory imminence*).^{9–13} These efforts to describe responses to predatory imminence included freezing and flight or fight but did not include tonic immobility. Subsequent animal research took place in silos, with some researchers focusing on the responses identified in relation to predatory imminence and others focusing on tonic immobility. In 1997 and 2000, Peter Lang and colleagues^{2,14} introduced the phrase defense cascade and used the notion of predatory imminence to investigate human behavior. And in 2004, Stefan Bracha^{15,16} developed a broader human model that included not only the freeze, flight, and flight responses associated with predatory imminence but also tonic and collapsed immobility.

history of the defense cascade. The second goal is to use that model to understand different clinical presentations and phenomena, and to determine appropriate treatment and management of patients.

Central to the analytical framework for this article is the *defense cascade*. All defense responses in the animal model of the defense cascade—arousal, freezing, flight or fight, tonic immobility, collapsed immobility, and quiescent immobility—are responses to threat mediated by neural circuits involving the extended amygdala, hypothalamus, periaqueductal gray (PAG), ventral pontine tegmentum, ventral and dorsal medulla, and spinal cord.^{22–25} Each defense response has a signature neural pattern that corresponds to a combination of activated connections within a descending neural network (see Figures 1 and 2). This descending network terminates at the level of the effector organs, where it controls a somatomotor component (which involves skeletal muscle), an autonomic/visceromotor component (which involves the viscera), and a pain-processing component. Changes in the patterns of activity of that network mediate the defense cascade and define the different types of defense responses that, taken together, form the defense repertoire of mammalian species. In any particular situation the defense response will be a function of the species-specific defense repertoire,⁸ genetic variations among strains,²⁶ characteristics of the threat, and context in which it occurs, all influenced by individual differences.^{§,27}

As noted previously, each defense response is accompanied by changes in pain processing and sensory processing. Adaptations in pain processing—in particular, the different roles that analgesia plays in each separate defense response—ensures that the animal is able to remain fully focused on the threat and to respond self-protectively, and that the animal’s attention is not distracted by aversive body states such as injuries. Non-opioid analgesia accompanies the “active” defense responses (flight or fight), and opioid analgesia accompanies the “passive” defense responses (freezing, tonic immobility, collapsed immobility, and quiescent immobility).^{28–30} Because opiates induce a state of well-being, it is probable that, during the passive defense responses, opioid analgesia functions on a subjective level to mitigate the intensity of subjective fear. Whereas pain processing has been extensively studied, comparatively little is known about the detailed dynamics of sensory processing during defensive mind-body states; of necessity, our scientific discussion of sensory processing as such (in the first nonclinical section of the article) is therefore limited.** Further information about pain

§ The study by Lanius and colleagues²⁷ investigated the responses of a husband and wife to traumatic script-driven imagery following a car accident in which they had been trapped, had watched a child burn to death, and had feared that they, too, would die. Whereas the husband experienced intense anxiety, arousal, and escape-focused cognitions (flight and fight), the wife experienced both numbness and a subjective sense of immobility (tonic immobility).

** For clinical articles regarding the primacy of data-driven processing—the processing of sensory impressions and perceptual characteristics—during trauma, see Halligan and colleagues (2003)³¹ and Ehling and colleagues (2008).³²

THE DEFENSE CASCADE

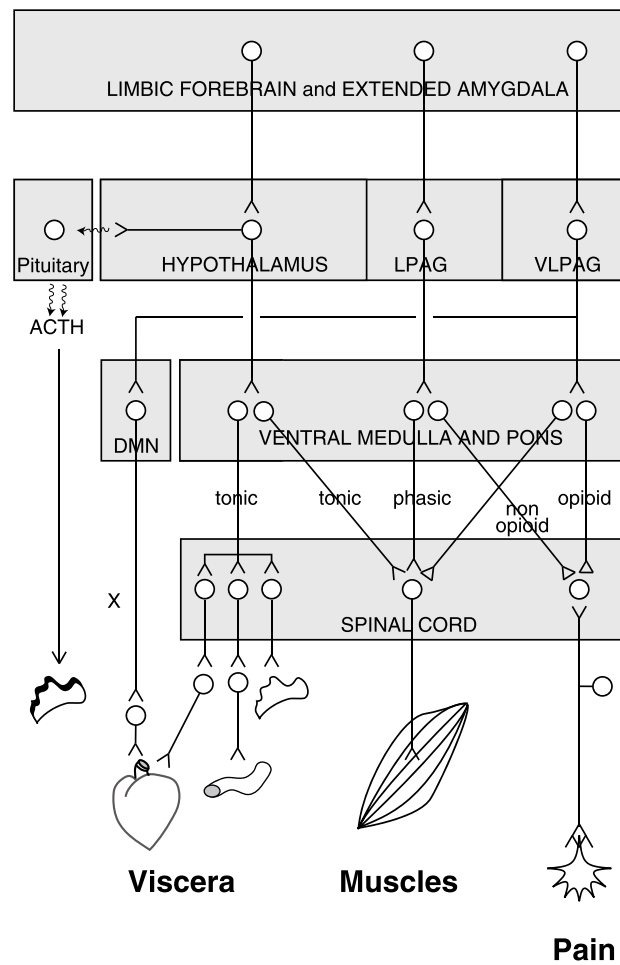
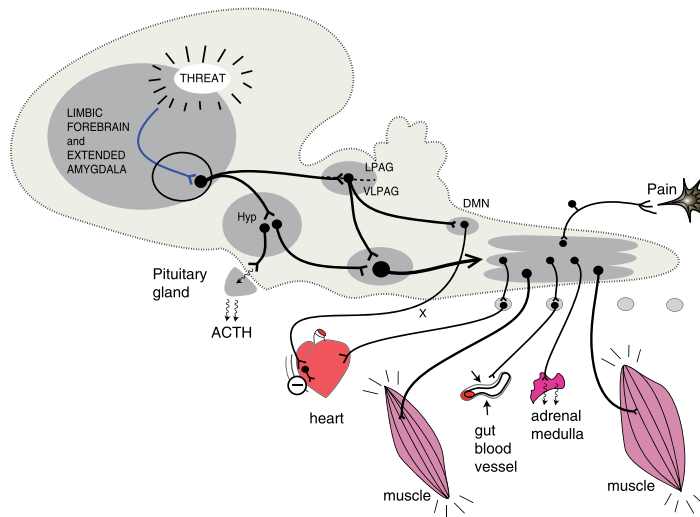


Figure 1. The defense cascade. Schematic views of the descending pathways connecting brain and spinal cord structures to some of the peripheral organs involved in the expression of defense behaviors. The upper panel shows the structures and pathways on a side view of a stylized mammalian brain. The bottom panel is a block diagram of the same information with more details. ACTH, adrenocorticotropic hormone; DMN, dorsal motor nucleus of the vagus; Hyp, hypothalamus; LPAG, lateral periaqueductal gray; VLPAG, ventrolateral periaqueductal gray; X, vagus nerve.

