

How Fear Differs From Anxiety

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Animal models conceptualize anxiety as a response to potential danger while fear is a response to present danger. The way humans experience anxiety involves our capacity for higher thinking while the human experience of fear appears to be much the same as the animal model. This article examines these differences at both a phenomenological and neurological level and highlights implications for the treatment of conditioned fear in PTSD. The stimuli for human fear are sensory-perceptual, while the stimuli for most forms of anxiety are conceptual-linguistic. Individuals in a state of fear/terror undergo a radical shift from top-down to bottom-up processing in which access to conceptual-linguistic thought processes is severely restricted and the frontal regions of the cortex are no longer able to override impulses from brain stem and midbrain regions. Conditioned fear involves actual neurological changes in the limbic system. To overcome a traumatic memory, the individual must (1) gain some level of access to the bottom-up state in order to habituate or extinguish the conditioned fear response, and (2) also achieve access to the top-down state in order to process the fear experience and establish explicit memory. Effective treatments for trauma vary in regard to the degree to which they require the client to enter the bottom-up state, but all activate the fear state and eventually facilitate top-down processing.

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Since PTSD was first identified as a distinct syndrome, it has been grouped with the anxiety disorders. People with PTSD have significant problems with anxiety, yet at the heart of the disorder is a conditioned fear response; it is the central obstacle which must be overcome for any individual to fully recover. The anxiety of individuals with PTSD is related to their fear of reexperiencing the trauma and the conditioned fear associated with it. Too often, fear and anxiety have been characterized as interchangeable constructs but there is a growing consensus that they are significantly different (Gray & McNaughton, 2000; Andreatini, Blanchard, Blanchard, Brandao, Carobrez, Griebel, et al., 2001). The common failure to distinguish between them probably reflects the fact that specific fears do underlie anxieties, and often we can make similar observations about an individual's fears and anxieties. But these observations refer to fearful thoughts; a state of extreme fear, or terror, is very different from the fearful thoughts that drive a state of anxiety.

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Researchers have developed models of animal defensive behavior to distinguish fear from anxiety (Blanchard & Blanchard, 1990a; Lang & Davis, 2000). In these models, fear is viewed as a response to present danger while anxiety is viewed as a response to potential danger (Blanchard & Blanchard, 1990b). Risk assessment is seen as the central component of anxiety in these models; it is operationalized in terms of approaching and scanning potentially dangerous situations. The mental activities associated with assessment of potential dangers would constitute anxiety in humans and would be the analogue of animal risk assessment (Blanchard, Hynd, Minke, Minemoto, & Blanchard, 2002).

The distinction between present and potential dangers provides us with a template for a better understanding of fear and anxiety among people with PTSD, and the past decade of neurobiological research on the fear state provides us with a better understanding of the internal processes that accompany these differences. Distinguishing between fear and anxiety in terms of (a) the source of the stimulus driving the state and (b) the nature of the neurobiological response can inform our treatment approaches and bring a new level of sophistication to our interventions.

The Phenomenology of Fear and Anxiety

Fear and anxiety can each produce a physiological arousal response, involving activation of the adrenergic system in the Central Nervous System (CNS) and the sympathetic branch of the autonomic nervous system (SNS) (Sullivan, Copland, Kent & Gorman, 1999). Because these same systems are involved in both conditions, the phenomenological experience of arousal is similar, possibly differing only in degree. The similarity of these arousal experiences contributes to the common tendency to regard fear and anxiety as either interchangeable concepts or as simply occupying different points along the same continuum. Although they may indeed exist on a continuum, there are significant differences in the two states and these differences have important implications for treatment.

The difference between fear and anxiety starts with the proximity of the threat. The individual in a state of fear perceives the threat to be real and immediate, demanding an active response. The anxious individual, on the other hand, does not perceive an immediate threat; he is focused on a *potential* threat that looms in the near or distant future. Because of the uncertain nature of the anxiety-producing threat, he usually cannot determine whether to act or how to act. Instead, the anxious individual thinks about the threat and what might be done to cope with it—if it indeed should come to pass. Thus, the solution to the fear state is appropriate action directed at the source of the fear while the solution to the anxiety state is murkier. Since the anxiety-producing threat is only a potential—something that might or might not happen—the anxious individual often can do little to solve the problem of the threat. Instead, the anxious individual either continues to think about the threat—and remains anxious—or does something to attend to the anxiety itself.

For example, an individual may be reluctant to leave his house because he has the thought that something bad will happen to him, perhaps he will be attacked. Although he is in a state of distress, which is associated with a fear, the fear of being attacked, he is not actually experiencing the fear of attack itself. Rather, he is experiencing distress about the possibility of being thrust into that feared situation. He is not reacting to an attack, or a perception of being attacked, but to his own thought about the possibility of being attacked. What he is experiencing is anxiety, not fear. If he perceives himself to actually be under attack, then he will enter an actual fear state.

We have a broad array of forms through which we are able to represent our subjective experience. Our immediate sensory images are formed into more complex perceptions when they are synthesized with past memory and generalizations. These sensory-perceptual representations differ from conceptual or categorical forms, which can be further distinguished from linguistic forms (Siegel, 1999). Both conceptual and linguistic forms stem from cognitive sources while perceptual forms stem from somatosensory data. It appears that the anxious individual is responding to his own cognitions—conceptual and linguistic forms—while the fearful individual is responding to sensory perceptions.

The Neurobiology of Fear

Recent advances in cognitive neuroscience have vastly increased our understanding of the biological processes involved in fear. Fear is registered by the lateral nucleus of the amygdala as incoming sensory information is routed to it from sensory regions of the cortex and the thalamus. The lateral nucleus projects to the central nucleus which activates two structures located in lower regions of the brain, the locus ceruleus and the hypothalamus (Nader, Majidishad, Amorapanth & LeDoux, 2001). The locus ceruleus is located in the brain stem; it has fibers connecting to higher level structures in the brain and releases norepinephrine when activated. The hypothalamus is composed of multiple nuclei located in the lower part of the limbic system; when it is signaled by the amygdala, it activates neurological and hormonal messenger systems that prepare the rest of the body (the fight or flight state). While the locus ceruleus sends excitatory transmitters to higher brain regions, the hypothalamus sends excitatory transmitters to lower brain regions, specifically the various centers of the sympathetic branch of the autonomic nervous system (SNS), located in the brain stem and the cerebellum. The hypothalamus also secretes a hormonal messenger, corticotropin releasing factor (CRF), which activates the hypothalamic-pituitary-adrenal (HPA) axis that releases stress hormones into the circulatory system.

The result of these changes is increased levels of the excitatory neurotransmitters, epinephrine and norepinephrine, in the CNS and ANS, and increased levels of stress hormones, the glucocorticoids, as well as epinephrine and norepinephrine (classified as

hormones when they travel through the bloodstream) in the circulatory system (LeDoux, 2000; Maren, 2001). The neural response is extremely rapid and the hormonal response follows within minutes. Non-essential bodily systems (such as digestion) are deactivated and essential systems (such as respiration) are given additional support. At a cellular level, energy resources—especially glucose and oxygen—are prioritized to essential systems (by restricting blood flow to non-essential systems) and glucose is metabolized more rapidly.

The Neurobiology of Conditioned Fear

Fear conditioning occurs when a neutral or non fear-producing event, the conditioned stimulus (CS), becomes associated with a fear-producing event, the unconditioned stimulus (US), and acquires the capacity to elicit the kinds of behavioral, autonomic, and endocrine responses that are normally expressed in the presence of danger. In effect, the CS is processed differently by the brain and acquires the capacity to trigger the fear state. Therefore, many scientists have sought to locate a site of specific change in the brain when fear conditioning has occurred.

Fear conditioning is a form of associative learning that is so powerful it can occur in a single episode. This kind of rapid learning usually involves highly plastic neural cells, particularly those that utilize the neurotransmitter glutamate and have N-methyl-D-aspartate (NMDA) receptors (Schauz & Koch, 2000). These cells allow long-term potentiation (LTP) in neural pathways—learning at a cellular level—to occur rapidly. These cells appear in the highest concentrations in the hippocampus, where they are involved in new learning and the establishment of episodic memory. For several years, it has been thought that some kind of LTP was occurring in the amygdala during fear conditioning (Rogan, Staubli, & LeDoux, 1997; McKernan & Shinnick-Gallagher, 1997; Gewirtz & Davis, 1997). Recent research now shows that neurological changes associated with fear conditioning occur in NMDA cells in the amygdala, especially the lateral nucleus of the amygdala (Blair, Schafe, Bauer, Rodrigues & LeDoux, 2001; Fendt, 2001) and probably throughout the basolateral amygdalar complex (Lee, Choi, Brown & Kim, 2001). Thus, the conditioned fear is a subcortical event; it does not appear to rely on the neocortex.

Disruption of Explicit Memory Formation

Fear conditioning does not occur in the same learning channels as does the kind of normal learning that leads to the establishment of explicit memory. It occurs in the amygdala itself, the same neural structure that sets off the cascade of arousal-related events that constitute the organism's response to a fear stimulus. In addition to activating the hypothalamus and the locus ceruleus, the amygdala acts on the hippocampus. The amygdala has collateral connections to the hippocampus and, during the fear state, it appears that the amygdala sends signals that inhibit hippocampal activity. The hippocampus (actually the septo-hippocampal system) has extensive connections with

associative areas of the cerebral cortex and plays a crucial role in normal learning and the establishment of explicit memory (Gray & McNaughton, 2000). When the fear state reaches the threshold at which hippocampal functioning is inhibited, normal learning and the establishment of explicit memory are disrupted.

In PET scan studies of subjects who are triggered into a state of conditioned fear (by stimulating the recall of traumatic memories through exposure to conditioned stimuli), the activation levels among different brain systems have been observed to change. Several higher cortical areas, including the speech area (Broca's area), become deactivated or less activated during the fear state. Since the semantic component of memory almost certainly involves the speech area, the amygdalar inhibition of the hippocampus probably plays a key role in the disruption of narrative forms of autobiographical experience and the establishment of explicit memory during trauma (van der Kolk, 1994; Metcalfe, 1996).

This disruption of learning is thought to account for many of the symptoms of PTSD; there is no opportunity for the fearful experience to be processed and transformed into the declarative memory system. Instead, the changes in cellular activity are confined to subcortical structures. Encounters with somatosensory stimuli associated with the trauma continue to trigger the conditioned fear and the cascade of events starts anew, thereby interfering with the opportunity to "learn" (explicitly) that the conditioned stimulus is not a real threat. More importantly, the implicit learning that the conditioned stimulus need not be feared must occur at a subcortical level—in the amygdala itself. Recent research suggests that extinguishing the conditioned fear is itself a form of learning that occurs in NMDA cells in the amygdala (Quirk, Russo, Barron & Lebron, 2000; Falls, Miserendino & Davis, 1992).

State of Fear versus State of Anxiety

The disruption of learning and explicit memory occurs during the fear state. Since there is also sympathetic activation during anxiety, perhaps differing only in degree, there is probably a threshold level of amygdalar activation at which the inhibition of the hippocampus is initiated. Prior to that level of activation, the individual might be aroused but still have access to learning and explicit memory processes.

Neuroscientists McNaughton and Gray (2000) have developed a model that describes the patterns of brain activity which they believe are associated with fear versus anxiety. Their view is that: (a) activation of the hippocampus but not the amygdala will constitute non-anxious rumination; (b) activation of the amygdala alone (presumably with the hippocampus inhibited) will constitute pure fear; and (c) activation of both together constitutes anxiety. This formulation is consistent with our knowledge that fear is stimulated by perception and anxiety is stimulated by cognition. In other words, fear is an alarm state precipitated by sensory input and anxiety is an alarm state precipitated by cognitive input (though the cognitions may be initially stimulated by sensory input).

Anxiety might thus be viewed as a state of partial fear in which the individual still does not perceive the threat to be immediately present. Instead, the stimulus driving the partial fear state is the thought about the threat. But at some point, partial fear becomes pure fear and the nature of the threat shifts from a possibility to a perceived reality. When this shift occurs, there is a massive reorganization of neurological functioning: access to thought processes becomes restricted and behavior becomes more reflexive and less cognitively controlled.

From Top-Down to Bottom-Up Processing

In addition to the interruption of normal learning and the establishment of explicit memory, the fear state disrupts other aspects of cortical functioning. The overall result is a change in how information is processed and decisions are made; there is a shift from cortical to subcortical control of many aspects of functioning. This change has been characterized as a shift from top-down to bottom-up processing of information.

Top-down processing is initiated by the frontal cortex and involves cognition, while bottom-up processing is initiated at brainstem and midbrain regions and involves less thought and more autonomic and reflexive actions. Impulsive, emotional actions probably reflect bottom-up processing (Ogden & Minton, 2000). During top-down processing the upper level of the brain can override, steer or interrupt the lower levels (LeDoux, 1996). This can be seen in appetitive behaviors, such as thirst (Lang, Bradley & Cuthbert, 1998). If an individual is not too dehydrated, he can decide to ignore bottom-up signals of thirst and continue other activities. However, if the thirst reaches a critical threshold, then processing may shift to the bottom-up level and he will no longer be able to override it cognitively. If he encounters water, he may rush to it even if he has cognitive reasons to hold back. So a thirsty man who walks out of the desert and finds water may rush to drink it even though he knows (cognitively) that he should purify it first.

The shift to bottom-up processing during the fear state involves a mechanism in which the lower levels of the brain are able to override the higher levels. This requires both (a) activation of the bottom-up regions and (b) interruption of the top-down regions. Both of these changes appear to occur as a result of the intense activation of the amygdala during the fear state. Activation of the bottom-up regions occurs through amygdalar stimulation of the hypothalamus, which activates the SNS and the HPA axis. The interruption of top-down regions occurs in at least two ways, (a) the amygdalar inhibition of the hippocampal pathway to the cortex (discussed in previous section) and (b) the locus ceruleus's disruption of cortical areas that oversee the functioning of the amygdala (discussed below).

After the amygdala provides an emotional valence to sensory input, the information is transmitted to the orbitofrontal cortex (OFC) which organizes the organism's response

based on the implications of the valenced sensory data. There is also a sort of feedback loop in which the sensory input to the amygdala is modulated by the OFC. The OFC thereby functions as the “master regulator for organization of the brain’s response to threat” (Scaer, 2001). In addition, it appears that the anterior cingulate cortex provides a “gating” function in which it doses the amount of sensory input from the cortical sensory regions to the amygdala (Hamner, Loberbaum & George, 1999). The OFC and anterior cingulate cortex thus protect the amygdala from being overwhelmed by emotionally laden input. However, when the organism goes into the fear/alarm state, the OFC and anterior cingulate receive intense noradrenergic input from the locus ceruleus and their functioning is interrupted (Scaer, 2001; Hamner, Loberbaum & George, 1999). They cease providing the modulation and gating of data going to the amygdala; the amygdala is then wide open to the inflow of threatening sensory data. The result is that the individual loses cortical control of the brain’s response to fear and shifts to a subcortical, reflexive mode of functioning. In a sense, the amygdala is able to override (through activation of the locus ceruleus) its own cortical governing system during emergencies.

Operating in the Bottom-Up State

When we are in a state of bottom-up processing, access to our normal thought processes is severely limited (Schoer, 1994). We are absorbed in the immediate moment and our intellectual capacities are focused on the perceived threat. Since the hippocampus and the speech center (Broca’s area) are less active during bottom-up processing, episodic memory is disrupted and we are largely unable to construct narrative expressions of our autobiographical experience. This is believed to interfere with our ability to incorporate autobiographical experience into our declarative memory system and may account for many of the symptoms of PTSD (van der Kolk, 1994).

This shift in processing constitutes one of the central differences between anxiety and fear—when an individual is in a state of sufficient fear, processing is bottom-up. When the individual is anxious, he is still functioning from the top-down but the activation of lower structures requires him to deal with—and probably account for—the state of physiological arousal. Since he is still processing from a top-down mode, his efforts to deal with the arousal state include his mental activities. This option is simply less available in the bottom-up processing mode.

The Stimulus: Conception versus Perception

“Emotions occur in two types of circumstances. The first type of circumstance takes place when the organism processes certain objects or situations with one of its sensory devices . . . The second type of circumstance occurs when the mind of an organism conjures up from memory certain objects and situations and represents them as images in the thought process.”

--Antonio Damasio, 1999, p. 56

The primary stimuli for anxiety are the thoughts about potential feared events (a secondary stimulus is the arousal state itself) but the actual likelihood of the feared events occurring still lies on a continuum from those events that are highly unlikely to events that are almost certain to occur. The more certainty we have that the event will occur, the closer the anxiety state moves toward being a fear state. In other words, the less the feared event is viewed as a possibility, and the more it becomes a certainty, the more our processing shifts from conceptual-linguistic to sensory-perceptual—and our state shifts from anxiety to fear. If an individual is near a construction site and begins to worry about the possibility that he might be hit by falling materials, he will be in a state of anxiety. If an individual sees that he is standing under a heavy construction pallet that is dangling from a broken cable, he either will be in a fear state or close to entering one: his focus will be on his perception, and he is likely to take action without even thinking about it.

The more likely the event becomes, the more the individual is responding to a perception of danger rather than a conception of danger. *The shift to bottom-up processing occurs when the concept of potential danger becomes a percept of actual danger.* This concept-percept distinction is reflected in memory processes. Tests of memory can assess the conceptual features of a word or they can assess the physical features of the same word. The conceptual features refer to the meaning of the word while the physical features refer to visible dimensions, such as its spelling. Such tests are able to distinguish conceptual memory from perceptual memory and a variety of studies now show that these two types of memory map to very different areas of the cortex, the perceptual memory being confined primarily to sensory areas (Blaxton, 1999).

Our response to a distressed individual will differ significantly depending upon whether we view his distress as a state of fear or one of anxiety: i.e., whether we think he is reacting to an actual perception. This can be seen in our response to the child who is anxious about encountering monsters at night. If he is anxious at bedtime, we may read to him, rock him, sing to him or even talk to him about his fears. But if he awakens in the middle of the night in a state of terror and insists there is a monster in the closet, we turn on the light and show him that his perception is incorrect. We do not simply soothe him or discuss the irrationality of his fears, we address the source of the fear—his perception. After we have responded to the fear state, we will probably soothe him and help him lower his anxiety so he can return to sleep but his state of fear demands that our first response address the source of the fear—his perception of danger.

Treatment Implications

In the treatment of anxiety, we can address either the anxiety or its source or both. We can soothe the individual's physiological state of arousal through techniques such as relaxation exercises or guided imagery. We can address the stimulus of the anxiety through approaches such as insight oriented therapy or cognitive restructuring. Or we can do both by soothing the arousal while we seek to change the individual's relationship with the stimulus through approaches like systematic desensitization or exposure therapy.

All of these approaches have shown some efficacy with anxiety disorders but that highlights the importance of identifying the different nature of a fear disorder like PTSD; conditioned fear cannot be effectively treated with many of the traditional approaches to anxiety. Simply soothing the arousal will not change a fear disorder because it does not address the conditioned fear. Dealing with cognitive stimuli, as in insight therapy, will not change a fear disorder because the stimulus is perception, not conception. In order to have any hope of extinguishing conditioned fear, the perception of danger and the accompanying fear state must be activated; i.e., the subject must be exposed to the feared stimuli.

The importance of exposure in trauma treatment has been clear for more than a decade (Solomon, Gerrity & Muff, 1992). Virtually all the approaches that have shown success in treating conditioned fear/trauma involve some kind of exposure that activates the fear structure (Foa, Riggs, Massie, & Yarczower, 1995; Foa, Keane, & Friedman, 2000). Most techniques involve forms of imaginal exposure, in which the subject accesses the fear state by recalling images of the trauma; however, some approaches utilize in vivo exposure techniques that utilize actual stimuli related to the trauma (Keane, 1995). Currently, prolonged exposure (PE) has become the standard against which other approaches are measured (Rothbaum & Foa, 1999; Foa, Dancu, Hembree, Jaycox, Meadows & Street, 1999). Prolonged exposure takes the subject into the fear state but transforms the experience into being less fear-producing through repeated exposures in a safe setting, thereby facilitating habituation. It seems likely that the reduction in arousal with repeated exposures allows the subject to resume top-down processing while responding to the conditioned stimulus, and this permits the somatosensory data finally to be committed to explicit memory.

Since activation of the fear state can cause a shift to bottom-up processing and interfere with the individual's capacity to cognitively process the experience, trauma clinicians have struggled with how to productively access the fear without simply recreating the fear state. This difficulty is often framed in terms of retraumatizing the client. A variety of approaches have been developed, spanning a range of how far the client is allowed to go into the state of bottom-up processing.

The Range of Exposure Treatments

The phenomenal growth in innovative trauma treatments was particularly spurred by van der Kolk's 1994 paper, *The Body Keeps the Score*. This publication marks the beginning of the widespread awareness that important parts of cortical functioning—especially access to the declarative memory system—become deactivated during traumatic arousal (when processing shifts from top-down to bottom-up). Foa and Kozak (1986) emphasized the importance of altering the memory structure that underlies irrational fear. The implication of van der Kolk's observations was that trauma clients not

only needed to access the fear state, but would ultimately need to (re)achieve top-down processing in order to alter that memory structure and commit it to explicit memory.

In the past decade, a vast number of treatments have been developed that either focus on the body or include attention to the body. All of them activate the perceptions associated with the fear state and activate the fear state itself to varying degrees. Some, such as Eye Movement Desensitization/Reprocessing (EMDR) (Shapiro, 1995) and Rothschild's somatic focus (2000), do not involve actual manipulation of the body. Others, such as Thought Field therapy (TFT) (Callahan & Callahan, 1996) and its many derivatives, involve physical touching of the body, either by the client or by the therapist. These approaches generally activate the fear state through imaginal exposure and then use physical techniques that serve to reduce the arousal level. Practitioners of these approaches conceptualize their therapeutic action from a variety of perspectives. However, in terms of the model being presented here, all exposure treatments are either inducing, or starting to induce, bottom-up processing while lowering arousal so that the individual can either maintain or shift back to top-down processing. This allows the individual to access, habituate, and process the conditioned fear.

Trauma treatment approaches can be differentiated according to the extent to which they activate bottom-up processing during exposure. Some approaches take clients toward the fear state but back away when it begins to build, others take clients into the fear state for limited doses or from a controlled distance, and some immerse the client in the fear state until the conditioned fear habituates.

Maintaining top-down processing. A therapy that exemplifies careful management of bottom-up processing has been developed by Rothschild (2000). She keenly attends to the client's somatic experience and approaches the fear state but prevents the shift to bottom-up processing. She uses imaginal exposure in the usual fashion but helps the subject to back away from the material as soon as she sees signs of sympathetic arousal. Thus, she helps her clients approach the state of total fear but prevents the full shift to bottom-up processing. By helping them to pull back, she keeps them in the top-down mode so that they are able to process the verbal/semantic components of explicit memory.

Visual Kinesthetic Dissociation (VKD) (Hossack & Bentall, 1996) has a different way of achieving the goal of maintaining top-down processing. The client is helped to establish a point of view on the traumatic memories that prevents him from becoming totally absorbed in the memory experience. Consequently, the client gets close to the state of bottom-up processing but does not fully enter it. Other approaches that operate in the top-down mode (approaching but not entering the bottom-up mode) include Thought Field Therapy (TFT) (Callahan & Callahan, 1996) and many of the energy therapies. These approaches typically activate the memories and then use a variety of physical techniques that serve to attenuate the arousal state. Some of these may fall into the next category, approaches that go further into the bottom-up state but exit it rapidly.

Briefly entering bottom-up states. Some approaches take the client into the fear state for a time-limited dose that allows containment of the bottom-up experience and an opportunity to do top-down processing when the client emerges from the fear state. Ochberg's Counting Method (1993) is perhaps the best example of this, he creates a context that allows the client to shift deeply into the traumatic memories and yet pull out of them again within a few seconds time. Thus, he is helping the client to access the trauma memories—and the bottom-up state—in doses.

In a less controlled approach to dosing, Traumatic Incident Reduction (TIR) (French & Harris, 1998) has the client run through the complete memory of a trauma from beginning to end, while safely (albeit silently) connected to the therapist. Forays into the bottom-up state are done repeatedly, hence the client has the experience of shifting back and forth between top-down and bottom-up processing a number of times. Again, the shift back to top-down processing presumably allows processing to occur. Another approach that is geared to limit the extent of bottom-up processing is Ogden and Minton's (2000) sensorimotor therapy.

EMDR probably falls in this category as well. The client is helped to engage the fear memories on a number of levels (sensory, somatic, cognitive) and then the eye movements or bilateral stimulation has an attenuating effect. This approach is more highly researched than the other non-traditional approaches and so it is interesting that the method has been conceptualized in terms of accelerated information processing (Shapiro, 1995). This is consistent with the view that resuming the top-down mode facilitates processing of the information associated with the trauma.

Habituating in the bottom-up state. At the furthest end of the continuum are approaches that induce the fear state and keep the client in it until it loses its power and there is a spontaneous shift back to top-down processing. Some of these, such as flooding, operate on a purely behavioral paradigm of habituation. Others utilize a somatic model that presumes an unfinished biological pattern that needs to run to completion. This view is expressed by Levine's (1997) somatically oriented therapy, which is based on observations of the freeze response among prey animals. Levine notes that these animals subsequently shake and go through a fixed pattern that he believes represents the completion of the fear response.

Conclusion

“The most fundamental aggression to ourselves, the most fundamental harm we can do to ourselves, is to remain ignorant by not having the courage and the respect to look at ourselves honestly and gently.”

Pema Chödrön, 2000, p.32

Fear differs from anxiety in at least two respects: (a) it is a response to an actual, perceived threat rather than a possibility and (b) it involves a shift from top-down to bottom-up processing. Individuals who live with conditioned fear have fearful thoughts about the possibility of entering the fear state; i.e., they develop anxiety. Rather than go into the fear state and remain there long enough for the conditioned fear to habituate, they develop the patterns of avoidance that lead to the development of the PTSD syndrome. Were they to approach their fear and examine it rather than avoid it, they would have a much greater opportunity to overcome it. This is what is accomplished in the various forms of exposure treatment; the individual is helped to approach the fear state and experience it in a safe setting.

The difference between top-down and bottom-up processing dictates very different directions in treatment. Forms of therapy that deal with the client's thoughts but do not approach the fear state may be effective with many anxiety disorders but they will not be effective with PTSD because they are not addressing the underlying conditioned fear. We may be able to talk an individual into a less anxious state but we cannot talk someone out of feeling fearful. Sometimes we may be able to change an individual's perception ("That's not an alligator, it's just a log") but if the perception does not change then we must address either the stimulus ("We will move the boat away from the alligator") or the response (the conditioned fear).

So in order to treat someone with PTSD, the fear state must be brought into the consulting room. Yet this emphasis on exposure to the fear state should not be taken to mean that PTSD treatment is purely about overcoming the conditioned fear. The conditioned fear is at the heart of the disorder and must be addressed or treatment will ultimately fail. But even when the conditioned fear has been extinguished, there are usually many related problems, particularly if the disorder has been chronic. These other problems range from disrupted schemas of the self and systems of meaning to problems with affect regulation and interpersonal difficulties. These kinds of problems require the full range of therapy approaches, from cognitive to insight-oriented to systems therapy, and all require a safe treatment relationship.

The combination of fear, anxiety, cognitive distortions and problems with affect regulation in PTSD creates a unique challenge for treatment and highlights the importance of distinguishing between top-down and bottom-up processing. We can only extinguish the conditioned fear response in the bottom-up state, but we can only process the experience into explicit memory and change the maladaptive ideas, attitudes and schemas of self and world in the top-down state. Sometimes we get access to a trauma client before he or she has developed the latter but, sadly, that is seldom the case. Too often, we must access the fear state in an individual who already has significant problems in his or her working models of self and other. Then, even when the bottom-up state has been addressed, there is still plenty of work to be done from the top-down.

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