Synthesizing Psychoanalytic and Biological Approaches to Trauma: Some Theoretical Proposals

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This paper offers some theoretical possibilities for synthesizing psychoanalytic and neurobiological approaches to understanding the effects of severe psychic trauma. The paper argues that biological and psychoanalytic perspectives can enrich and enhance each other in our attempt to understand and ameliorate the damaging consequences of trauma. The findings of biological researchers and the observations of psychoanalysts are integrated, as they apply to two functions that are damaged by trauma: the capacity for representation and the capacity for self-regulation. These capacities are interdependent and interactive. Disrupting either will affect the other. The combined result of this disruption can profoundly alter subjective experience. The paper discusses how helping patients to symbolize traumatic experiences, affect, and other mental states and helping patients regulate physiology and affect through nonverbal, affect-regulating interactions, and through the use of medication, lead to an improved capacity to symbolize, to experience meaning, and to relate and can lead to a subjective sense of increased strength.

In this paper I propose some theoretical possibilities for synthesizing biological and psychoanalytic perspectives to understand the effects of psychic trauma. Neurobiologists observe how trauma alters the structure and function of the brain and nervous system. Psychoanalysts observe the subjective impact of trauma: how traumatic stress alters and even shatters one’s experience of meaning and of self. This speculative inquiry is a first step toward integrating findings from, at least, these objective and subjective points of view and will hopefully enable us to better appreciate our patients’ experience with the devastating impact of trauma in their lives.

I shall divide the damaging effects of traumatic stress into two categories: (1) the capacity for representation, and (2) the capacity for self-regulation. These domains of disturbance are not independent of one another, but are interdependent. By combining findings from neurobiology and psychoanalysis, we can better appreciate the nature of these disruptions and how they interact.

Representation

A core feature of trauma, as discussed in the psychoanalytic literature, is that the mind cannot represent the experience or organize it on a symbolic level. The experience may not be registered in awareness at all, leaving an absence or “black hole” that disrupts subjective awareness (Cohen, 1983; Laub & Auerhahn, 1993; Lifton & Olson, 1976). When the experience does register, it is often in the form of concrete, unintegrated, somatosensory fragments (Laub & Auerhahn, 1993; van der Kolk & Fisler, 1995). There is usually no verbal component, and no story into which the perceptual fragments can be placed. The degree to which the mind represents trauma symbolically is on a continuum (Laub & Auerhahn, 1993), from concrete perceptual fragments in various unintegrated sensory modalities, to forms of perceptual representation that are more coherent but remain rigid, concrete, and meaningless and cannot be used in the service of imagination or play, to symbolic structures that can represent...
multiple meanings. By symbolic I mean an image or verbal symbol that can refer to meanings other than the concrete experience itself.

Case 1

Ms. D was at a restaurant with her young daughter and her mother on the anniversary of her father’s death. This incident was several years before she had any psychotherapeutic treatment, and at the time she had no memory of sexual abuse. Later, memories of incest by her father became increasingly clear and coherent and were verified by an aunt in whom she had confided as a child. She had a long history of somatic symptoms such as hysterical blindness, fainting episodes, and confusing reactions of nausea and disgust when near her father. She also had confusing physical sensations such as a disgusting “sticky” feeling on her face whenever she splashed water on it. During the incident reported here, her mother said something about her father, and Ms. D started to feel enraged, then disoriented. She became flooded with fragmented, incoherent images of his face, tongues, a penis, and patterns of lines and colors. “These awful images and thoughts started, and I was frantically trying to make sense of these images. Then suddenly I felt myself slipping away—that terrified me so much.” It was as if she were going to die or her self would dissolve into a void.

The crude and fragmented images that overtook these patients’ awareness were not symbols of something hidden, like hysterical symptoms. In hysteria the body is used as symbol, to disguise forbidden desire or to contain and express conflict. The experiences reported by Ms. D and Ms. M are different. They are concrete fragments of experience that they were unable to represent or organize on a symbolic level.

The inability to recall trauma, such as occurred for these two women, is usually framed in terms of memory. Framing this inability as a problem of memory has led to a misleading debate over “repressed memories.” I believe that the matter goes beyond memory and relates to the more general problem of representation. Memories (i.e., conscious, verbal, or “episodic” memory) are formed only when experience can be encoded and organized symbolically. With severe trauma, there is a failure to encode and organize the experience on a symbolic level. How can we speak of memories being repressed when they have not yet been formed? Traumatic experiences may be encoded and retained in other forms, but not organized on a symbolic level that would allow for the formation of episodic, conscious memory. And again, there is a continuum in the degree to which and coherence with which experiences are represented and organized symbolically. Fonagy (1999) has also argued that early or traumatic memories that are not represented in consciousness cannot be properly described as repressed. He writes that “repression can only act on events experienced at a developmental stage which allows encoding into autobiographical memory” (p. 219), and that, for traumatized persons, “accurate encoding of events is disrupted” (p. 220). In contrast to my own position, though, Fonagy believes that these experiences cannot be “recalled” or represented on a symbolic level at some later time; they are gone forever. I submit that traumatic experiences can be “recalled,” but what appears to be a lifting of repression is instead a newly formed capacity, in relation to certain experiences, to encode and organize these experiences on a symbolic level. Loewald (1955) presented a similar understanding in his discussion of Freud’s (1893) concept of repressed childhood memories:
It is often clear that in a so-called recovery of a childhood memory we are confronted, not with the recall of something forgotten, but with a creative event in which something for the first time can be put into words. The definite impression is that it had never been put into words, and more, that it never had been capable of being put into words because the original experience had taken place on a level of integration which did not render it available for preconscious or conscious integration. Recollection, as against reminiscence as used by Freud in this paper, is not a re-recording, but the creation of something new. While originating in something old, inscribed into the organism as an unconscious memory trace (body memory), it had not existed in this form before. [p. 41]

There are biological and psychological reasons why overwhelming experiences cannot be encoded or organized as verbal symbols. Psychoanalysts have noted multiple factors, including:

1. The experiences are so immediate and intense, so oppressive, that they overwhelm the perspective and distance necessary to grasp and symbolize an experience (Laub & Auerhahn, 1993).

2. There are no cultural categories or narratives that can organize trauma and give the experience meaning (Laub & Auerhahn, 1993), and trauma shatters basic assumptions about the world and ourselves (Janoff-Bulman, 1992).

3. In the case of child abuse, there is no consensual validation or acknowledgment of the experience within the family, and no help from adults in naming or understanding the experience (Ferenczi, 1932; Freyd, 1996).

4. Because the terror and rage associated with child abuse is so intense, and because the child must hold on to an image of a good parent in order to survive the overwhelming intensity of feeling, the child must not register the abuse and must register the bad parent as good. Shengold (1979, 1989) describes this as a “mind splitting operation” that leads to nonregistration of trauma (see also Ferenczi, 1932).

The net result of all of these factors is that there are no words or linguistic categories to contain, organize, or articulate trauma. Without words or symbols, there is no meaning and no imaginative elaboration.

Biological consequences of extreme stress also undermine the ability to symbolize trauma. Biological factors include the following:

1. Hyperarousal and physiologic dysregulation in general are disorganizing and can interfere with the capacity to symbolize.

2. Patients with posttraumatic stress disorder (PTSD) show general impairments in learning and memory, particularly verbal learning and verbal memory (Bremner, Randall, et al., 1995a; Vasterling, Graily, Constans, & Sutker, 1998; Yehuda, Keefe, et al., 1995).

3. Magnetic resonance imaging (MRI) studies show decreased volume of the hippocampus both in combat veterans and in women who were abused as children (Bremner, Randall, Scott, et al., 1995a; Bremner, Randall, Vermetten, et al., 1997; Gurvits et al., 1996). In veterans, the decrease in hippocampal volume correlates with impairment in verbal memory (Bremner, Randall, Scott, et al., 1995b).

4. Functional brain imaging shows that when PTSD patients are reexperiencing an aspect of their trauma, there is activation of right-sided limbic and paralympic areas and right secondary visual cortex, and deactivation of left-sided middle temporal and inferior frontal cortical areas, including Broca’s area (Rauch et al., 1996; Shin et al., 1997).

The fact that there is deactivation of left-hemisphere cortical structures such as Broca’s area is interesting for at least two reasons. First, the left hemisphere is involved in linguistic processing or coding information into verbal symbols. Second, neuroscientist Michael Gazzaniga (1998a, 1998b, 2000) believes that the left hemisphere serves an “interpreter function,” which is to organize inchoate experience into a meaningful and coherent personal narrative.

Structural damage to the hippocampus has figured prominently in theorizing about the inability to represent and recall trauma (Bremner, Krystal, Southwick, & Charney, 1995). High cortisol levels released during extreme stress are damaging to hippocampal neurons (McEwen, Gould, & Sakai, 1992). The hippocampus is crucial for forming declarative memories (Eichenbaum, 1994; Mesulam, 1998), for ordering an experience in space and time (O’Keefe & Nadel, 1978), and for encoding information as nonverbal and verbal symbols (Bucci, 1997a, 1997b). With damage to the hippocampus, traumatic experiences are encoded and processed as nonverbal, unconscious, “subsymbolic” forms that cannot be placed in space or time.

Impaired functioning of the hippocampus probably plays some role in the inability to encode, retrieve, or contextualize traumatic experiences for many people. But it must be only part of the story. There are several problems with placing a faulty hippocampus at the center of the problem:

1. Structural damage to the hippocampus does not account for how information and experience can
at one time be unavailable for representation and recall and become accessible for symbolization and recall at a later time, such as occurred for Ms. D and Ms. M.

2. In terms of placing an experience in space and time, the hippocampus maps within very brief time intervals. Moscovitch (1994) states that while the hippocampus maps experience in time and space, it is “concerned with time intervals that span only a few seconds, well within the realm of simple temporal contiguity, but far short of the range of our capacity for temporal ordering” (p. 296). Placing experience in a larger spatial and temporal context, such as locating an experience in a personal narrative and assigning meaning to the experience, is a higher, perhaps frontal-lobe, function (Moscovitch, 1994) and probably a function of the linguistic specialization of the left cerebral hemisphere (Gazzaniga, 1998a, 1998b, 2000).

3. Damage to the hippocampus may also fail to account for the subtle degrees of concreteness in how these experiences are registered, or the continuum in the degree to which they cannot be assigned meaning or narrated, as observed by psychoanalysts. Again, this deficit is likely to involve higher cortical functions.

There are two models from cognitive neuroscience that fit the phenomenology of how traumatic experiences are registered and that can incorporate biological and psychological factors that underlie experiences like those of Ms. D and Ms. M. One is a model for how information is processed into symbolic form, which has been articulated by Wilma Bucci (1997a, 1997b), a cognitive scientist interested in integrating cognitive science and psychoanalysis. Neuroscientist Marcel Mesulam (1998) articulates the other model in a paper entitled, “From Sensation to Cognition,” published in the journal Brain. These models complement each other; both have to do with binding and integrating information that is stored in a distributed form throughout the brain.

According to Bucci, cognitive science recognizes symbolic and subsymbolic forms of encoding and processing information. The two systems have different properties. The nonverbal, subsymbolic system, “incorporates representations and processes in all sensory modalities as well as motoric and somatic forms” (Bucci, 1997b, p. 155). Information from different sensory modalities occupies “the same channels as perceptual experience itself” (Bucci, 1997b, p. 155), so that the information is reactivated as crude perceptual traces. Information from different perceptual channels is processed simultaneously and in parallel (parallel-distributed processing), and it is processed continuously as opposed to sequentially. The flow of continuous, uncategorized perceptual information is assembled or “chunked” into functional categories, such as table or tree, and represented as coherent, prototypic images. The hippocampus plays a major role in “chunking” or categorizing continuous perceptual information into coherent, symbolic images (Bucci, 1997a, 1997b; Mesulam, 1998). Nonverbal symbols (images) are processed further into verbal symbols. Verbal symbolic information is processed in a sequential, amodal, single-channel format, as opposed to subsymbolic processing which is in a multimodal (multichannel), parallel-distributed format. Once verbally encoded, information can be assigned meaning and incorporated into narrative structures. This step involves higher cortical functions, including areas involved with linguistic processing.

Mesulam’s (1998) model fills in some neurologic detail on how sensory information that is distributed in modality-specific (unimodal) association cortices is accessed and integrated into multimodal experience and conceptual knowledge or, in Bucci’s terms, encoded in symbolic form. There are several “transmodal nodes” whose function is to bind and integrate information, which is distributed in unimodal association cortices. The hippocampus and entorhinal cortex are crucial hubs that bind distributed information into coherent episodic memory; Wernicke’s and Broca’s areas integrate distributed sense data into language; prefrontal nodes integrate distributed information in working memory and with motivation and emotion. These nodes are not sites where convergent knowledge is stored. Their role is, “predominantly, to create directories (or address codes, maps, look-up tables) for binding distributed, modality-specific fragments into coherent experiences, memories, and thoughts” (Mesulam, 1998, p. 1024).

Memory can be disrupted at the level of the hippocampus or the prefrontal cortex. With lesions of the hippocampal/entorhinal components of the limbic system, “fragments of new and recent events cannot be encoded with the type of coherence necessary for declarative recall” (Mesulam, 1998, p. 1027). Lesions here do not obliterate the entire memory but interfere with coherent binding of the distributed information. Transmodal nodes in the prefrontal cortex participate in memory functions such as “reconstruction of context and temporal order, guidance of encoding and retrieval, associative search for internal data stores, contextual constraints to keep reconstructed memories within the bounds of plausibility . . . ” (Mesulam, 1998, p. 1029). The prefrontal region serves as the transmodal node that looks up and binds information in
working memory, which “would seem to be critical for integrating scenes, events, and internally generated representations into a coherent conscious experience, and for allowing present events to enter into associative interactions with past memories, the present context and contemplated goals. Working memory thus modulates the texture of consciousness” (Mesulam, 1998, p. 1029).

These two models provide somewhat different but complementary possibilities for understanding the fragmented and concrete forms in which traumatic experiences intrude into awareness and for understanding the failure to register these experiences on a symbolic level. Using Bucci’s (1997a, 1997b) model, information that is processed by subsymbolic systems cannot be processed as symbols, perhaps because the hippocampus, which “chunks” sensory information into coherent images, has been disrupted by high cortisol levels. The hippocampal/entorhinal cortex is also the transmodal node that looks up and binds sensory information for declarative memory in Mesulam’s (1998) model. Interfering with transmodal nodes in Wernicke’s and Broca’s area can interfere with the ability to bind sensory information with linguistic symbols. Disrupting transmodal nodes in the prefrontal cortex can interfere with looking up information and binding it in working memory.

What sorts of influences can disrupt the ability of transmodal nodes to look up and bind distributed sensory information? I have mentioned the damaging consequences of cortisol for neurons in the hippocampus. Other physiologic and psychological factors can block sensory information from access to transmodal nodes and thus from multimodal binding and integration. Mesulam (1998) states that the transmodal nodes are “neural bottlenecks,” “in the sense that they constitute regions of maximal vulnerabilities for lesion induced deficits in the pertinent cognitive domain” (p. 1024). These “neural bottlenecks” are subject to “bottom-up” and “top-down” influences. Neurons originating in the brainstem and in the limbic system have a “bottom-up” effect on attention and working memory, so that arousal, motivation, and emotion can influence transmodal nodes in the prefrontal cortex. The prefrontal lobes exert a powerful “top-down” influence and can accentuate or inhibit activity even at the level of unimodal neurons. In this way, motivation, affect, and psychic conflict can inhibit the access of sensory information to transmodal processing by the prefrontal lobes, and presumably by nodes in Wernicke’s and Broca’s areas. One could suppose that these “neural bottlenecks” are vulnerable to becoming overwhelmed or clogged if flooded with information, arousal, and affect, which then temporarily disrupts their capacity to look up and bind information into integrated, multimodal representations in the relevant cognitive domain.

Another question is, where in the brain are the concrete fragments of traumatic experience stored? Bucci (1997a, 1997b) states that subsymbolic sensory information is stored in the same channels as the perceptual experience itself. According to Mesulam (1998), “the information continues to be encoded in the neocortical association cortex in a manner that supports implicit learning” (p. 1027), so that while the information cannot be coherently integrated and recalled, it does continue to influence behavior. Sensory information associated with traumatic experiences is not stored in a different form or place in the brain. The problem in trauma is that when the information is to be activated, the directories or “neural bottlenecks” that look up and bind this distributed information into a multimodal whole are disrupted. Which transmodal node is disrupted determines the cognitive domain that is affected by the inability to look up and integrate sensory information.

Consider Ms. D’s experience in the restaurant and Ms. M’s experience on the bus or when falling asleep. These sensations and fragmented images are in the form of “trace activation” of isolated perceptual channels. The information is in subsymbolic form and has not been integrated into coherent, nonverbal symbols. According to Mesulam’s (1998) model, any one of a number of transmodal nodes or directories may be disrupted in its capacity to look up and bind the information that is distributed in unimodal association cortexes. With time, the sensory fragments become integrated into a coherent image or nonverbal symbol. But they remain cut off from verbal symbols, such as words and stories.

Contrast the sensory fragments and images reported by Ms. D and by Ms. M with the images in a dream reported by Ms. M after several years of psychotherapy.

Case 3

There was a hunting lodge that was finished in dark mahogany. The beams in the lodge came together in a point, which resembled a tetrahedron, as when three walls meet the ceiling in a point at a corner. A flame began and surrounded the room as it spread along the mahogany edge. The flame quickly engulfed the room, reaching the pointed ceiling. She was helpless to stop the fire and feared it would involve the nearby village and its inhabitants. She associated the lodge with a relative whom she met as an adult and disliked intensely. She was currently angry with him over a recent event that demonstrated his self-centeredness. She linked the fire with her
rage. In my own associations I pictured a little girl focusing on the upper corner of a room, and I asked her what the edges and ceiling looked like in the room in which she was abused. They were dark mahogany: she spoke of crouching in the corner of the room, staring at the upper corner while anticipating another sexual assault. We elaborated the visual metaphor in terms of her rage at her relative and the janitor (who had several features in common), her helplessness before her rage, and her fear that it could not be contained.

Ms. M’s rage had become more contained insofar as it could be expressed, and thus contained, by symbols. The images refer to more than one meaning. When traumatic experiences are encoded as crude, somatosensory perceptual fragments, they refer to “no-thing.” More coherent visual images may refer only to the concrete experience itself. The metaphorical properties of Ms. M’s dream, which Ms. M could elaborate and extend and with which we could play together, indicated processing in the form of linguistic symbols.

Laub and Auerhahn (1993) have observed a continuum in the degree to which traumatic experiences are registered and organized on a symbolic level. Forms of traumatic memory range from perceptual fragments, to coherent but concrete perceptual images, to meaningful narratives and symbolic expressions of desire and conflict. This continuum probably reflects a continuum in the degree to which information is processed as nonverbal and then verbal symbols, as opposed to subsymbolic forms of processing. The degree to which traumatic experience is organized and elaborated on a symbolic level depends on which transmodal node can access and bind information distributed in the sensory neocortex. The disruption of nodes in the hippocampal/entorhinal cortex leads to an inability to integrate sensory information into coherent, declarative memory. Disrupting Wernicke’s and Broca’s areas will interfere with integrating this information with verbal forms. Interrupting transmodal nodes in the prefrontal cortex interferes with the ability to look up and bind information in working memory and thus present this information to consciousness as an integrated, textured scene. Mesulam (1998) argues that the prefrontal cortex integrates information in modality-specific association cortexes, which “encode the most veridical representations of experience” (p. 1013) into a more subjective and individually textured representation of the world. So the prefrontal cortex allows for a greater symbolic and subjective elaboration of experience, in contrast to more concrete, blunt representations of traumatic experience. Access of information to transmodal nodes in the prefrontal cortex represents a step up on the continuum in the degree of symbolic organization and elaboration of experience. Both prefrontal nodes and nodes in Wernicke’s and Broca’s area are necessary for elaborating experience into a meaningful, personal narrative.

The continuum on which experiences are encoded and organized as symbols includes nonverbal (images) and verbal symbols. There are advantages to encoding and processing experience as verbal symbols. Language allows the greatest degree of elaboration of the meaning of an experience. When we capture an experience in language, we make possible and convey to others multiple perspectives on that experience (Tomasello, 1999). When processed as linguistic symbols, experiences can be placed in a narrative and assigned greater meaning (van der Kolk & Fisler, 1995); they are more able to refer to multiple meanings. The possibility of multiple perspectives that is conveyed through language and the narration of experience creates a distance or buffer, in contrast to the concrete and blunt immediacy of traumatic experience, which obliterates perspective. Language, according to psychoanalyst Julia Kristeva (1989), is the “ideational and emotional carrier of desire, hatred, conflicts” (p. 36). Language inherently contains the existence of an other subjectivity, as our words either incorporate the subjective perspective of an other, or are meant to influence the perspective of an other (Tomasello, 1999). This contrasts with the absence of an other in the solipsistic, concrete encoding of traumatic sensory fragments and concrete images. Language holds out hope for restoring subjectivity and meaning following their collapse in the face of trauma. The poet Cellan, as quoted by Felman (1995), expresses this: “Within reach, close and not lost, there remained, in the midst of the losses, this one thing: language.”

The problem of how information previously unavailable to memory is later available for representation and recall can now be reframed in terms of the form of representation—that is, whether the information is encoded and processed by subsymbolic or symbolic systems. According to Mesulam’s (1998) model, the form of representation depends on which transmodal nodes are able to look up and bind information that is distributed in the various modality-specific association cortexes. Anything that prevents translating information into the symbolic system, or anything that interferes with the function of one of the transmodal nodes, will interfere with representation, recall, or the capacity to assign meaning to experience. As noted above, there are also psychological, interpersonal, and cultural factors that prevent us from translating trauma into verbal symbols and metaphors.
How is it that sensory information is not accessible at one time and becomes accessible for representation and recall at some later time? According to this model, the information was always there, distributed throughout unimodal association cortices. To quote Mesulam (1998) again, “the information continues to be encoded in the neocortical association cortex in a manner that supports implicit learning” (p. 1027). At some later date the transmodal node or “neural bottleneck” may recover its capacity (perhaps the “neural bottleneck” becomes unclogged) to look up and bind the information for representation in the relevant cognitive domain, be it declarative memory, language, or working memory.

There are different levels, then, at which information can be excluded from integrated conscious awareness. On one level, information has not been encoded in symbolic form and remains in subsymbolic form. This is often the mechanism for the inability to represent trauma. This may occur when the transmodal nodes in the hippocampal/entorhinal cortex or in Wernicke’s and Broca’s areas are disrupted from accessing and binding sensory experience. At another level, information that has been encoded as verbal symbols may be blocked from conscious awareness due to conflict or painful affect. This information is represented in fairly coherent form, though inhibited in degree of imaginative elaboration and integration. At this level, it is often the meaning of the experience that is disavowed or disguised. This mechanism may involve executive control by the dorsolateral prefrontal cortex, as demonstrated in an experiment on the suppression of unwanted memories published in the journal Nature by Anderson and Green (2001). Such a mechanism would be consistent with inhibiting the activity of transmodal nodes in the prefrontal cortex that then inhibits access of sensory information to working memory and inhibits the degree of associative elaboration of an experience. An example of this latter form of disavowal is illustrated by the experience of Mr. F.

Case 4

An uncle sexually molested Mr. F while on a vacation when he was 7 years old. He had retained the experience in his conscious awareness for three years, accompanied by frightening feelings of rage toward the uncle. He now recalls the moment he seemingly willfully split the experience from conscious access. He was in bed when his parents received a call from the police, who were looking for the uncle in response to an accusation of child molestation by another boy. He felt terrified listening to the resulting panic and emotional commotion. At that moment, he “decided” to push the experience out of his mind. He went on for many years experiencing strong emotional reactions—rage, disgust, avoidance—toward his uncle, and he experienced severe inhibitions in his sexual life as an adult, but none of this did he consciously connect with the molestation by his uncle. When the uncle died, Mr. F, by then in his thirties, helped another relative clean out the uncle’s apartment. They discovered a cache of child pornography. Mr. F was shaken and as he was driving home, “it all came back,” in its entirety. He revealed the molestation to his family, whereupon two male cousins confided that this uncle had also molested them.

Mr. F had clearly encoded and processed the experience of sexual molestation on a symbolic level, in fairly coherent form. He retained clear episodic memory of the experience until his conflict was made intolerable by his uncle’s arrest. He then disavowed the entire experience. Exploration in psychotherapy confirmed this to have been a classical hysterical defense, consistent with his use of hysterical defenses in response to other conflicts. The contrast between Mr. F versus Ms. M and Ms. D represents opposite extremes on a continuum. Clinically, one is more likely to encounter a mixture in the degree to which there is a failure to symbolize versus the degree to which symbolically encoded experiences are disguised or otherwise disavowed in the service of defense.

The distinction between the different forms of encoding information parallels similar distinctions in the psychoanalytic literature. Loewald used extensively the idea that information at different levels is represented and organized in different forms in his explications of the concepts of repression and recollection (1955), memory (1976), trauma and the repetition-compulsion (1973), primary and secondary process, and language (1978). Loewald’s discussions included the idea that information is organized at one level in the form of somatic-motor-perceptual traces and on another level in linguistic form, and that the mind translates experience from the first level of representation and organization into the second.

Killingmo (1989) distinguishes deficit from conflict. Deficit is a failure to generate or represent meaning, while conflict reflects a disguising of meaning. The task in conflict pathology is to uncover and interpret meaning, while the task in deficit pathology is to construct or represent meaning. The notion of deficit parallels subsymbolic processes that are prevented access to symbolic forms of encoding and processing. Conflict pathology, on the other hand, reflects inhibitory processes, perhaps executed by the dorsolateral prefrontal cortex.
(Anderson & Green, 2001), in which verbally encoded information is inhibited from conscious access. Again, most people have both mechanisms at work, perhaps one predominating in a particular area of experience or at a certain time.

Fonagy et al. (1993) make a similar distinction between psychopathology based on distorted representations versus a more pervasive and primitive disturbance in mental processes. In the latter form of disturbance, entire forms of thinking or mental processes or capacities, such as the capacity to form representations, are inhibited and ill developed. These authors avoid the notion of deficit and see the massive inhibition and maldevelopment of mental processes or capacities as caused by the need to avoid intolerable affect and danger. I would submit that the inability to “recall” or represent trauma is one such form of inhibited or disrupted mental capacity or process. Inhibiting or disrupting the different transmodal nodes or “neural bottlenecks” will inhibit the capacity to bind and integrate distributed sensory data into coherent representations in different cognitive domains, including memory. Again, there are a number of physiological and psychological factors that can inhibit or disrupt the capacity of these vulnerable “neural bottlenecks” to look up, bind, and integrate distributed sensory information, including but not limited to avoiding painful affect. Fonagy et al. (1993) believe that inhibited mental processes or capacities can be reactivated in psychoanalytic treatment. I agree and would add that this reactivation could include a reactivated capacity to look up, bind, and integrate distributed sensory data for coherent representation in declarative and working memory in some but not all cases where there is a failure to represent traumatic experience.

The psychotherapist must discern the level at which information is encoded or represented. Consider the chaotic fragments of sensory experience that overtook Ms. D’s or Ms. M’s awareness. Should we explore their multiple symbolic meanings, as we would a dream? I submit that this would not be helpful, because their concrete, somatosensory fragments or subsymbolic information are not symbolic structures or organizations. People who have encoded traumatic experiences at this level cannot use their fragmented images for defense or symbolic play or to refer to multiple meanings. That would require organization on a symbolic level. The task is not to decode and interpret hidden meanings. Rather, the task is to encode. We need to help the patient construct and organize experience on a symbolic level, to raise the information to the level of words, narratives, and metaphors. Again, Loewald (1955) presented a similar idea: “Working through is not primarily conceived of here as the undoing of repressions or other defenses, but is seen as the strenuous task of helping to lift unconscious processes onto a new level of integration, the level of secondary process” (p. 39).

On the other hand, the imagery in Ms. M’s dream is registered and processed as visual and linguistic symbols. As such, they can represent multiple meanings. The lodge refers to the room in which she was abused and to her relative. The fire represents rage, her destructive wishes, and fears toward her relative and the janitor, and her fear that these could not be contained. At least with this dream, Ms. M does not need help to encode and organize her images on a symbolic level. The images in this dream are visually coherent, reflect extensive verbal processing, and allow for imaginative elaboration. The therapist’s task here is to interpret, or help the patient interpret and elaborate, the meanings of her visual metaphors.

Self-regulation

Trauma disrupts self-regulation by disrupting basic physiologic processes and by undermining the ability to symbolize. I argue that self-regulation and the ability to symbolize are interdependent and interactive. Disrupting one will adversely affect the other and will alter the subjective experience of self.

Regulation deficits and trauma

Traumatized persons are unable to regulate basic biological safety and alarm mechanisms and show evidence of profound and long-lasting physiologic dysregulation. PTSD researchers have documented abnormalities in regulating catecholamines and serotonin, arousal, and the overall stress response (Bremner, Innis, et al., 1997; Charney et al., 1993; Morgan, Grillon, Lubin, & Southwick, 1997; Southwick et al., 1993, 1997), the CRH-ACTH-cortisol system (Yehuda, Southwick, Krystal, et al., 1993; Yehuda, Resnick, Kahana, & Giller, 1993; Yehuda, Boas, et al., 1995) and the cortisol response to stress (Resnick, Yehuda, Pittman, & Foy, 1995), and in regulating endogenous opioids (Pitman et al., 1990; van der Kolk et al., 1989). PTSD patients also have trouble regulating aggression (Chemtob et al., 1997), attachment (Lindy, 1988), and attention and memory (Bremner, Randall, Scott, et al., 1995a; Vasterling, Graily, Constans, and Surker, 1998; Yehuda et al., 1995). Conditioned fear reactions are mediated by the amygdala and are activated by stimuli that bypass the hippocampus and cortex (subsymbolic information) (LeDoux, 1996). This fear becomes an enduring response to simple cues.
associated with trauma, without awareness of the context that triggers them and without conscious control. The net result of the physiologic disruption cited above is a profound deficit in self-regulation.

Self-regulation and symbolization

The physiologic effects of trauma disrupt self-regulation directly. But trauma also interferes with the capacity for symbolic thinking. This impaired capacity to symbolize experience and affect also disrupts affect regulation and other dimensions of self-regulation. Self-regulation and the ability to symbolize are tied to each other. The ability to symbolize improves the ability to regulate affect and to regulate physiologic states. Psychoanalyst Peter Fonagy (1991) writes: “The ability to represent the idea of an affect is crucial in the achievement of control over overwhelming affect” (p. 642).

Neuropsychological theories of self-control emphasize functions attributed to the prefrontal lobes that involve some form of symbolic representation, such as private, self-directed speech (Barkley, 1997; Bronowski, 1977; Vygotsky, 1987). Dialogues with others are internalized and directed to the self. While these theories focus more on cognitive and behavioral self-control, they include control and regulation of affect as a function of internal language. According to Barkley (1997): “the separation of the affective tone of a message from its content may be most evident and most necessary in situations that are highly emotionally charged for the individual. This separation of affect, I believe, represents the more contemporary concept in developmental psychology of the self-regulation of emotion” (p. 87).

Barkley (1997) proposes a theory of self-control that involves multiple forms of representing information to the self. One of these is the capacity to represent time and to organize behavior across time (Barkley, 1997; Fluster, 1989; Levine et al., 1998). This ability includes a sense of time as well as representing one’s personal past and a potential future. Terr (1984) has described profound distortions in trauma survivors in the ability to represent time, including the inability to order events in time and the inability to represent a future. I submit that the subversions of time sense described by Terr are a subset of more widespread failures in symbolic representation following trauma and are a source of impaired self-regulation in traumatized persons.

This ability to organize behavior across time requires a cross-temporal sense of self that, in part, depends on episodic memory of one’s personal past. In the journal Brain, Levine et al. (1998) describe a patient with damage to the right ventral cortex and to the connections between this brain structure and the temporal cortex. The injury caused a deficit in episodic memory that resulted in a “self-regulatory disorder” and a disturbed sense of personal identity. The authors connect the patient’s memory deficit, and thus his inability to access information about the self, with his impaired self-regulation. They write:

We propose that M.L.’s impaired self-regulation (both inside and outside the laboratory) and his mnemonic deficits can be unified within the concept of impaired autonoetic awareness that affects behavior across the time dimension. His behavior is driven by generic information that he has learned about how one should behave, rather than by goals and intentions that arise from one’s own identity. [Levine et al., 1998, p. 1968]

Levine and colleagues use the notion of a “mnemonic scotoma” to show how deficits in autonoetic awareness affect self-experience and self-regulation. They state:

in healthy adults referral to the self, defined here as a multimodal distributed network of associations accrued over a life-time of experiences, gives rise to autonoetic awareness, which in turn supports the formulation of future goals, especially in unstructured situations where the goal cannot be derived from the environment or habit. Brain damage can cause mnemonic scotoma for information contained in this network, especially damage to regions important to the indexing or triggering of recollection of personal past events (i.e., anterior temporal or frontal lobes), resulting in failure of analysis or online maintenance of information concerning the self (or some aspect of it) as a continuous entity across time. [p. 1968]

These brain-damaged patients cannot hold self-specific information on-line, integrate it with mental representations of the past and the future, or use it to drive his behavior towards personally relevant goals. [p. 1969]

Psychic trauma interferes with the ability to represent personal experiences or to relate these experiences to the self. I submit that this can lead to a relative deficit in autonoetic awareness in the area of experience affected by trauma, which leads to impaired self-regulation and often to discontinuities in self-experience. Autonoetic awareness can be blocked due to biological and psychological deficit as well as due to psychic conflict. So, biological and psychological factors combine to produce impairments in representation and self-regulation like that of the brain-damaged patient described above.

The resulting problem with symbolization and self-regulation is similar to a condition described by psychoanalyst Peter Fonagy and his colleagues as
impairment in reflective self-functioning (RSF) (Fonagy, 1991; Fonagy, 1995). RSF includes the ability to reflect on one's self, as well as to represent one's affects and intentions and to represent the mental states of others. RSF is essential for self-regulation and for relatedness. Conflicts and deficits that arise from conditions of childhood trauma and neglect undermine RSF.

Impaired self-regulation, then, is cause and consequence of the impaired symbolic thinking that follows trauma. Self-regulation and symbolization are interdependent. The combined result of their disruption can cause profound alterations in the subjective life of traumatized patients.

Self-regulation, symbolization, and subjectivity

Traumatized patients often report an inner sense of concreteness, deadness, and emptiness or a sense of being devoid of meaning. At its most extreme, the ability to sustain an integrated sense of personal identity breaks down, leading to a subjective experience referred to by some psychoanalysts as a "black hole" (Laub & Auerhahn, 1993; Cohen, 1983). For example, when Ms. D was hyperaroused and flooded with fragmented images, she felt her self slipping into a void and thought she was dying. She consciously struggled to focus on just being, existing.

There are many and complex reasons for this disturbance of subjective life, which include both conflict and deficit (Laub & Auerhahn, 1993). A sense of inner deadness or emptiness can result from massive "anal" defense against overwhelming affect (Shengold, 1979, 1989), from an inability to symbolize affect (alexithymia) (Krystal, 1978), from an inability to represent a sense of time with a past and future (Terr, 1984), as well as from other factors.

The disturbed subjectivity that often follows trauma can be the combined result of impaired symbolization and impaired physiologic regulation. Others have argued that some form of coordination of symbolic capacity and physiologic processes is necessary to sustain a meaningful and integrated subjective life. Psychoanalyst Julia Kristeva (1989) places a failure to symbolize at the basis of the impaired subjectivity and meaning seen in depression. She asks, "what mechanisms erase symbolic impact within the subject" (p. 37), and she speculates that there is a disconnection between biologic and symbolic processes. "Is it conditioned by the neuronal and endocrine network that underlies (but in what fashion?) psychic representations and particularly word representations, and also the channels that link them to hypothalamic nuclei?" (Kristeva, 1989, p. 37).

According to Kristeva (1989), reconnecting biological and symbolic mechanisms, through interpretation and with the use of medications, restores the experience of meaning: "language, considered empty up to this point because cut off from affective and vocal inscriptions, [is] revitalized and may become a space of desire, that is of meaning for the subject" (p. 57).

According to neurologist Antonio Damasio (1994), our most basic self-awareness emerges from combining primordial and continuous representations from the body, including physiologic state, with perceptual representations of external objects. Changes that occur in body state and emotional state before, during, and after perceptual processing give rise to "a dispositional representation of the self in the process of changing as the organism responds to an object" (Damasio, 1994, p. 242). "I propose that subjectivity emerges during the latter step when the brain is producing not just images of an object, not just images of organism responses to the object, but a third kind of image, that of an organism in the act of perceiving and responding to an object" (p. 243).

According to Damasio’s (1994) model, our core biological sense of self could be disturbed by trauma, either by disrupting basic physiologic regulation or by interfering with the capacity to represent internal or external objects of perception. Again, this core self experience (or protoself) emerges from the convergence of these two neural processes (Damasio, 1994, 1999). Damasio (1994) writes: "at each moment the state of the self is constructed, from the ground up. It is an evanescent reference state, so continuously and consistently reconstructed that the owner never knows it is being remade unless something goes wrong with the remaking" (p. 240). This could explain how disruptions in subjective awareness can be dynamic and fluctuating. Subjective awareness may be disrupted during states of physiologic dysregulation or when incompletely symbolized traumatic experiences overtake perception. Normal self-experience may be restored at other times.

Damasio (1999) also proposes another, higher level of self experience, which he calls the biographical self and which requires a more extended form of consciousness. Extended consciousness and the biographical self depend on the functioning of the more basic, core biological self. Extended consciousness also requires access to biographical memory and the capacity to hold extensive information in working memory. Based on Mesulam’s (1998) model, we could say that Damasio’s (1999) extended consciousness or biographical self depends on the ability of transmodal nodes in the hippocampal/entorhinal cortex and in the prefrontal
cortex to access and bind distributed sensory experience. Though extended consciousness and the biographical self do not require language, I assume that linguistic encoding allows the higher level of self—a greater degree of associative elaboration and depth. This would require the intact functioning of transmodal nodes in Wernicke’s and Broca’s areas. When transmodal nodes become temporarily unable to access and bind information, perhaps because they are clogged with information, arousal, or affect, the higher-level sense of self—or biographical self—may be transiently restricted, impoverished, or lost altogether. In sum, I submit that by disrupting core physiologic regulation, by disrupting one’s capacity to organize perception on a symbolic level, and by disrupting one’s capacity to access biographical memory, trauma can disrupt the sense of self either at the most basic, core level, or at the higher, biographical level.

Finally, psychoanalyst Arnold Modell’s (1993) recent work can be applied to the experience of inner restriction and deadness reported by trauma survivors and is consistent with the notion presented in this paper of a combined disruption of symbolization and self-regulation. According to Modell, intrusions of affect that are experienced as outside the domain of the self’s control have a profoundly demoralizing and devitalizing effect on one’s subjective sense of self. He argues that affects that are not within one’s control are experienced as alien to the self and are labeled as “It.” I suspect that the same is true for experiences that one is unable to symbolize or name, such as occurs with trauma and with other conditions where symbolization is compromised. Both the inability to regulate affect and physiology and the inability to synchronize experience lead to areas of experience that are alien to the self and are experienced as non-self. This can devitalize and restrict the sense of self. As persons are more able to regulate affect and other reactions, and as they are more able to symbolize and represent experiences and feelings, these reactions, feelings, and experiences are brought within the domain of self-control and are no longer experienced as “It.” Modell (1993) notes: “If intense affective experiences can be contained within the self, the self extends its domain, and the individual will feel strengthened, alive and authentic” (p. 54).

Treatment implications

Impaired self-regulation, then, is cause and consequence of impaired symbolic representation. Patients need help to symbolize and verbalize traumatic experience, as well as their feelings and other mental states. They also need help to regulate their feelings and other physiologic functions. This help takes various forms, including articulating feelings, non-verbal affect-regulating interactions, and medication. Patients also need help to repair their ability to relate to others, which is damaged as a consequence of trauma. We rely on relationships to some degree for self-regulation and for symbolizing experience and affect.

Medication is often an important component in the psychotherapy of traumatized patients (Saporta & Case, 1993). Various medicines help regulate affect, arousal, and conditioned fear reactions (Davidson, 1997; Saporta & Case, 1993). An example is medications that enhance serotonin function in the brain, such as the selective serotonin reuptake inhibitors (SSRIs), which are effective in treating PTSD symptoms (Kline et al., 1994; Marmar et al., 1996; Rothbaum, Ninan, & Thomas, 1996; van der Kolk et al., 1994). SSRIs may work in PTSD by correcting abnormalities in serotonin function that may be induced by extreme stress. Stress can decrease serotonin function in animal models for PTSD (for reviews of this evidence, see Saporta & Case, 1993; van der Kolk & Saporta, 1991). Depletion of serotonin in animals leads to hyperarousal, increased reactivity, increased aggression, and increased response to amphetamines (Breese, Cooper, & Mueller, 1974; Davis & Sheard, 1976; Depue & Spoont, 1986; Gerson & Baldessarini, 1980; Poschlova, Masek, & Krisiak, 1977; Samanin & Garattini, 1976). Decreased serotonin levels have been correlated with hostility, impulsivity, and aggression in humans (Coccaro et al., 1989; van Kammen, 1987). Serotonin appears to mediate a behavioral inhibition system in the brain (Depue & Spoont, 1986; Gray, 1987; Soubrié, 1986). This system inhibits behaviors that have been motivated by emergency responses or previous reward but are no longer adaptive. So, serotonin abnormalities may contribute to the noradrenergic dysregulation, hyperarousal, hyperreactivity, difficulties in regulating aggression, impulsivity, and the repetition of learned emergency responses seen in traumatized persons. These medications can improve self-control and self-regulation in multiple domains, including regulation of affect, arousal, aggression, and impulsivity (Casper, 1998).

SSRIs and other medicines may enhance the traumatized patient’s ability to engage in psychotherapy (Saporta & Case, 1993). Extreme arousal and conditioned fear reactions that are out of one’s control undermine the feeling of safety in one’s own body and consequently in the therapeutic relationship. Improved self-regulation with medication promotes the self-control, safety, and trust necessary for establishing a holding environment and for deepening analytic work. Improved physiologic regulation also...
enhances the ability to symbolize experience and affect that is necessary for psychotherapy. As patients are more able to regulate physiologic and affective reactions, they feel subjectively strengthened. Modell (1993) writes: “Intensity of feeling from whatever source may be threatening, but there is some gain in recognizing these feelings as being part of the self. If the self can experience these incursions as something within its control, the self is strengthened” (p. 54).

Conclusion

Persons such as Ms. D and Ms. M first “know” their traumas in the form of perceptual and somatic fragments (Laub & Auerhahn, 1993; van der Kolk & Fisler, 1995). They are experienced as an alien “It,” with no relation to their self or their personal history. As these persons improve in their ability to symbolize, and their images become more integrated and coherent, they are increasingly able to relate these images and experiences to their self and their personal history. The more they can be symbolized in language, the more their meaning can be elaborated and extended. The more a person is able to symbolize experience and affect, the better he or she is able to regulate affects and other mental states. Better symbolic capacity and better self-regulation lead to a subjective sense of expansion and strength of the self. Affect regulation and self-regulation more generally, plus the ability to relate, are promoted by empathic, affect-regulating interactions in therapy and by other biological interventions such as medication.

We can begin to see a system of complex and interdependent components. Change in each of the domains mentioned above—self-regulation, the ability to symbolize, and the capacity to relate to others—is interdependent with change in each of the other domains. Nothing changes in isolation. By combining psychological and biological perspectives, we are more able to appreciate the complex system of interacting components that treatment must address. I hope that I have been able to convey that both psychoanalytic and biological perspectives on trauma can enrich and enhance each other as we try to understand and help our patients.

REFERENCES


Poschlova, N., Masek, K., & Krisiak, M. (1977). Ampheta-


