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Clinical Implications of Neuroscience Research in PTSD

BESSEL A. VAN DER KOLK

Boston University School of Medicine, The Trauma Center, 1269 Beacon Street, Brookline, Massachusetts, USA **Q1**

ABSTRACT: The research showing how exposure to extreme stress affects brain function is making important contributions to understanding the nature of traumatic stress. This includes the notion that traumatized individuals are vulnerable to react to sensory information with subcortically initiated responses that are irrelevant, and often harmful, in the present. Reminders of traumatic experiences activate brain regions that support intense emotions, and decrease activation in the central nervous system (CNS) regions involved in (*a*) the integration of sensory input with motor output, (*b*) the modulation of physiological arousal, and (*c*) the capacity to communicate experience in words. Failures of attention and memory in posttraumatic stress disorder (PTSD) interfere with the capacity to engage in the present: traumatized individuals "lose their way in the world." This article discusses the implications of this research by suggesting that effective treatment needs to involve (1) learning to tolerate feelings and sensations by increasing the capacity for interoception, (2) learning to modulate arousal, and (3) learning that after confrontation with physical helplessness it is essential to engage in taking effective action.

KEYWORDS: PTSD; affect regulation; neuroimaging; meditation; yoga; HRV; introspection; movement; action; medial prefrontal cortex; autonomic nervous system

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The discovery that sensory input can automatically stimulate hormonal secretions and influence the activation of brain regions involved in attention and memory once again confronts psychology with the limitations of conscious control over our actions and emotions. This is particularly relevant for understanding and treating traumatized individuals. The fact that reminders of the past automatically activate certain neurobiological responses explains why trauma survivors are vulnerable to react with irrational-–subcortically initiated

Address for correspondence: B.A. van der Kolk, M.D., Boston University School of Medicine, 1269 Beacon Street, Brookline MA 02446. Voice: 617-247-3918; fax: 617-859-9805.
e-mail: bvanderk@traumacenter.org

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responses that are irrelevant, and even harmful, in the present. Traumatized individuals may blow up in response to minor provocations; freeze when frustrated, or become helpless in the face of trivial challenges. Without a historical context to understand the somatic and behavioral residues from the past, their emotions appear out of place and their actions bizarre.

Our first neuroimaging study of PTSD[1] using a script-driven imagery symptom provocation paradigm demonstrated that imaging studies can help clarify the underlying neurobiological changes responsible for the problems with reliving, attention, and arousal characteristic of PTSD. Exposed to traumatic reminders, subjects had cerebral blood flow increases in the right medial orbitofrontal cortex, insula, amygdala, and anterior temporal pole, and in a relative deactivation in the left anterior prefrontal cortex, specifically in Broca's area, the expressive speech center in the brain, the area necessary to communicate what one is thinking and feeling. This, and subsequent research supporting those findings[2–4] demonstrated that when people are reminded of a personal trauma they activate brain regions that support intense emotions, while decreasing activity of brain structures involved in the inhibition of emotions and the translation of experience into communicable language. These and other findings related to neuronal activation in response to traumatic reminders have enormous potential for articulating the targets for effective intervention and treatment.

BRAIN AS AN ORGAN DEVOTED TO TAKING EFFECTIVE ACTION

Neuroscience research has provided important new insights into the processing of intense emotions. The laboratories of Antonio Damasio,[5] Joseph LeDoux,[6] Jaak Panksepp,[7] Steve Porges,[8] Rodolfo Llinas,[9] and Richie Davidson[10] have shown that living creatures more or less automatically respond to incoming sensory information with relatively stable neuronal and hormonal activation, resulting in consistent action patterns: predictable behaviors that can be elicited over and over again in response to similar input. Under ordinary conditions the executive and symbolizing capacities of the prefrontal cortex can modify these behaviors by providing the ability to observe, know, and predict by inhibiting, organizing, and modulating those automatic responses. This allows people to manage and preserve their relationships with their fellow human beings on whom they so profoundly depend for meaning, company, affirmation, protection, and connection.

At the end of the 19th century the British neurologist John Hughlings Jackson first proposed that the brain is hierarchically organized—from the "bottom up." The organism responds to incoming information by automatically activating emotional and arousal systems that stimulate action tendencies that can be modified by thought. The highest level of integration and coordination

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depends on prefrontal activity that allows the organism to flexibly adjust to the environment. Jackson proposed that "the higher nervous arrangements inhibit (or control) the lower, and thus, when the higher are suddenly rendered functionless, the lower rise in activity."[11] A similar trilevel model is also seen in MacLean's triune brain.[12]

What makes people unique in the animal kingdom is their flexibility: their capacity to make choices about how to respond. This flexibility is the result of the property of the human neocortex to integrate a large variety of different pieces of information, to attach meaning to both the incoming input and the physical urges (tendencies) that these evoke, and to apply logical thought to calculate the long-term effect of their actions. This allows people to continuously discover new ways of dealing with information and to modify their responses on the basis of the lessons they learn. This accounts for the fact that human behavior is much more complex than the purely instinctual and conditioned behavior seen in other species.

However, this capacity to respond in a flexible manner emerges only slowly during the course of human development and is easily disrupted. Small children have little control over their crying and clinging when they feel abandoned, nor do they have much control over showing their excitement when they are delighted. They depend on their adult caregivers to take action after they signal their distress. That caregiver needs to figure out what is going on and needs to change the conditions in order to restore the homeostasis of the child. Throughout the life cycle, the presence of familiar and trusted human beings continues to have a profound affect on the modulation of autonomic arousal (e.g., see Ref. 8). Children only develop autonomy when they start developing a prefrontal cortex. This allows them to appraise their internal states and to execute the actions necessary to restore disturbances in homeostasis. According to Jean Piaget, the goal of development is "decentration": having *your* emotions, not *being* them.

Adults remain prone to automatically engage in relative fixed action patterns-routine ways of dealing with life that are interrupted when the usual actions do not achieve the required results. Thwarting activates emotions—signals that something is wrong: feelings of frustration, discouragement, disgust, or rage, which, in turn, either propel people to change their course of action or to enlist the help of others. People (and animals) execute whatever "action tendency" is associated with any particular emotion: confrontation and inhibition with anger, physical paralysis with fear, physical collapse in response to helplessness, an inexorable impulse to move toward sources of joy, such as running toward people one loves, followed by an urge to embrace them, etc.

The rational mind, while able to *organize* feelings and impulses, does not seem to be particularly well equipped to *abolish* emotions, thoughts, and impulses. Neuroimaging studies of human beings in highly emotional states reveal that intense emotions of fear, sadness, anger, and happiness cause increased activation in subcortical brain regions and significant reductions of blood flow in

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various areas in the frontal lobe.[13] This provides a neurobiological understanding of the clinical observation that people usually have difficulty organizing a modulated behavioral response when they experience intense emotions.

Emotions occur not by conscious choice, but by disposition: limbic brain structures, such as the amygdala tag incoming stimuli and determine their emotional significance. Emotional significance, in turn determines the response, what action is taken. In other words, emotional valence decides the *physical* reaction of the organism.[5] Charles Darwin,[14] Ivan Pavlov,[15] and William James[16] all noted that the function of emotions is to take physical action. As Roger Sperry, Nobel Prize 1981, said: "the brain is an organ of and for movement": The brain is the organ that moves the muscles. It does many other things, but all of them are secondary to making our bodies move."[17] Sperry claimed that even perception is secondary to movement: "In so far as an organism perceives a given object, it is prepared to respond to it. . .The presence or absence of adaptive reaction potentialities, ready to discharge into motor patterns, makes the difference between perceiving and not perceiving."[17]

Nina Bull,[18] Jaak Panksepp,[7] Antonio Damasio,[5] and others have demonstrated that each particular emotional state automatically activates distinct action tendencies: a programmed sequence of actions that function to help the organism cope with environmental challenges. NYU neuroscientist Rodolfo Llinas summarizes the role of the central nervous system (CNS) in generating action as follows: in order to make its way in the world any activelymoving creature must be able to predict what is to come and find a way to where it needs to go. Prediction occurs by the formation of a sensorimotor image, based on hearing, vision, or touch. This contextualizes theexternal world and compares it with the existing internal map. "The . . . comparison of internal and external worlds [results in] appropriate action: a movement is made" (p.38). People experience the combinations of sensations and an urge for physical activation as a physical feeling or an emotion.[9]

People who suffer from PTSD seem to lose their way in the world. Since at least 1889 it has been noted that traumatized individuals are prone to respond to reminders of the past by automatically engaging in physical actions that must have been appropriate at the time of the trauma, but that are no longer relevant.[19] In "the Traumatic Neuroses of War" Kardiner[20] described how WWI veterans riding on the New York subway were prone to duck in fear and behave as if they were back in the trenches when the train entered a tunnel. As Pierre Janet noticed: "traumatized patients are continuing the action, or rather the attempt at action, which began when the thing happened and they exhaust themselves in these everlasting recommencements."[21]

Neuropsychology and neuroimaging research demonstrate that traumatized individuals have problems with sustained attention and working memory, which causes difficulty performing with focused concentration, and hence, with being fully engaged in the present. This is most likely the result of a

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dysfunction of frontal–subcortical circuitry, and deficits in corticothalamic integration.[22,23]

Many traumatized children and adults, confronted with chronically overwhelming emotions, lose their capacity to use emotions as guides for effective action. They often do not recognize what they are feeling and fail to mount an appropriate response. This phenomenon is called "alexithymia,"[24] an inability to identify the meaning of physical sensations and muscle activation. Failure to recognize what is going on causes them to be out of touch with their needs, and, as a consequence, they are unable to take care of them. This inability to correctly identify sensations, emotions, and physical states often extends itself to having difficulty appreciating the emotional states and needs of those around them. Unable to gauge and modulate their own internal states they habitually collapse in the face of threat, or lash out in response to minor irritations. Futility becomes the hallmark of daily life.

Psychology and psychiatry, as disciplines, have paid scant attention to the deficient orientation and action patterns that are triggered by sensory input, and, instead, tend to narrowly focus on either neurochemistry or emotional states. They thereby may have lost sight of the forest for the trees: both neurochemistry and emotions are activated *in order to* bring about action: either to engage in physical movements to protect, engage, or defend or displaying bodily postures denoting fear, anger, or depression that invite others to change their behavior. Pharmacotherapy helps to address some of the neurochemical problems associated with PTSD, thereby helping to modulate some of the embarrassing and upsetting behaviors and emotions, but drugs seem to not really be able to *correct* whatever abnormality underlies these behaviors and emotions.

When clinicians rediscovered the profound disruptions in the experience of physical sensations and the automatic activation of fixed action patterns in traumatized children and adults they found themselves at a loss on how to address these deficits. One thing was clear: the rational, executive brain, the mind, the part that needs to be functional in order to engage in the process of psychotherapy, has very limited capacity to squelch sensations, control emotional arousal, or change fixed action patterns. The problem that Damasio articulated had to be solved: "*We use our minds not to discover facts but to hide them. One of things the screen hides most effectively is the body, our own body, by which I mean, the ins and outs of it, its interiors. Like a veil thrown over the skin to secure its modesty, the screen partially removes from the mind the inner states of the body, those that constitute the flow of life as it wanders in the journey of each day. The elusiveness of emotions and feelings is probably . . . an indication of how we cover to the presentation of our bodies, how much mental imagery masks the reality of the body" (p.28).*[5]

Given that understanding and insight are the main staples of both cognitive behavioral therapy (CBT) and psychodynamic psychotherapy, the principal therapies currently taught in professional schools, the discoveries of

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neuroscience have been difficult to integrate into therapeutic practice. Neither CBT protocols nor psychodynamic therapeutic techniques pay sufficient attention to the experience and interpretation of disturbed physical sensations and preprogrammed physical action patterns. Since Joseph LeDoux had shown that, at least in rats, "emotional memories are forever" and that the dorsolateral prefrontal cortex (dlPFC), which is involved with insight, understanding, and planning for the future, has virtually no connecting pathways to the brain centers that generate and elaborate emotions, the best therapy claimed to offer is to help people inhibit the automatic physical actions that emotions provoke—limited extinction, and helping people with "anger management" and quieting them down before blowing off the handle, such as by counting to 10 and taking deep breaths.[2]

The realization that insight and understanding are usually not enough to keep traumatized people from regularly feeling and acting as if they are traumatized all over again forced clinicians to explore techniques that offer the possibility of reprogramming these automatic physical responses. It was only natural that this would involve addressing awareness of internal sensations and physical action patterns. The closest mainstream protocolized therapeutic technique that involves such "mindfulness" currently is dialectical behavior therapy (DBT).[25] However, many non-Western cultures have healing traditions that activate and use physical movement and breath, such as yoga, chi qong, and tai chi all of which claim to regulate emotional and physiological states. In contrast, in the West working with sensation and movement has been fragmented and has stayed outside the mainstream of medical and psychological teaching. Yet, working with sensation and movement has been extensively explored in such techniques as focusing, sensory awareness, Feldenkrais, Rolfing, the F.M. Alexander Technique, body–mind centering, somatic experiencing, Pesso-Boyden psychotherapy, Rubenfeld synergy, Hakomi, and many others. While each of these techniques involves very sophisticated approaches, the nature and effects of these practices are not easily articulated and, as Don Hanlon Johnson[26] notes, their meanings are not easily captured in the dominant intellectual categories. The closest integration of mainstream science and body-oriented therapies occurred when Nico Tinbergen devoted his 1973 Nobel Prize speech to the Alexander technique.

IMMOBILIZATION VERSUS TAKING ACTION

The notion that sensory triggers reinstate hormonal and motoric responses relevant to the original trauma raises important clinical issues: one of the most critical factors that renders a situation traumatic is the experience of physical helplessness—the realization that no action can be taken to stave off the inevitable. Trauma can be conceptualized as stemming from a failure of the natural physiological activation and hormonal secretions to organize an effective response to threat. Rather than producing a successful fight or flight

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response the organism becomes immobilized. Probably the best animal model for this phenomenon is that of 'inescapable shock," in which creatures are tortured without being unable to *do anything* to affect the outcome of events.[27,28] The resulting failure to fight or escape, that is, the physical immobilization, becomes a conditioned behavioral response.

Joseph LeDoux and his colleagues have demonstrated that the lateral nucleus of the amygdala is the critical anatomical structure in the formation of conditioned fear memories. This structure, in turn, communicates with the central nucleus of the amygdala, which distributes its output to brainstem areas that control the response of the autonomic nervous system (ANS), while connections with the periaqueductal gray region control freezing or immobility, and connections with the paraventricular hypothalamus control endocrine responses of the hypothalamic-pituitary-adrenal (HPA) axis. LeDoux and his colleagues showed that animals that respond actively to the threat thereby divert the flow of information from the lateral amygdala to the motor circuits of the striatum for active coping, preventing the establishment of conditioned endocrine and behavioral responses.[29] Interestingly, decreased activation of the corpus striatum has been found in several neuroimaging studies of PTSD.[3,4]

LeDoux and his colleagues showed that, in rats, it is possible to redirect the fear conditioned pathway that is responsible for initiating autonomic and endocrine reactions and behavioral immobilization. When rats are given the option of physically escaping from the stimulus they lose their conditioning, even after a conditioned fear response is well established. This work suggests that action diverts the flow of information from the lateral nucleus of the amygdala away from the central nucleus to the basal nucleus of the amygdala, which, in turn projects on motor circuits in the ventral striatum. LeDoux and Gorman state: "By engaging these alternative pathways, passive fear responding is replaced with an active coping strategy. This diversion of information flows away from the central nucleus to the basal nucleus, and the learning that takes place, does not occur if the rat remains passive. It requires that the rat take action. It is "learning by doing," a process in which the success in terminating the conditioned stimulus reinforces the action taken."[30]

Most traumas occur in the context of interpersonal relationships, which involve boundary violations, loss of autonomous action, and loss of self-regulation. When people lack sources of support and sustenance, such as is common with abused children, women trapped in domestic violence, and incarcerated men, they are likely to learn to respond to abuse and threat with mechanistic compliance or resigned submission. Particularly if the brutalization has been repetitive and unrelenting, they are vulnerable to continue to become physiologically dysregulated and go into states of extreme hypo- and hyperarousal, accompanied by physical immobilization. Often, these responses become habitual, and, as a result, many victims develop chronic problems initiating effective, independent action, even in situations where, rationally, they could be expected to be able to stand up for themselves and take care of things.

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In our clinic and laboratory we have taken the findings from neuroscience about the rerouting of conditioned responses by taking effective action very seriously. Neuroscience research provides the theoretical underpinning of our work with action-oriented programs with traumatized adolescents and adults, involving improvisational theater,[31] "model mugging" (in which women who have been raped are taught self-defense and learn to actively fight of a simulated attack by a potential rapist), and other interventions that involve physical action.[32]

AROUSAL MODULATION AND CONTROL OF THE ANS

Describing traumatic experiences in conventional verbal therapy is likely to activate implicit memories, that is, trauma-related physical sensations and physiological hyper- or hypoarousal, which evoke emotions, such as helplessness, fear, shame, and rage. When this occurs trauma victims are prone to feeling that it is still not safe to deal with the trauma and, instead, are likely to seek a supportive relationship in which the therapist becomes a refuge from a life self-experience of anxiety and ineffectiveness. Learning to modulate one's arousal level is essential for overcoming the resulting passivity and dependency.

Damasio draws attention to the fact that: "*It makes good housekeeping sense that* [the brain] *structures governing attention and structures processing emotion should be in the vicinity of one another. Moreover, it also makes good housekeeping sense that all of these structures should be in the vicinity of those which regulate and signal body state. This is because the consequences of having emotion and attention are entirely related to the fundamental business of managing life within the organism, while, on the other hand, it is not possible to manage life and maintain homeostatic balance without data on the current state of the organism's body proper.*"[5]

The role of the ANS in PTSD has been well studied: threat activates the sympathetic and parasympathetic nervous systems. Exposure to extreme threat, particularly early in life, combined with a lack of adequate caregiving responses significantly affect the long-term capacity of the human organism to modulate the response of the sympathetic and parasympathetic nervous systems in response to subsequent stress.[8] The sympathetic nervous system (SNS) is primarily geared to mobilization by preparing the body for action by increasing cardiac output, stimulating sweat glands, and by inhibiting the gastrointestinal tract. Since the SNS has long been associated with emotion, a great deal of work on the role of the SNS has been collected to identify autonomic "signatures" of specific affective states. Overall, increased adrenergic activity is found in about two-thirds of traumatized children and adults.[33–35] Q2

The parasympathetic branch of the ANS not only influences HR independently of the sympathetic branch, but makes a greater contribution to HR, including resting HR.[36–38] Vagal fibers originating in the brainstem affect

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emotional and behavioral responses to stress by inhibiting sympathetic influence to the sinoatrial node and promoting rapid decreases in metabolic output that enable almost instantaneous shifts in behavioral state.[8,38,39] The parasympathetic system consists of two branches: the ventral vagal complex (VVC) and the dorsal vagal complex (DVC) systems. The DVC is primarily associated with digestive, taste, and hypoxic responses in mammals. The DVC contributes to pathophysiological conditions including the formation of ulcers via excess gastric secretion and colitis. In contrast, the VVC has the primary control of supradiaphragmatic visceral organs including the larynx, pharynx, bronchi, esophagus, and heart.[36,40]

The VVC inhibits the mobilization of the SNS, enabling rapid engagement and disengagement in the environment.[41] Deficient vagal modulatory capacity has been well documented in traumatized boys and in school children with internalizing problems.[42,43] Lack of ventral vagal modulation is likely to contribute to the problems that affect regulation and lack of responsiveness to interpersonal comfort in traumatized individuals.

Power spectral analysis (PSA) of heart rate variability (HRV) provides the best available means of measuring the interaction of sympathetic and parasympathetic tone, that is, of brainstem regulatory integrity.[44] Low HRV has been associated with anxiety and depression,[45–48] with coronary vascular disease, and increased mortality,[49] while high HRV is associated with positive emotions[50] and resistance to stress.[8]

PTSD involves a fundamental dysregulation of arousal modulation at the brain stem level. PTSD patients suffer from baseline autonomic hyperarousal and lower resting HRV compared to controls, suggesting that they have increased sympathetic and decreased parasympathetic tone.[51] When presented with mental challenges, such as arithmetic tasks people with PTSD show more arousal and less vagal control over their heart rate.[52]

Abnormally high baseline HR can result from high tonic sympathetic activity, low tonic parasympathetic activity, or both.[53] While pharmacological control over sympathetic arousal in PTSD has been fairly well studied (see Pitman *et al.*, this volume; Saxe *et al.*, this volume) the parasympathetic branch of the ANS has an independent and greater influence on basal HR than the sympathetic branch, and has been specifically implicated in cardiovascular risk factors, disease processes, and outcomes. In a recent study we found strong inverse relationships between heart rate and HRV in individuals with PTSD. A substantial proportion of PTSD patients did not have elevated basal HRs. In patients with elevated HR, there clearly was a parasympathetic contribution independent of the SNS, which supports the notion that poor vagal tone may play a significant role in PTSD.[54]

It seems that, in order to come to terms with the past it may be essential to learn to regulate one's physiological arousal. Currently, little is known about how people can learn to do that, even though a number of techniques claim to be able to help people control their HRV. However, no study has been published

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to date to show how changing HRV affects PTSD symptomatology. Since lack of arousal modulation is such a dominant issue in traumatized individuals we decided to systematically study whether (1) there might be an effective way of increasing HRV and (2) whether increased HRV would be associated with improvement in PTSD symptomatology. Since yoga is a very common practice for self-care in our culture, and since numerous yoga websites claim that yoga can change HRV, we decided if we could verify that claim (we could find no studies to support the notion that yoga, in fact, changes HRV).[1]

In order to test the proposition that yoga can change HRV we built a custom version of the MEDAC System/3 (NeuroDyne Medical Corporation, Cambridge, MA, USA) that allowed eight subjects to be monitored simultaneously for HRV. Data were sampled at 250 samples per sec and the interbeat interval (IBI) determined. Normalization of IBI values was carried out using the standard algorithms in the Log-a-Rhythm HRV analysis software, version 3.0 (Nian-Crae, Inc., Cambridge, MA, USA). The normal control yoga group ($N = 11$) significantly changed HRV over eight sessions of hatha yoga: paired samples t-tests were conducted to examine the effects of yoga on HRV. There was a mean improvement in SDNN of 12.8, ($SD = 16.8$; $t(8) = 2.287$; $P \leq 0.05$). Yoga significantly improved PTSD symptomatology, as measured by the CAPS: total pre–post yoga $t(10) = 4.052$; $P \leq 0.01$; CAPS reexperiencing pre–post yoga $t(10) = 0.5.164$; $P \leq 0.001$ and CAPS avoidance pre–post yoga $t(10) = 2.620$; $P \leq 0.01$. Hyperarousal was nonsignificant in the t-tests)When measuring HRV from session to session the yoga exhibited a large number of movement artefacts during the yoga relaxation phase (Shavasana) throughout the active treatment phase, as well as significant peripheral vasoconstriction, which interfered with getting accurate readings of HRV in this group. This suggests that the PTSD group had muscular and vascular concomitants of PTSD that interfered with the measurement of HRV peripherally.

In another pilot study eight female patients between the ages of 25 and 55 years with PTSD were randomly assigned to eight sessions of group therapy based on DBT or to 75 min of simple hatha yoga exercises, and rated by rating on the following outcome measures: Davidson PTSD Scale, the PANAS, and Trauma Center Body Awareness Scale. Samples t-tests were conducted to examine the effects of yoga and DBT on various symptoms of PTSD. In comparison with DBT only the yoga group showed significant decreases in frequency of intrusions and severity of hyperarousal symptoms between time1 and time 2 ($t(6) = 3.44$; $P < 0.05$; $t(6) = 3.2$; $P < 0.05$, respectively).There are no significant increases or decreases in these-–these numbers mean that they are related pre–post, but say nothing about significant increase and decrease-–the t-tests reveal no significant changes for PANAS or body awareness

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The subjective reports of the yoga PTSD group were intriguing; members of the group made statements, such as: "I have always hated my body and I learned how to take care of it," "Having grown up obese and self-conscious it was wonderful to be able to move gently," "I learned to be able to focus and sense where my body was," "I was able to go shopping and know what I needed," and "I learned for the first time how to focus."

MINDFULNESS AND INTEROCEPTION

One of the most robust findings of the neuroimaging studies of traumatized people is that, under stress, the higher brain areas involved in "executive functioning": planning for the future, anticipating the consequences of one's actions, and inhibiting inappropriate responses, become less active. Specifically, neuroimaging studies of people with PTSD have found decreased activation of the medial prefrontal cortex (mPFC).[55,56] The medial prefrontal comprises anterior cingulate cortex (ACC) and medial parts of the orbitofrontal prefrontal cortices.[57] (See chapter 20 in this volume; see also Ref. 58.) The anterior cingulate (ACC) specifically has consistently been implicated in PTSD (see chapters xx, xx, this volume.). The ACC plays a role in the experiential as- Q3
pects of emotion, as well as in the integration of emotion and cognition. It has extensive connections with multiple brain structures, including the hypothalamus, amygdala, and brain stem autonomic nuclei. Thus, the ACC is part of a system that orchestrates the autonomic, neuroendocrine, and behavioral expression of emotion and may play a key role in the visceral aspects of emotion.[58] The mPFC plays a role in the extinction of conditioned fear responses by exerting inhibitory influences over the limbic system, thereby regulating the generalization of fearful behavior,[59] by attenuating peripheral sympathetic and hormonal responses to stress,[57,60,61] and in the regulation of the stress hormone cortisol by suppressing the stress response mediated by the HPA axis.[63] Hence, dysfunction of the mPFC is likely to contribute to the arousal dysregulation in PTSD.[22] The fact that the mPFC can directly influence emotional arousal has enormous clinical significance, since it suggests that activation of interoceptive awareness can enhance control over emotions.

Clinical experience shows that traumatized individuals, as a rule, have great difficulty attending to their inner sensations and perceptions—when asked to focus on internal sensations they tend to feel overwhelmed, or deny having an inner sense of themselves. When they try to meditate they often report becoming overwhelmed by being confronted with residues of trauma-related perceptions, sensations, and emotions:[63] they report of feeling disgusted with themselves, helpless, panicked, or experiencing trauma-related images and physical sensations. Trauma victims tend to have a negative body image—as far as they are concerned, the less attention they pay to their bodies, and

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thereby, their internal sensations, the better. Yet, one cannot learn to take care of oneself without being in touch with the demands and requirements of one's physical self. In the field of trauma treatment a consensus is emerging that, in order to keep old trauma from intruding into current experience, patients need to deal with the internal residues of the past. Neurobiologically speaking: they need to activate their mPFC, insula, and anterior cingulate by learning to tolerate orienting and focusing their attention on their *internal* experience, while interweaving and conjoining cognitive, emotional, and sensorimotor elements of their traumatic experience.

Sarah Lazar and colleagues at the Massachusetts General Hospital recently completed an fMRI imaging study of 20 people engaged in meditation involving sustained mindful attention to internal and external sensory stimuli and nonjudgmental awareness of present-moment stimuli without cognitive elaboration.[64] They found that brain regions associated with attention, interoception, and sensory processing were thicker in meditation participants than matched controls, including the prefrontal cortex and right anterior insula. The largest between-group difference was in the thickness of right anterior insula. It has been proposed that by becoming increasingly more aware of sensory stimuli during formal practice, meditation practitioners gradually increase their capacity to navigate potentially stressful encounters that arise throughout the day. Lazar concludes that this Eastern philosophy of emotion is in line with Damasio's theory that connections between sensory cortices and emotion cortices play a crucial role in adaptive decision making.

Lazar's study lends support to the notion that treatment of traumatic stress may need to include becoming mindful: that is, learning to become a careful observer of the ebb and flow of internal experience, and noticing whatever thoughts, feelings, body sensations, and impulses emerge. In order to deal with the past, it is helpful for traumatized people to learn to activate their capacity for introspection and develop a deep curiosity about their *internal* experience. This is necessary in order to identify their physical sensations and to translate their emotions and sensations into communicable language—understandable, most of all, to themselves.

Traumatized individuals need to learn that it is safe to have feelings and sensations. If they learn to attend to inner experience they will become aware that bodily experience never remains static. Unlike at the moment of a trauma, when everything seems to freeze in time, physical sensations and emotions are in a constant state of flux. They need to learn to tell the difference between a sensation and an emotion (How do you know you are angry/afraid? Where do you feel that in your body? Do you notice any impulses in your body to move in some way right now?). Once they realize that their internal sensations continuously shift and change, particularly if they learn to develop a certain degree of control over their physiological states by breathing, and movement, they will viscerally discover that remembering the past does not inevitably result in overwhelming emotions.

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After having been traumatized people often lose the effective use of fight or flight defenses and respond to perceived threat with immobilization. Attention to inner experience can help them to reorient themselves to the present by learning to attend to nontraumatic stimuli. This can open them up to attending to new, nontraumatic experiences and learning from them, rather than reliving the past over and over again, without modification by subsequent information. Once they learn to reorient themselves to the present they can experiment with reactivating their lost capacities to physically defend and protect themselves.

CONCLUSION

Interoceptive, body-oriented therapies can directly confront a core clinical issue in PTSD: traumatized individuals are prone to experience the present with physical sensations and emotions associated with the past. This, in turn, informs how they react to events in the present. For therapy to be effective it might be useful to focus on the patient's physical self-experience and increase their self-awareness, rather than focusing exclusively on the *meaning* that people make of their experience—their narrative of the past. If past experience is embodied in current physiological states and action tendencies and the trauma is reenacted in breath, gestures, sensory perceptions, movement, emotion and thought, therapy may be most effective if it facilitates self-awareness and self-regulation. Once patients become aware of their sensations and action tendencies they can set about discovering new ways of orienting themselves to their surroundings and exploring novel ways of engaging with potential sources of mastery and pleasure.

Working with traumatized individuals entails several major obstacles. One is that, while human contact and attunement are cardinal elements of physiological self-regulation, interpersonal trauma often results in a fear of intimacy. The promise of closeness and attunement for many traumatized individuals automatically evokes implicit memories of hurt, betrayal, and abandonment. As a result, feeling seen and understood, which ordinarily helps people to feel a greater sense of calm and in control, may precipitate a reliving of the trauma in individuals who have been victimized in intimate relationships. This means that, as trust is established it is critical to help create a *physical* sense of control by working on the establishment of physical boundaries, exploring ways of regulating physiological arousal, in which using breath and body movement can be extremely useful, and focusing on regaining a physical sense of being able to defend and protect oneself. It is particularly useful to explore previous experiences of safety and competency and to activate memories of what it feels like to experience pleasure, enjoyment, focus, power, and effectiveness, before activating trauma-related sensations and emotions. Working with trauma is as much about remembering how one survived as it is about what is broken.

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REFERENCES

1. RAUCH, S., B.A. VAN DER KOLK, R. FISLER, *et al*. 1996. A symptom provocation study of posttraumatic stress disorder imagery in Vietnam combat veterans. Arch. Gen. Psychiatry 970–975. Q4
2. HULL, A.M. 2002. Neuroimaging findings in post-traumatic stress disorder. Br. J. Psychiatry **181:** 102–110.
3. LANIUS, R.A., P.C. WILLIAMSON, M. DENSMORE, *et al*. 2001. Neural correlates of traumatic memories in posttraumatic stress disorder: a functional MRY investigation. Am. J. Psychiatry **158:** 1920–1922.
4. LINDAUER, R.J., J. BOOJI, J.B.A. HABRAKEN, *et al*. 2004. Cerebral blood flow changes during script driven imagery in police officers with posttraumatic stress disorder. Biol. Psychiatry **56:** 853–861.
5. DAMASIO, A.R. 1999. The Feeling of what Happens: body and Emotion in the Making on Consciousness. Harcourt Brace. New York.
6. LEDOUX, J.E., L. ROMANSKI & A. XAGORARIS. 1991. Indelibility of subcortical emotional memories. J. Cog. Neurosci. **1:** 238–243.
7. PANKSEPP, J. 1998. Affective Neuroscience—The Foundations of Human and Animal Emotions. Oxford University Press. New York.
8. PORGES, S., J.A. DOUSSARD-ROOSEVELT, A.L. PORTALES, *et al*. 1996. Infant regulation of the vagal brake predicts child behavior problems: a psychobiological model of social behavior. Dev. Psychobiol. **29:** 697–712.
9. LLINÁS, R. 2001. I of the Vortex: From Neurons to Self. MIT Press. Cambridge, MA.
10. DAVIDSON, R.J., J.S. MAXWELL, & A.J. SHACKMAN. 2004. The privileged status of emotion in the brain. PNAS **101:** 11915–11916.
11. JACKSON, J.H. 1958. Evolution and dissolution of the nervous system. *In* Selected Writings of John Hughlings. J.J. Taylor, Ed.: 45–118. Stapes Press. London.
12. MACLEAN, P.D. 1990. The Triune Brain in Evolution: Role in Paleocerebral Functions. Plenum Press. NewYork.
13. DAMASIO, A.R., T.J. GRABOWSKI, A. BECHARA, *et al*. 2000. Subcortical and cortical brain activity during the feeling of self-generated emotions. Nat. Neurosci. **3:** 1049–1056.
14. DARWIN, C. 1872. The Expression of Emotions in Man and Animals. John Murray. London.
15. PAVLOV, I.P. 1928. Lectures on Conditioned Reflexes. Trans. W.H. Gantt. Liveright. New York.
16. JAMES, W. 1890. The Principles of Psychology, Vol. 1. Dover Publishing. London.
17. SPERRY, R.W. 1952. Neurology and the mind-brain problem. Am. Scientist **40:** 291–312.
18. BULL, N. 1951. The Attitude Theory of Emotion. Nervous and Mental Disease Monographs. New York.
19. JANET, P. 1889. L'Automatisme Psychologique. Félix Alcan. Paris.
20. KARDINER, A. 1941. The Traumatic Neuroses of War. Hoeber. New York.
21. JANET, P. 1919. Les Médications Psychologiques, 3 Vols. Félix Alcan. Paris.
22. VASTERLING, J., K. BRAILEY, N. CONSTANS, *et al*. 1998. Attention and memory dysfunction in posttraumatic stress disorder. Neuropsychology **12:** 121–133.
23. CLARK, R.C., A.C. MCFARLANE, P.L.P. MORRIS, *et al*. 2003. Cerebral function in posttraumatic stress disorder during verbal working memory upgrading: a positron emission tomography study. Biol. Psychiatry **53:** 474–481.

UNCORRECTED PROOF

24. KRYSTAL, H. 1988. Integration and Self-Healing. The Analytic Press. Hillsdale, NJ.
25. LINEHAN, M.M., H.E. ARMSTRONG, A. SUAREZ, *et al*. 1991. Cognitive-behavioural treatment of chronically parasuicidal borderline patients. Arch. Gen. Psychiatry **48:** 1060–1064.
26. HANLON – JOHNSON, D. & I.J. GRAND, Eds. : 1998. The Body in Psychotherapy: Inquiries in Somatic Psychology. North Atlantic Books. Berkeley, CA.
27. MAIER, S.F. & M.E.P. SELIGMAN. 1976. Learned helplessness: theory and evidence. J. Exp. Psychol. Gen. **105:** 3–46.
28. VAN DER KOLK, B.A., M. GREENBERG, H. BOYD, *et al*. 1985. Inescapable shock, neurotransmitters, and addiction to trauma: toward a psychobiology of posttraumatic stress disorder. Biol. Psychiatry **20:** 314–325.
29. AMORAPANTH, P., J.E. LEDOUX & K. NADER. 2000. Different lateral amygdala outputs mediate reaction and actions elicited by a fear-arousing stimulus. Nat. Neurosci. **3:** 74–49.
30. LEDOUX, J.E. & J.M. GORMAN. 2001. A call to action: overcoming anxiety through active coping. Am. J. Psychiatry 1953–1955. **Q5**
31. KISIEL, C., M. BLAUSTEIN, J. SPINAZZOLA, *et al*. 2005. Evaluation of a theater based youth violence prevention program for elementary school children. J. School Viol. **5:** 19–36.
32. MACY, R., L. BEHAR, R. PAULSON, *et al*. 2004. Community-based, acute post-traumatic stress management: a description and evaluation of a psychosocial–intervention continuum. Harv. Rev. Psychiatry **12:** 217–228.
33. PITMAN, R. 1989. Post-traumatic stress disorder, hormones, and memory. Biol. Psychiatry **26:** 221–223.
34. DE BELLIS, M.D., M.S. KESHAVAN, D.B. CLARK, *et al*. 1999. Developmental traumatology part II. brain development. Biol. Psychiatry **45:** 1271–1284.
35. DEBELLIS, M., L. LEFTER, P. TRICKETT, *et al*. 1994. Urinary catecholamine excretion in sexually-abused girls. J. Am. Acad. Child Adolesc. Psychiatry **33:** 320–327.
36. PORGES, S. 1991. Vagal tone: an autonomic mediator of affect. *In* The Development of Affect Regulation and Dysregulation. J. Garber & K.A. Dodge, Eds.: 111–128. Cambridge University Press. New York.
37. PORGES, S.W., J.A. DOUSSARD-ROOSEVELT & A.K. MAITI. 1994. Vagal tone and the physiological regulation of emotion. *In* Emotion Regulation: Behavioral and Biological Considerations. N.A. Fox, Ed.: 59 (2-3, Serial No. 240), 167–186. Monograph of the Society for Research in Child Development.
38. PORGES, S. & J. DOUSSARD-ROOSEVELT. 1997. The psychophysiology of temperament. *In* The Handbook of Child and Adolescent Psychiatry. J.D. Noshpitz, Ed.: 250–268. Wiley Press. New York.
39. PORGES, S., J. DOUSSARD-ROOSEVELT, A. PORTALES, *et al*. 1994. Cardiac vagal tone: stability and relation to difficultness in infants and three-year-old children. Dev. Psychobiol. **27:** 289–300.
40. PORGES, S. 1995. Orienting in a defensive world: mammalian modifications of our evolutionary heritage. A Polyvagal theory. Psychophysiology **32:** 301–318.
41. MEZZACAPPA, E., R. KELSEY, E. KATKIN, *et al*. 2001. Vagal rebound and recovery from psychological stress. Psychosom. Med. **63:** 650–657.
42. MEZZACAPPA, E., A. EARLS & D. KINDLON. 1998. Executive and motivational control of performance task behavior, and autonomic heart-rate regulation in children: physiologic validation of two-factor solution inhibitory control. J. Child. Psychol. Psychiatry **39:** 525–531.

UNCORRECTED PROOF

43. SEGUIN, J., R. PIHL, P. HARDEN, *et al*. 1995. Cognitive and neuropsychological characteristics of physically aggressive boys. J. Abnorm. Psychol. **104:** 614–624.
44. PORGES, S. 1991. Vagal tone: an autonomic mediator of affect. *In* The Development of Affect Regulation and Dysregulation. Garber & Dodge, Eds.: 111–128. Cambridge University Press. New York.
45. CARNEY, R.M., M.W. RICH, K.E. FREEDLAND, *et al*. 1988. Major depressive disorder predicts cardiac events in patients with coronary artery disease. Psychosom. Med. **50:** 627–633.
46. MCCRATY, R., M. ATKINSON, D. TOMASINO, *et al*. 2001. Analysis of twenty-four hour heart rate variability in patients with panic disorder. Biol. Psychol. **56:** 131–150.
47. RECHLIN, T. 1994. Are affective disorders associated with alterations of heart rate variability? J. Affect. Disord. **32:** 271–275.
48. SHIBAGAKI, M. & T. FURUYA. 1997. Baseline respiratory sinus arrhythmia and heart-rate responses during auditory stimulation of children with attention-deficit hyperactivity disorder. Percept. Mot. Skills **84:** 967–975.
49. DEKKER, J.M., E.G. SCHOUTEN, P. KLOOTWIJK, *et al*. 1997. Heart rate variability from aged and elderly men. Am. J. Epidemiol. **145:** 899–908.
50. MCCRATY, R., M. ATKINSON, W.A. TILLER, *et al*. 1995. The effects of emotions on short-term power spectrum analysis of heart rate variability. Am. J. Card. **76:** 1089–1093.
51. COHEN, J., J. PEREL, M. DEBELLIS, *et al*. 2002. Treating traumatized children: clinical implications of the psychobiology of posttraumatic stress disorder. Trauma Viol. Abuse **3:** 91–108.
52. SAHAR, T., A. SHALEV & S. PORGES. 2001. Vagal modulation of responses to mental challenge in posttraumtic stress disorder. Biol. Psychiatry **49:** 637–643.
53. BERNSTON, G.G., J.T. BIGGER JR., D.L. ECKBERG, *et al*. 1997. Heart rate variability: origins, methods, and interpretive caveats. Psychophysiology **34:** 623–648.
54. HOPPER, J.W., J. SPINAZZOLA, W.B. SIMPSON, *et al*. 2005. Preliminary evidence of parasympathetic influence on basal heart rate in posttraumatic stress disorder. J. Psychosom. Res. In press. **Q6**
55. MARKOWITSCH, H.J., J. KESSLER, G. WEBER-LUXENBURGER, *et al*. 2000. Neuroimaging and behavioral correlates of recovery from mnestic block syndrome and other cognitive deteriorations. Neuropsychiatry, Neuropsychol. Behav. Neurol. **13:** 60–66.
56. SHIN, L.M., P.J. WHALEN, R.K. PITMAN, *et al*. 2001. An MRI study of anterior cingulated function in posttraumatic stress disorder. Biol. Psychiatry **50:** 932–942.
57. DEVINSKY, O., M. MORRELL & B.A. VOGT. 1995. Contributions of anterior cingulated to behavior. Brain **118:** 279–306.
58. LANIUS, R., R. BLUHM, U. LANIUS, *et al*. 2005. A review of neuroimaging studies of hyperarousal and dissociation in PTSD: heterogeneity of response to symptom provocation. J. Psychiatr. Res. In press.
59. MORGAN, M.A., L.M. ROMANSKI & J.E. LEDOUX. 1993. Extinction of emotional learning: contribution of medial prefrontal cortex. Neurosci. Lett. **163:** 109–113.
60. LEDOUX, J.E. 2000. Emotion circuits in the brain. Ann. Rev. Neurol. **23:** 155–184.
61. MILAD, M.R. & G.J.L. QUIRK. 2001. Neurons in medial prefrontal cortex signal memory for fear extinction. Nature **420:** 70–74.

UNCORRECTED PROOF

62. LANE, R.D. & K. MCRAE. 2004. Neural substrates of conscious emotional experience: a cognitive-neuroscientific perspective. *In* Consciousness, Emotional Self-Regulation and the Brain. M. Beauregard, Ed.: 87–122. John Benjamins Publishing. Philadelphia. Q7
63. YEHUDA, R. 2000. Biology of posttraumatic stress disorder. J. Clin. Psychiatry **61:** 14–21.
64. LAZAR, S.W., C.E. KERR, R.H. WASSERMAN, *et al*. 2005. Meditation experience is associated with increased cortical thickness. Neuroreport **16:** 1893–1897.

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Queries

Q1 Author: Please confirm affiliation.

Q2 Author: Please confirm if the edit in the sentence—"Since. . .states" retains the intended sense.

Q3 Author: Please provide the volume and chapter numbers.

Q4 Author: Please provide volume number for reference 1

Q5 Author: Please provide volume number for reference 30.

Q6 Author: Please provide volume number and full page range for reference 54.

Q7 Author: reference 62 is not cited in the text. Please check.