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### Depotential of Symptom-Producing Implicit Memory in Coherence Therapy

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## DEPOTENTIATION OF SYMPTOM-PRODUCING IMPLICIT MEMORY IN COHERENCE THERAPY

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*In this second of three articles, we suggest criteria defining the optimal use of neuroplasticity (synaptic change) in psychotherapy and provide a detailed examination of the use of neuroplasticity in coherence therapy. We delineate a model of how coherence therapy engages native mental processes that (a) efficiently reveal specific, symptom-generating, unconscious personal constructs in implicit emotional memory and then (b) selectively depotentiate these constructs, ending symptom production. Both the psychological and the neural operation of this methodology are described, particularly how it defines and follows the built-in rules of change of the brain–mind–body system. On neuroscientific grounds, we suggest a fundamental distinction between transformative change, which permanently eliminates symptom-generating constructs and neural circuits, and counteractive change, which creates new constructs and circuits that compete against the symptom-generating ones and is inherently susceptible to relapse. We propose that coherence therapy achieves transformative change through the reconsolidation of memory, a recently discovered form of neuroplasticity, and present evidence consistent with this hypothesis. Subjective attention emerges as a critical agent of change in both the phenomenological and neural viewpoints, profoundly connecting these two domains.*

The constructivist clinical methodology now known as coherence therapy and its conceptual framework, coherence psychology,<sup>1</sup> emerged from an ongoing, phenomenological study of psychotherapeutically induced change by Ecker and Hulley (1996).

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Launched in 1985, this study was carried out in the local clinical scientist tradition (Stricker & Trierweiler, 1995), in which a practicing clinician systematically uses his or her own sessions as an effectiveness research laboratory.<sup>2</sup> The goal of this study was to identify the brain–mind–body system’s native processes and rules of change. This was sought through close scrutiny of the phenomenology whenever a therapy client of Ecker or Hulley had an experience that led unambiguously to lasting symptom cessation.

The aim was to let the native phenomenology guide the shaping of the methodology—that is, to identify the elements of the client’s internal process of change very accurately in hindsight (if necessary by querying the client about it); to discern exactly which client–therapist interaction had successfully fostered that change process; to distill the pattern and principle of that interaction into a method that could be applied knowingly with other clients; and to assemble a therapy comprising only methods selected in that manner, methods shaped for cooperating closely with the identified processes (Ecker & Hulley, 1996, 2000a, 2000b, 2004).

Therapeutic methods not selected in by this process were discarded, even if they were deeply familiar, widely practiced, and time honored, such as offering interpretations. The working assumption was that a therapy formed entirely in this way—dictated by the brain–mind–body system’s own terms—would be an optimized therapy that would yield a marked increase in effectiveness.

A principal result was the finding that clients’ in-session experiences that resulted in lasting change virtually always stemmed from interactions in which the therapist had completely stopped trying to counteract or prevent the client’s symptoms or difficulties. The therapist had focused instead solely on eliciting from the client how and why the symptom actually made sense to have within the client’s world of meaning. This hidden coherence of the symptom proved to be quickly discoverable in most cases, not as a speculation but as a lucid experience of a personal emotional truth, even if the client initially had no conscious notion of it whatsoever.

The observation of consistent, striking effectiveness resulting from coherence-focused work, in contrast to the field’s standard,

counteractive approaches, led to the development of simple methods for quickly detecting and revealing the cogent themes and purposes actually maintaining clients' seemingly irrational symptoms. Upon verifying with virtually all clients the coherence of their symptoms and problems in living, Ecker and Hulley (1996) defined "symptom coherence" as a comprehensive model of symptom production.

Independently, research in neuropsychology has developed a complex knowledge of the brain's native processes and rules of change on the reductionistic level of the brain's physiology, neurons, synapses, genes, and molecules. Neuroscientists too think and write in terms of "rules" of change, and they strive to identify the exact physiological and biochemical implementation of those rules.

If coherence therapy's account of the brain–mind–body system's built-in rules of change is correct, it should be possible to demonstrate that these phenomenological rules correspond closely to neurodynamic rules and processes. Neuroscience has recently developed to the point where the correspondences between the reductionist findings of the brain sciences and the holistic phenomenology of psychotherapy can begin to be assessed. According to a neurophysiology researcher at the Massachusetts Institute of Technology, "Ten years ago we only knew what some of the key components of synapses are. Now we know what *most* of them are" (Olson, 2004, p. 16). More than 1,000 different proteins are active agents in controlling the operation of synapses and neurotransmitters. Nobel laureate Eric Kandel states, "Although we do not have a complete understanding of how synapses work and how they are regulated for plasticity, learning and memory, we do have a satisfactory understanding" (Olson, 2004, p. 23).

Attempting to model the psychobiology of optimal therapeutic change is new territory for us, for psychotherapy, and for neurobiology, so it is necessary to create an orienting framework. We suggest that a defining feature of a maximally effective psychotherapy is an optimal use of the brain's capacity for neuroplasticity, or synaptic change. We propose a three-part definition of the optimal use of neuroplasticity:

- (a) recruitment of the most potent types of synaptic change,
- (b) in the brain regions and neural circuits causing symptom production,
- (c) on the shortest timescale neurally possible.

This definition specifies the what, where and when that could reasonably be regarded as an optimal recruitment of neuroplasticity. We will use this definition in this and the following article as a gauge of psychotherapeutic optimization.

Familiarity with the previous (first) article in this series (Toomey & Ecker, 2007) is assumed here, particularly the overview of coherence therapy, the definition of symptom coherence, and the case example of Carol. Our purpose now is to delineate:

- the brain–mind–body system’s built-in rules for change as defined by coherence therapy in phenomenological terms;
- the methodological procedures of coherence therapy and how they efficiently follow those rules for change; and
- the neural mechanisms of change that are plausibly recruited by these procedures.

These three strands, woven together, form a unified fabric of neural and phenomenological knowledge of how the specific implicit memories driving symptom production can be efficiently depotentiated, ending production of a wide range of symptoms reliably and rapidly.

With few exceptions, we consider only the main features of the methodology, not the finer-grain techniques and skills involved in its implementation (for a more complete clinical discussion, see Ecker & Hulley, 1996, 2000a, 2004). As described in our prior article, the methodology that defines coherence therapy has three main components: The creation of experiences that (1) discover, (2) integrate, and (3) transform prosymptom positions. For each of these areas of methodology, we will address the three strands listed above.

It is also worth noting in advance that in each area of the methodology, the experiences that are created involve guiding the client’s *attention* in specialized ways. The coherence-guided use of attention is the central activity of coherence therapy. A

central theme of this article will be the experiential and neural effects of attention deployed in accurate cooperation with the native processes of change.

### **The Creation of Experiences That Discover Prosymptom Positions**

Coherence therapy identifies the following native capacities or processes of the brain–mind–body system and uses them as the guiding rules for the discovery work.<sup>3</sup>

- Unconscious, implicit personal constructs are immediately accessible to awareness.
- Accessing them occurs through experiencing them subjectively, emotionally, somatically.
- Accessing them requires methods that match their qualitative characteristics as knowledge structures.

We will describe each of these, first in coherence therapy's experiential–phenomenological terms and then in terms of their probable neurodynamic substrates.

The *immediate accessibility of unconscious constructs* (Ecker & Hulley, 1996, 2000a, 2004) is apparent in the frequent observation while carrying out coherence therapy that a person can access and directly experience longstanding, nonverbal, fully unconscious, symptom-requiring constructs after a few minutes of suitably designed process. The guiding of attention to the unconscious material can occur at any time, from the first session; passage of time per se is not an intrinsic requirement for the accessing to occur. Even constructs formed in childhood and unconscious for decades usually can be brought into experiential awareness through methods that accurately bring attention into them. The unconscious status of a personal construct evidently means only that it is chronically unattended, whether habitually or purposefully, not that it is inaccessible. The observation by Ecker and Hulley that the unconscious/implicit constructs and schemas driving symptom production are, as a rule, available for prompt, direct experiencing stands in contradiction to the view in cognitive psychology that only the manifested *effects* of an implicit schema are accessible to awareness, not the unconscious schema itself

(see, for example, Nisbett & Ross, 1980). In this way unconscious personal constructs appear to differ from, say, the unconscious schemas governing generative grammar or perceptual parsing, which are not transparently available upon introspection.

Although a person's universe of unconscious constructs is vast and dark, the small constellation of unconscious prosymptom constructs driving production of a given symptom usually can be induced to glow in the dark, as it were, and be spotted experientially. This is done by selectively eliciting these constructs through focusing on their coherence. That is, the prosymptom constructs are unique by virtue of being the constructs that compellingly require the existence of the symptom. Coherence therapy's discovery methodology exploits this property. The therapist structures an experience designed to elicit from a prosymptom position a response that is noticeable to the client. The therapist then guides the client's attention to focus on this manifestation (an inner image, meaning, feeling, body sensation, knowing, verbal thought, memory, etc.).

Various simple, experiential techniques are reliably effective in bringing this about. This occurs without the therapist knowing or hypothesizing about the content of any prosymptom position, as shown in our case example. Carol was guided to experience a spontaneous completion of the sentence fragment, "If I were to like having sex with my husband, I'd feel ....." Her neocortical, conscious attention noticed an emotional response arising from a nonneocortical, presumably subcortical prosymptom position, which she verbalized with the word "embarrassed." The prosymptom source of this response is immediately evident to the therapist from the symptom-requiring nature of the content: To feel embarrassed to have sex inclines her not to have sex, which is the symptom.

The moment when the client's attention first registers the elicited prosymptom response is the moment when accessing has begun—the first contact between the neocortex's conscious attention and the prosymptom knowings residing presumably in subcortical brain systems harboring implicit emotional memory.

A prosymptom position activated in daily life is not normally accompanied by any conscious attention whatsoever, because it is an implicit memory, a knowing one does not know one knows. The discovery methodology is designed to involve attention in the

activation of this memory, like following a pathway that was always there but never before noticed.

The neural correlate of this event—conscious attention registering a specific prosymptom response—is the formation of a new synaptic linkage between the neocortex and the part of the amygdalar and/or other subcortical neural network that encodes the prosymptom constructs. For an event in neural network A to show up directly as new information in neural network B means that a synaptic link exists between the two networks. (The first suggestion that the creation of new mental contents corresponds on the cellular level to the creation of new connections between neurons was made by Ramon y Cajal [1894]. It was then 76 years until direct, empirical verification of this model was achieved by Castellucci, Pinsker, Kupfermann, & Kandel [1970] and by Kupfermann, Castellucci, Pinsker, & Kandel [1970].)

In coherence therapy, the client's attention drives the formation of this synaptic linkage. A neurodynamic finding of central relevance to coherence therapy is the fact that synaptic connections are generated by the subjective focusing of attention on an item in the conscious field that has subjective importance (Bailey, Kandel, & Si, 2004; Kentros, Agnihotri, Hawkins, Muller, & Kandel, 2001; Kentros, Agnihotri, Streater, Hawkins, & Kandel, 2004). The use of focused attention to drive synaptic change in the neural network maintaining symptom production is the essence of coherence therapy, from the neurodynamic perspective. This begins with the discovery work, in which the neocortex's conscious attention to the contents of the subcortical prosymptom position generates new, transient synaptic connections linking those two neural systems.

The timescale of the clinically observed immediate accessibility of unconscious constructions—the time between the therapist's spoken evocation and a manifested, noticed response from the prosymptom position—is typically 1 to 10 seconds. The rapid, short-term activation of synapses involved in this immediate accessing is presumably no different than that involved when any novel perception occurs. The discovery methods of coherence therapy induce the prosymptom neural network to be the source of the novel perception, as described above.

Teskey (2001), reviewing findings from across the research on neuroplasticity, noted that in response to a new experience,



“synaptic potentiation or depression [of existing, functioning synapses] can take place on millisecond time scales” (p. 7). Other forms of neuroplasticity, such as the activation of nonfunctional, “silent” synapses or the creation of entirely new synapses, have been observed on a timescale of hours (Kim et al., 2003), too slow to account for moment-to-moment, first-time accessing (but just right to account for the observed timescale of integration, discussed below). Therefore, the neural substrate of coherence therapy’s creation of discovery experiences can be assumed to be the formation of a new linkage of neurons via alterations to existing, active synapses.

The client may or may not immediately comprehend the prosymptom significance of the noticed, emergent material. The therapist does, however, and keeps the client’s attention focused on this material, deepening the client into a fuller emotional experience of it. It is the client’s continuing attention to the newly noticed feature that maintains the new synaptic linkage. If her or his attention wanders away from that feature, within seconds it is completely forgotten. This corresponds to the deactivation of the newly active synapses, which return to their prior condition. Neuroscientists have directly observed the depotentiation of memory-encoding synapses to be accompanied by the concurrent disappearance of the behavioral responses driven by that memory (Bailey & Chen, 1989; Bailey & Kandel, 1993). The therapist must actively guide the client’s attention to remain engaged with, or to return to, the prosymptom position’s first manifestation.

Sustained attention on any one emergent element of a prosymptom position not only preserves and strengthens the synapses involved but also strongly tends to elicit other, linked elements of that prosymptom position. This occurs either spontaneously or through another structured prompting by the therapist—a process termed *serial accessing* by Ecker and Hulley (1996). The attended experiencing of each additional element of prosymptom material corresponds to a more thorough synaptic linkage between neocortex and the subcortical prosymptom neural network. Discovery continues until the entire prosymptom position is directly attended, experienced, and synaptically linked.

At that point, the coherence of symptom production is transparent: The client is lucidly aware of how and why the symptom

is necessary to have, including the experience of his or her own agency in producing a symptom that had previously seemed to have a life of its own. This initial, subjective experiencing of a prosymptom position is an altered state, a state-specific knowing (Singer & Salovey, 1988), that involves inhabiting a personal reality quite different than that of the everyday conscious personality (Tart, 1975). (To reach this point takes one to six sessions for about two-thirds of the clients in a therapy practice that is nonspecialized with respect to symptoms; six to 12 sessions for about one-quarter of all clients; and more than 12 sessions for about 10% who have multiple, entangled prosymptom positions).

This discovery process is entirely phenomenological: *The therapist learns from the client* the content of the prosymptom constructs (theme, purpose, knowings, feelings, images, tactics), not the other way around. The therapist offers no interpretations. The client would discover the same material by working with any proficient coherence therapist.

Accessing a prosymptom position, then, consists of the initial formation of a live, experiential and synaptic link between at least two neural networks: that of the conscious, verbal module in the neocortex and that of the prosymptom module in one or more brain systems harboring emotional implicit memory. This link cannot be generated by the neocortex's verbal-cognitive, semantic knowings *about* the prosymptom contents, just as a map drawn on a piece of paper cannot bring itself to the place represented. A cognitive insight is an internal event in the neocortex that is also experienced by the neocortex, so the synapses created by the insight do not lead outside the neocortex.

In contrast, when the conscious attention of the neocortex notices an internal, experiential event whose unconscious source is a prosymptom position, those two neural networks, the neocortex and the brain system holding the prosymptom position, begin to be linked by synapses.

By *conscious attention* we mean not merely having thoughts *about* the object of attention but, rather, having the subjective experience of the living encounter with the object of attention. To give attention to the moon is to experience seeing the moon, which is different than giving attention to thoughts about the moon. To give attention to prosymptom material is to experience

this material's emotional, perceptual, somatosensory, kinesthetic, and energetic elements. It is the immersive, multimodal, subjective experience and recognition of these contents as felt emotional truth that is the actual attending to them and the psychological and neural accessing of them.

That is the second of the three built-in rules of discovery: *Accessing requires subjective experiencing*. A nonimmersive, conceptual-semantic recognizing of these same contents does not actually access them, no matter how closely focused on the material it may be. Cognitive insights result from, but as a rule do not produce, a direct experience of the implicit, prosymptom material. Experiential rather than cognitive methods are required, the defining features of which we describe below.

The phenomenological principle that accessing requires subjective experiencing has a distinct neural basis. It was established recently, both molecularly and behaviorally, that *only while a long-term memory (knowledge structure) is evoked and reactivated can its stable, encoding synapses be switched into a labile, changeable state* (Alberini, 2005; Nader, 2003; Nader, Schafe, & LeDoux, 2000; Sara, 2000; Walker, Brakefield, Hobson, & Stickgold, 2003). This requirement of memory reactivation is the neural correlate of coherence therapy's tenet that accessing occurs only through subjective experiencing. In order for a knowledge structure in memory (such as a prosymptom position) to be changeable, it must be evoked, reactivated, and experienced, not merely conceptualized. We will return to this critically important neural mechanism in the section on transformation, below. Yet even the initial accessing involved in discovery brings about a neural change, in that the presumably subcortical neurons in which the prosymptom knowings are encoded are forming new synaptic links, which will be used for transformation later.

Of course, prosymptom positions usually involve elements of core meaning and emotion—deeply tender, vulnerable, or painful areas of personal experience. If either the emotional discomfort felt by the client or the client's resistance makes it apparent that the opening steps of discovery are not workable for the client, the therapist reduces the size of the discovery steps down to a level that allows the client to go forward with the process, which can then proceed immediately. Also, for the vast majority of therapy clients, if the therapist has an emotionally safe presence, there is

no delay required for trust building. These conditions have been well defined by Ecker and Hulley (2004).

The new circuits created by discovery experiences between the subcortical neural networks harboring the prosymptom material and the neocortical networks subserving the conscious, verbal self undoubtedly are complex. The amygdala, for example, has strong connections to the orbital and medial prefrontal cortex, which are major centers subserving conscious emotional experience and which in turn link the amygdala to more cognitive neocortical circuits. (See Halgren, 1992, for a detailed review of the reciprocal connections between cortical areas and the amygdala.) The neocortex's conscious verbal module is in the left cortical hemisphere. The right cortical hemisphere handles the nonverbal experience of conscious emotion and emotional meaning, and is anatomically far more connected to emotional centers in the limbic system than is the left hemisphere. The elicited responses of a prosymptom position therefore can be assumed to register most directly in the emotional awareness of the right cortical hemisphere, particularly the right orbitofrontal region. Directing conscious attention to this experience of emotional meaning can create a link, via the corpus callosum, across to the left orbitofrontal region, where it is now available for verbalizing by the left neocortex's verbal module (Schoore, 2001).

In effect, the right hemisphere is the verbal left hemisphere's window into the limbic world of unconscious, personal, emotional truth. When Carol said she felt "embarrassed," her left hemisphere had recognized verbally that in her right hemisphere there was a social *feeling* of embarrassment, which in turn presumably had its source in a more primary subcortical recognition of endangerment due to exposure (which had been brought to conscious attention in the discovery experience prompted by therapist). It is through the verbalization that the conscious personality knows what is being experienced by the other brain systems and in the body nonverbally.

Accessing an unconscious emotional response and the implicit schema generating it usually occurs in those two steps: First, via the right hemisphere, is the nonverbal, affective, and somatic experiencing of activated subcortical material, which is then supplemented by the verbalized knowing of this experience in the left hemisphere. Therapy clients vary widely in the amount

of time and help they need with each of these two steps. The complete discovery experience is the unified feeling-knowing and verbal-knowing of the elicited subcortical material. The neocortex has been informed of prosymptom meanings and feelings by the subcortex.

The creation of a discovery experience therefore entails the formation of new synaptic circuits linking several brain regions. The main links presumably are those from limbic subcortex (the home of the implicit memory driving symptom production) to right orbitofrontal cortex to left neocortex, inaugurating the neural integration of these systems both vertically and horizontally. A number of other regions can also be expected to participate importantly in the process. The hippocampus, which is essential for forming and storing the explicit (conscious) memory of new personal experiences, must be involved, as are regions responsible for the experience of specific emotions, such as the subgenual cingulate and dorsal insula (for sadness, grief, and depression), or the ventral insula and anterior temporal cortices (for anxiety; Liotti et al., 2000). Other regions likely to play a role include the anterior cingulate cortex (involved in the directing of focused attention and in modulating emotional responses), and the ventral medial prefrontal cortex (involved in recognizing and understanding emotional significance).

Therapy clients also appear to vary widely in where, along the main pathway from left neocortex to right cortex to limbic system, they position their consciousness while having a discovery experience. Those who keep their awareness based in their verbal module remain fully in their familiar, conscious personality. From that secure, controlled vantage point, they observe and verbalize the emotional and perceptual material that their right hemisphere presents to their observing awareness. Such clients remain almost matter of fact as they register and verbalize the high-stakes, subjective material elicited.

Those who allow their locus of experiencing to slide over into the right hemisphere, allowing a subjective immersion in the emotional and perceptual material playing there, have a very different experience and manner. They are *in* the elicited feelings and knowings and display the various, congruent signs of actually having an emotional experience, which is a moderately altered state.

Those who allow their attention to slide even further, through the right hemisphere and all the way into the prosymptom material in its subcortical home, are more fully immersed in the vivid, compelling, living emotional knowledge carried there. The client has stepped through the looking glass—the right hemisphere—into limbic wonderland, a fully altered state of inhabiting an emotional reality in which little remains of the usual adult personality or ego state. Instead, the experience typically is composed of a younger developmental state of identity and a subjective immersion in a specific schema, its associated feelings, and perhaps an image or scene of its instantiation. Brain imaging has shown that infants live primarily in the emotional and sensory vividness of their limbic system (Chugani, Muller, & Chugani, 1996).

The therapist's own mirror neurons (Gallese, 2003)<sup>4</sup> presumably facilitate the shared trance in which the therapist empathically and vicariously accompanies the client into a particular emotional reality, perceives some of its experiential features, and guides the client's exploration of it. The therapist needs to be able to experience concurrently in two modes: the vicarious mode and the cognitive mode that allows ongoing reference to his or her cognitive map of therapeutic methodology. For example, the verbalizing necessary for a complete discovery experience is not a natural act for the client in the altered state of a discovery experience, and needs to be prompted and assisted by the therapist. In prompting this verbalizing, the therapist in effect is instructing the client, who may be immersed deep in the limbic sea, "Now, stay right here, but reach for your neocortex and bring it here." Such is the experience of neural integration for therapy clients who can allow the full slide through the rabbit hole.

Many clients can be guided to allow an incremental slide into their subjectivity, despite initial resistance. The greater the subjective immersion, the stronger the accessing of the material and the more available for change it becomes. A small fraction of therapy clients have an incapacity for subjective immersion that appears profound. It remains to be seen whether the cause of such incapacity is psychological or perhaps a structural lack of connectivity between the neural networks involved.

It remains true, however, that the new synapses formed even during the fully immersed, more intense discovery experiences would quickly dissipate—and the rich emotional reality of the

prosymptom position would disappear like a vivid dream that is suddenly irretrievable—unless they are rendered durable through repeated use, which is the task of the *integration* stage of the methodology, described below.

The third native property shaping the discovery work in coherence therapy is the requirement for clinical methods to match the qualitative nature of the subcortical personal constructs driving symptom production. Therapeutic methods that fail to match the nature of the material are far less effective than those that do. Three major features characterize therapeutic methods that are well matched to the symptom-generating material:

- coherence-focused;
- experiential, empathic, noninterpretive, phenomenological; and
- noncounteractive.

Therapeutic methods are *coherence focused* by virtue of serving efficiently to find and integrate into awareness unconscious constructs that coherently require the production of the symptom (such as the constructs requiring Carol's aversion toward marital sex in the previous article [Toomey & Ecker, 2007]). The opposite of a coherence-focused method is a counteractive method (as discussed below and in the previous article), because to counteract is to disown rather than embrace the symptom-requiring material.

The symptom-requiring constructs are nonverbal, implicit knowings that were formed in response to experiences earlier in life that had intense subjective significance. The brain–mind–body system is functioning properly, not dysfunctioning, in holding such templates indefinitely, in activating any that match the present situation, and in launching the response that an activated schema knows is necessary (such as a Carol's suppression of sexual feelings). In order to efficiently find, reactivate, and access the prosymptom schema stored in implicit memory, methods of discovery have to use the fact that the content of the schema is coherently symptom-necessitating, because that is the only feature that distinguishes this schema from thousands of other schemas that the person holds in implicit memory. For example, Carol was asked by the therapist to say this sentence and allow it to complete

itself spontaneously: "If I were to like having sex with my husband, I'd feel -----." Like a key custom shaped to fit a unique lock, this seemingly simple step is, in fact, accurately designed to engage the coherence of the as-yet-unknown constructs necessitating Carol's specific symptom. It prompts Carol to sample imaginatively an experience of being *without* the symptom, which is highly likely to make constructs that urgently *require* the symptom become activated and responsive in a noticeable manner, as indeed occurred most fruitfully when "embarrassed" arose to complete the sentence. (This *symptom deprivation* tactic, here implemented in the form of a *sentence completion*, can be carried out in several other ways and is itself only one of many different coherence-focused methods of creating discovery experiences).

In addition to being coherence focused, methods of discovery need to have the interrelated qualities of being *experiential*, *empathic*, *noninterpretive*, and *phenomenological* in order for the client to reactivate, experience, and direct attention to the symptom-requiring material and translate the material accurately into explicit, neocortical representation. In coherence therapy, the client is guided to encounter and abide with the very material causing all the trouble, just as it is. This occurs to a degree and extent that is counterintuitive even for most depth-oriented psychotherapists. We offer here only a few comments on key aspects of these four related qualities, as a thorough treatment is beyond our scope.

While carrying out any experiential method, the therapist's style of language is a critical ingredient for its success. Verbal language that accurately attunes to, captures, and helps elicit unconscious emotional truth is quite different from ordinary social vernacular. The needed style is concrete, present tense, highly personal, and vividly descriptive of sensory perceptions; and it is utterly candid in naming what is at stake as well as the accompanying strong emotions, meanings, and urgency (Ecker & Hulley, 1996, 2004; Martin, 1991; Watson, 1996). Our earlier case vignette provides an example in the verbalization written on the card given to Carol at the end of her session. The verbalizing of emergent prosymptom positions is crafted to foster the client's inhabiting and embracing of the discovered material just as it is. Accordingly, it is unconcerned with adult properness, face saving, or notions of rationality and morality, and excludes all



styles of phrasing that would shift the client to an intellectual viewpoint.

The therapist's consistent empathy toward the emotional tableau in the prosymptom position is critically important for fostering discovery and accessing. As already noted, this tends to be vulnerable territory, and it will be safe to enter only in a climate of authentic empathy and acceptance. Furthermore, each emotional schema formed by the limbic system includes the person's developmental state of identity at the time of formation. This is why, when a therapy client is experientially inhabiting a prosymptom position, the altered state typically feels young, as noted earlier. The therapist's tone, empathy, and choice of words need to be attuned accordingly, in an authentic manner.

Giving the client interpretations, the in-depth therapy field's longstanding, mainstay technique, is ruled out because it is the antithesis of ushering the client into his or her own experiential discovery of the emotional truth of the symptom. It is also the antithesis of the therapist learning from the client how the symptom is necessary to have.

The use of *noncounteractive* methods was discussed in the overview of coherence therapy in the opening article of this series, but the importance of this point warrants a succinct reiteration here. Accessing the coherent, symptom-requiring material means heading toward it and staying with it. This cannot occur through counteractive methods, which attempt to oppose, prevent, fix, eradicate, avoid, override, weaken, or get away from the symptom or its underlying material. All such methods remove attention from, rather than bring attention to, the constructions *requiring* the symptom. Examples of counteractive methods of psychotherapy include positive thinking, self-soothing, reframing, rational correction of irrational beliefs, relaxation techniques, all standard protocols used by anger management therapists, behavior therapy, cognitive-behavioral therapy, and solution-focused therapy. For some therapists it is difficult to comprehend how, for instance, teaching an anxious client relaxation techniques is counteractive until they understand that while the attempt to induce relaxation is in concert with the client's antisymptom position (the wish to be rid of the anxiety), it is a direct attempt to counteract and override the prosymptom position's cogent necessity for *having* anxiety. To try to replace the symptom with a preferred state is always

counteractive, whether carried out cognitively, behaviorally, or chemically.

Many therapists know only counteractive methods, and to such practitioners, ruling out these methods might seem debilitating. However, what remains is the universe of noncounteractive, experiential methods, which is extensive, rich, and therapeutically potent. A large class of such methods involves a guided imaginal process in which the client has experiences that could not “really” occur in the therapist’s office. Such techniques, properly applied, are highly effective for accessing, because implicit schemas are activated in response to both real and imagined perceptions. Kreiman, Koch, and Fried (2000) recorded responses from individual neurons in the amygdala and other parts of the limbic system. They found that of neurons that fired in response to both the real (retinal) and imagined image of a particular object, but did not fire in response to other objects, 88% had identical receptive fields (the same set of objects to which they respond by firing, whether the image is imagined or real). On this result the authors commented, “The existence of a [neuron’s] selective response in the absence of concomitant retinal activity constitutes one of the strongest possible invariance properties of a representation that is correlated with perception” (Kreiman et al., 2000, p. 11). In other words, this is compelling evidence that “volition-induced imagined percepts share a common representation [with retinally generated images] in humans at the level of single neurons” (Kreiman et al., 2000, p. 204, footnote 67).

That neurodynamic finding confirms a key operational aspect of the discovery work in coherence therapy: The therapist structures visualizations and imaginal interactions designed to reactivate a specific prosymptom position, causing it to respond with internally noticeable manifestations that reveal more of the content of this symptom-necessitating schema. For example, it is most often fitting and fruitful to begin discovery work by guiding the client to imaginally revisit a recent, concrete situation in which the symptom or problem occurred (Ecker & Hulley, 2004). This in itself begins to reactivate the client’s prosymptom position, priming it for conscious eliciting. Although there are numerous other ways of creating discovery experiences, the usefulness and effectiveness of this tailored, imaginal type of discovery cannot be overstated.

## The Creation of Experiences That Integrate Prosymptom Positions

After the first experiential revelation of a prosymptom position, the next methodological aim is the stabilization of its availability as a conscious experience. This long-term stabilization of a new conscious knowledge is termed *integration* in the parlance of psychotherapy and is known as the *consolidation* of an explicit memory (encoded knowledge) in neuroscience. The process of consolidation has been recognized and studied for a century behaviorally, anatomically, biochemically, and neurally (Izquierdo & Medina, 1997; McGaugh, 1966, 2000).

As noted earlier, the synapses initially formed in discovery are nondurable. Clients often completely lose touch with newly conscious material that was lucid seconds earlier. Ecker and Hulley (1996, 2000a, 2004) have delineated the following built-in rules of the brain–mind–body system for achieving integration.

- A prosymptom position becomes part of the conscious personality through repeated, affective–cognitive–somatic experiences of it.
- Integration occurs through repeatedly relating to the problem *from* and *in* the prosymptom position requiring it (rather than from the antisymptom position initially expressed) and, while in the felt experience of this position, verbalizing all of the specific knowings, feelings, purposes, and tactics that constitute it in highly personal, declarative, adequately intense phrasing.
- The cost, hardship, or suffering due to having the symptom must be made sense of in relation to the discovered emotional truth of how and why the symptom is actually more necessary to have than not to have.

Repeated integration experiences install the prosymptom material increasingly firmly in the person's conscious world until it is robustly established there, no longer dissociates into unconsciousness, and is at any time readily conscious as a unified feeling-knowing and verbal-knowing of the emotional truth of the symptom.

Integration begins when the client's attention has for the first time gone away from the newly discovered material and then returns to it, reusing the new synapses linking the material to the neocortex. This first going away and coming back of attention often happens unnoticeably, soon after the initial discovery experience: The client has already lost track of the first prosymptom fragment but then finds it again upon hearing the therapist make comments that refer to it. This first coming back also can occur when the therapist first structures an integration experience, perhaps simply by asking, "Please tell me again what you just discovered," or by asking, for example, "Would you be willing to picture your brother and say directly to him what you just got in touch with?"

These in-session repetitions of accessing the prosymptom material are followed by a structured, between-session task of daily reexperiencing the material, such as Carol's daily reading of the index card as a cue to internally reexperience her prosymptom position as a felt emotional truth. At the start of the next session, the therapist assesses the degree of integration achieved and continues to create integration experiences if and as needed, as well as further discovery experiences, until the entire prosymptom position is routinely conscious.

The essence of this ongoing series of integration experiences is the client's repeated, subjective act of bringing neocortical attention into the initially subcortical prosymptom material. From the neurodynamic standpoint, we may reasonably assume that integration occurs because this repetitive, attentional use of the new synapses linking neocortex to prosymptom implicit memory strengthens both these linking synapses as well as the synapses that now encode the neocortex's own new representation of the prosymptom position.

It is plausible that two different synapse-strengthening mechanisms are recruited by integration experiences.

First, Hebb's (1949) basic rule applies: A synapse strengthens if the neurons it connects repeatedly fire together and weakens if they do not. In integration experiences, we have the repeating, joint firing of the neocortex's synaptic encoding of the prosymptom position, the subcortical synaptic encoding of it, and the synapses that link the two (which involves other brain systems, as noted earlier).

Second, because the contents of prosymptom positions always have strong subjective importance, each integration experience is, in itself, an emotional experience and therefore activates the hormonal system of emotionally enhanced memory encoding. Emotional arousal releases adrenal stress hormones that enhance hippocampal encoding of explicit memory of the experience (Cahill & Alkire, 2003; McGaugh & Roozendaal, 2002). Events that evoke emotional responses are likely to be important for survival and so are more important to remember, which the hormonal mechanism promotes.<sup>5</sup> The memory being enhanced in this way during an integration experience consists of the content of the prosymptom position. That is, integration itself is enhanced by the hormonal mechanism.

The Hebbian and the hormonal mechanisms would make the transient synapses created in discovery experiences undergo a molecular and cellular transition to a more stable, enduring state of potentiation. Neuroscientists have studied the long-term potentiation (LTP) of synapses intensively for 30 years, relying heavily on tests of excised groups of neurons in cell cultures. These studies of the artificial, *in vitro* stimulation of LTP (and of LTD, long-term depression or depotentiation) have identified numerous molecular mechanisms of synaptic strengthening and weakening. The dependence of long-term memory *in vivo* on specific molecular processes detected in LTP *in vitro* was demonstrated initially by pharmacologically blocking the *in vivo* functioning of a specific type of synaptic receptor known to be critical to LTP, and observing a resultant incapacity for long-term memory formation in animals (Kentros et al., 1998; Morris, Anderson, Lynch, & Baudry, 1986; see also reviews by Bailey et al., 2004, and Kandel, 2001).

However, until 2006 there was no proof that the synaptic states formed by LTP *in vitro* are the same synaptic states that are responsible for consolidation—the durable encoding of memory, the persistence of knowings—in the brains of living animals. Two studies have finally produced the long-awaited evidence (Pastalkova et al., 2006; Whitlock, Heynen, Shuler, & Bear, 2006). We will therefore now take the liberty of using LTP to refer to the enduring potentiation of synapses *in vivo* (whereas previously the term usually denoted the *in vitro* case).

Achieving stable integration of a prosymptom position means, then, that implicit, subcortical, emotional knowings have been translated and consolidated into stable, explicit, verbal–conceptual neocortical knowings. The person now readily experiences both kinds of knowing jointly—subcortical (and right cortical) feeling-knowing and neocortical verbal-knowing. These emotional and verbal structures are now firmly linked neurally, so that a reactivation of the affective material also reactivates the verbal–semantic knowing of this material, and vice versa.

As noted previously, it is well established that the hippocampus, a component of the limbic system, carries out the consolidation of explicit memory (although little as yet is known about how it does this). We may therefore reliably infer that integration experiences recruit the hippocampus to translate the prosymptom position from its implicit, subcortical form into explicit form in the neocortex.

In each integration experience, the person focuses attention on and into his or her symptom-requiring position, in order to inhabit and experience it. This directing of sustained, subjective attention to the prosymptom material is the critical action taken by the person in this methodology. The neurodynamic correlate of this key phenomenological element would consist of a mechanism by which subjective attention drives lasting synaptic potentiation and memory consolidation.

This mechanism was identified quite recently. By recording the firings of individual neurons *in vivo* in the hippocampus of freely behaving mice, neuroscientists studying LTP were surprised by the unexpected finding that an animal's subjective attention toward an emotionally important feature in its environment itself triggered LTP, forming long-term memory (Kandel, 2001; Kentros et al., 2001).<sup>6</sup> Further study led Kentros et al. (2004) to “propose a model whereby attention provides the requisite neuromodulation to switch short-term homosynaptic<sup>7</sup> plasticity to long-term heterosynaptic<sup>8</sup> plasticity” (p. 283). These authors infer that hippocampal synaptic plasticity, in particular the establishment of LTP, “is governed by a higher-order cognitive process. . . . We define selective attention operationally as the cognitive processes resulting from the animal assigning significance to [specific features] of its sensory experience” (p. 291).

This demonstration by neuroscientists of the potent role of attention in driving synaptic plasticity is a significant corroboration and correlate of coherence therapy methodology. In more recent work, the critical influence of subjective attention on neurodynamics was strikingly demonstrated by recording the firing of single neurons in the visual cortex of an alert macaque monkey as the monkey was searching among many objects for one with a specific feature, as it had been trained to do (Bichot, Rossi, & Desimone, 2005, reviewed by Wolfe, 2005). Each recorded neuron was responsive only to a certain spot on the retina, off to the side of the visual field. If a neuron's receptive field for color preferred red stimuli, then that neuron was found to respond more vigorously whenever a red object happened to be at that spot in the peripheral visual field. When in addition the monkey had been trained to search for a red object, that red-sensitive, off-center neuron produced a still larger response, even when the monkey's next eye movement was not directed to the red object. The red item had not yet become the focus of conscious attention, but the response of the neuron still received a boost because red was the color being consciously sought. This demonstrates that the monkey's attention—in this case, imaginal attention in the form of the intention to find a red object, perhaps in the form of holding a mental image of redness—had enhanced the synapses governing the firing of that red-sensitive neuron.

These studies show the effectiveness of focused attention in creating changes in synapses and, therefore, in the knowings that synapses encode. We have so far considered discovery and integration, in which attention is used to create new synapses and to strengthen and potentiate them. When we come to transformation (in the next section), we will see how attention is used in coherence therapy to turn off—depotentiate—the specific synapses that are driving symptom production.

It is significant, with regard to integration, that parts of the conscious, left-brain attention system also play a crucial role in language use (Frishkoff, Tucker, Davey, & Scherg, 2004). This means that verbalizing inherently recruits attention toward the experience being verbalized. This is presumably why the verbalizing of prosymptom knowings is so strongly facilitative of their integration.

Prior to integration, the person's conscious, neocortical construction of what it means to have the symptom is the anti-symptom position—the view of the symptom as entirely undesirable, senseless, valueless, involuntary, egregious, and pathological. However, as the neocortex, during integration, pays attention to the implicit emotional truth of how and why the symptom is urgently necessary to have and adopts this understanding of the symptom and forms its own rendition of it, the antisymptom constructs are largely dissolved. From the neuropsychological perspective, integration in coherence therapy can be understood as the consolidation in the neocortex of an encoded representation of the prosymptom knowings held in emotional implicit memory. In other words, the neocortex becomes informed by and aligned with the subcortical prosymptom knowings. This result—the aligned state of neural integration and shared, multimodal knowledge between these brain systems—is a principal aim of any neurobiologically informed psychotherapy, according to a consensus of experts (see, for example, Panksepp, 2004; Schore, 2003; Siegel, 1999). Affective neuroscientist Panksepp, for example, stated that the aim of “healthy development is to generate harmonious, well-integrated layers of emotional and higher processes, as opposed to conflicts between [the] emotional and cognitive” (2004, p.132).

Of course, the symptom still entails hardship or suffering that continues to be disliked by the person, but now he or she is lucidly aware of harboring the expectation of an even worse suffering *without* the symptom. For example, Carol knew, once her prosymptom position was integrated, that part of her truly expected that if she no longer stifled her sexuality, she would cause her own daughter the same terrible suffering that had been inflicted on herself as a girl. She knew, at this point, that the marital unhappiness caused by her shunning of sexuality was, for a part of her, a price worth paying to avoid that even worse suffering.

This clear recognition by the person of how and why the symptom is more necessary to have than not to have, even with the suffering it causes, is the goal of the integration work. It is referred to as the *pro/antisynthesis* (Ecker & Hulley, 1996, 2004), meaning simply that there is no longer a split or dissociation between the prosymptom knowings of why the symptom is necessary and



the antisymptom wish to be rid of the symptom because of the suffering it entails.

The observed timescale of integration agrees closely with the timescales of synaptic change, providing another important neurodynamic corroboration of coherence therapy's methodology. As noted earlier, transient activation of synapses occurs in milliseconds in response to a new experience. First discovered in 1951 (Fatt & Katz, 1951), the neurotransmitters for this fast mechanism are simple ions. This fast activation can segue into a second form of synaptic potentiation, lasting minutes and using more complex neurotransmitters (Greengard, 1976). A third, more persistent synaptic action, lasting days and using modulatory transmitters (such as serotonin) applied to principal neurons by interneurons, was then discovered.

The fourth and longest-lasting class of mechanisms, collectively called long-term potentiation or LTP, allows synapses to remain in force from weeks to decades. LTP was discovered in the early 1970s (Bliss & Lomo, 1973) and began to be understood at the molecular level in the 1980s. The highly complex intra- and interneural biochemistry that carries out the molecular as well as structural changes involved in LTP is now known to a significant degree (Bailey et al., 2004; Kandel, 2001; Malenka & Bear, 2004). The molecular details of synaptic and neuronal functioning are nearly the same in fruit flies, mollusks, and mammals (Kandel, 2001). The stable integration/consolidation sought in coherence therapy by definition recruits LTP.

Synapse studies have shown that LTP can occur immediately in response to a single, vivid experience that has strong subjective significance, or it can occur after a less intense experience is repeated sufficiently over time. There are several different molecular pathways that can bring it about, and these are used differently by different types of neurons. The most durable, "late-stage" LTP involves synaptogenesis, the structural growth of new physical synapses, which have been directly observed visually at 24 hours after LTP-triggering stimulation (Knott, Quairiaux, Genoud, & Welker, 2002, reviewed in Zito & Svoboda, 2002). In one such study, at 24 hours two-thirds of the newly functioning synapses had been newly created and one-third were activations of previously inactive synapses (Kim et al., 2003). Several weeks are required for the synapses encoding a new explicit memory

to reach final, maximum stability as long-term memory (Milner, Squire, & Kandel, 1998). Spatial analyses of dendrites and axons in the brain have concluded that huge numbers of potential synapses are always available for the creation of new long-term synapse patterns (reviewed in Chklovskii, Mel, & Svoboda, 2004; whether or not the adult brain can also create new whole neurons for the synaptic encoding needed in response to experience is a controversial question at present, with contradictory indications provided by different studies).

The observed range of timescales for synaptic plasticity closely fits the phenomenology of integration regularly observed in carrying out coherence therapy. The manifestation of all three short-term plasticity timescales is distinctly apparent: Therapy clients in early stages of integration sometimes lose all awareness of a clearly experienced prosymptom position after seconds, minutes, or days, which prompts the therapist either to again create integration experiences that restore and further strengthen the new neocortical connections and memory traces or to probe for and work with resistance to integration (Ecker & Hulley, 1996, 2004), the neural correlate of which is presumably the presence of a superordinate, inhibitory neural network.

On the other hand, some therapy clients display a firm degree of integration—a routine, conscious experiencing of the prosymptom position—in their next session one week later, which is more than enough time for the strongest type of long-term potentiation to be underway. The primary indicator of stable integration is the client articulating the living experience of the prosymptom material in its specifics, on his or her own initiative, with no reminder from the therapist (Ecker & Hulley, 2004). The therapist actively invites this articulation and probes for degree of integration by asking, for example, “How was it to live day to day with what we put on that card, last time?” or “What we put on the card seemed to feel like real emotional truths at the time. Did it continue to feel that way?”

### **The Creation of Experiences That Transform Prosymptom Positions**

The goal in coherence therapy is a transformation (either a major restructuring or a complete dissolution) of each of a client’s

prosymptom positions maintaining a given symptom, such that there no longer exists any constructions of reality in implicit memory in which the symptom is necessary to have. As soon as there no longer exists any prosymptom positions, the immediate cessation of the symptom is highly predictable, based on extensive clinical observations (Ecker, 2003, 2005; Ecker & Hulley, 1996, 2000a, 2002a, 2002b; Martignetti & Jordan, 2001; Neimeyer, 2000; Neimeyer & Bridges, 2003; Neimeyer & Raskin, 2001; Thomson & Jordan, 2002).

The discovery work reveals prosymptom positions phenomenologically. The integration work renders a prosymptom position readily available to conscious attention and experiencing. No longer an insulated, autonomous capsule of constructs, an integrated prosymptom position is susceptible to immediate transformation. Exactly how that depotentiation process then operates is the subject of this section, including the important matter of how depotentiation is verified.

A fundamental distinction is made in coherence therapy between *transformational change* that actually alters or dissolves a prosymptom position and *counteractive change* that merely competes against fully intact prosymptom position(s) with the aim of overriding, avoiding, or managing symptom production. The difference between transformational change and counteractive change is particularly clear when understood at the neural level.

The neural process in counteractive change is epitomized by the classical extinction of a Pavlovian conditioned response. In animal studies, when an unconditioned stimulus (US, such as a mild, harmless but unpleasant electric shock) is repeatedly accompanied by a conditioned stimulus (CS, such as an auditory tone), the result is an aversive conditioned response (CR) when only the CS is presented. An acquired fear memory of the CS-US pairing has formed, which is encoded neurally in the amygdala (Phelps & LeDoux, 2005). (The conditioned response to the tone corresponds, we suggest, to a client's symptom, and the acquired memory to a client's prosymptom position.) Extinction of the conditioned response is then brought about by repeatedly presenting the CS (tone) without the US (shock) until the CR (aversive behavior) no longer occurs. It is rigorously established that the neural mechanism of this extinction is not an alteration,

erasure, or forgetting of the acquired memory created by the first training, but is, rather, a second, separate learning that the tone is unimportant (Bouton, 2004; Myers & Davis, 2002; Rescorla, 2001). The nonaversive response learned second counteracts and overrides the aversive response learned first, but both learnings are still held in memory.

The continued existence of the acquired fear memory even after full extinction is graphically demonstrable through several different methods of reevoking the conditioned response after extinction and without retraining (Rescorla, 2001). For example, by simply positioning the subject in a different environment (context) than the one in which the extinction was carried out, the conditioned response (fear behavior) occurs again following presentation of the conditioned stimulus (tone), showing that the acquired memory still exists (and that extinction-learning is context-dependent). Even in the original context, spontaneous recurrence of the conditioned response may occur after extinction, particularly if other stressors are present. The CR can also be strongly reevoked by a brief reminder of the original training (presentation of one or two CS-US pairings), which is far too weak in itself to establish a conditioned response.

The extinction procedure is the explicit prototype upon which behavior therapy is based. The neurodynamics of extinction—the creation in memory of a separate, new, dominant knowledge structure—would seem to be the underlying neural mechanism of all counteractive methods of change in therapy, that is, all methods designed to prevent symptom production by forming a new, symptom-free state, without having actually transformed the implicit knowledge structures responsible for symptom production.

Whereas conditioned fear responses are consolidated into long-term memory in the basolateral amygdala (Maren, 1999a, 1999b), mounting evidence indicates that extinction learnings are stored in the medial prefrontal cortex (mPFC), which has modulatory connections to the amygdala (Phelps, Delgado, Nearing, & LeDoux, 2004; Quirk, Likhtik, Pelletier, & Pare, 2003). There is also evidence that an extinction learning's initial, short-term encoding occurs in the amygdala, followed by transfer to the mPFC for long-term memory or consolidation (Milad & Quirk, 2002).

We suggest that, like extinction training, counteractive therapeutic methods do not directly depotentiate the brain's neural circuits driving symptom production but, rather, create new learnings (synapse changes) in other regions that compete against the symptom-generating circuits.

For example, standard cognitive-behavioral therapy (CBT) relies heavily on the cultivation of neocortical thoughts designed to counteract unwanted emotional reactions and moods. It has been shown using fMRI brain imaging that such counteractive thoughts during moderate (photograph-induced) amygdalar reactions of fear do recruit the medial prefrontal cortex to partially suppress amygdalar activation (Kalisch et al., 2005; Ochsner et al., 2004).

With that counteractive approach, the amygdalar fear response circuits remain fully intact, the fear response needs to be managed in that cognitive manner indefinitely, and relapse can occur at any time. Furthermore, among therapy clients with presenting symptoms of anxiety or panic, the fear is far more intense than that induced in subjects in the studies just cited, and as any experienced therapist knows, cognitive antidotes for raw fear are not strongly effective. (To our knowledge, the efficacy of CBT has not yet been demonstrated to surpass that of a rigorously designed placebo therapy, that is, a placebo that is structurally equivalent to the active treatment. See for example the meta-analysis of Baskin, Tierney, Minami, and Wampold [2003], which examined 21 efficacy studies, 14 of which were CBT studies.)

If further research confirms that many or all other counteractive methods of psychotherapy operate through suppression of subcortical centers of activation by the mPFC or other brain centers, this would mean that counteractive methods are not an optimal use of neuroplasticity according to our definition, because the brain region and the implicit memory circuits actually responsible for symptom production are not made to undergo potent synaptic change. Such extinction-like methods are broadly vulnerable to recurrence of symptoms, particularly when the person is in new contexts or under stress, or when a reminder (a conditioned stimulus, or trigger) occurs, all of which routinely happen in day-to-day life.

Yet psychotherapy as currently practiced consists mainly of counteractive methods. These have many different procedural

forms, which may also differ in how they recruit various brain systems so as to suppress (but not transform) implicit schemas driving symptom production in the amygdala or elsewhere. Despite such differences, the counteractive strategy is the same. This reliance on a strategy having a neurobiologically limited effectiveness may prove to be a major reason why all such therapies are consistently found to have essentially the same modest level of efficacy—the well-known “Dodo bird verdict” (see, e.g., Luborsky et al., 2002; Stiles, Barkham, Twigg, Mellor-Clark, & Cooper, 2006; Wampold et al., 1997).

In contrast to the counteractive strategy, the aim in coherence therapy is always a transformative alteration or dissolution of prosymptom positions—a profound depotential of emotional implicit memory such that symptom production is radically ended and no longer can reoccur.

The robust stability over time of prosymptom implicit memories is impressive. Left alone, they remain perfectly intact through hundreds or thousands of reactivations spanning most of a century. During most of those reactivations, the perceived situation does not unfold in the dire manner predicted by the stored schema, yet extinction does not occur. As an example, consider a 45-year-old person with an unconscious, prosymptom position that consists of the knowing that hearing another’s angry voice means “I’m going to be hit” and brings corresponding bodily and emotional symptoms of anxiety, with no awareness of the reason for these responses. For over 30 years, getting hit has never happened after hearing such a voice hundreds of times. Yet, no extinction of this unconscious fear memory has occurred.

This immunity of prosymptom positions to extinction could result from two different mechanisms of which we are aware. The first was recently identified in animal studies of the time dependence of extinction learning (Cain, Blouin, & Barad, 2003). Twenty-four hours after rats were conditioned to fear a sound that had been paired with a mild foot shock, the sound was presented repeatedly without the shock. If the time period between repeated shock-free sounds was 5 seconds, strong, rapid extinction of the fear occurred, but if the period was 20 minutes, there was no extinction for most rats, and some became even more fearful of the sound. A duration of 20 minutes is presumably long enough,

for a rat, for each presentation of the sound to be experienced as an isolated, single occurrence that functions more strongly as a reminder, a reevoking of the conditioned fear, than as an extinction learning. (Cain, Blouin, and Barad [2004] subsequently confirmed that the widely spaced, shock-free sounds induce two separate brain responses—the conditioned fear response and an extinction learning—but the latter is subordinate to the former.) The environmental stimuli that reactivate a person's prosymptom position likewise typically occur occasionally, and so do not produce extinction learning.

A second mechanism that prevents extinction of prosymptom positions is, we propose, the highly anticipatory and proactive nature of human construing (Kelly, 1955), which plays out in the following manner. Every prosymptom position includes not only a knowledge or model of a particular type of harm or jeopardy—how to spot its presence and how it operates—but also a procedural knowledge of a specific tactic for protecting oneself from that harm, a tactic that is implemented immediately upon reactivation of the prosymptom position (entailing production of the presenting symptom). When the harm or jeopardy is perceived to be imminent but then does not actually occur, the meaning that is implicitly construed for this is that the protective tactic worked, not that the jeopardy did not really exist, which is the meaning that would produce extinction learning. In that way, extinction learning in the natural course of living is ruled out by the proactive, protective purpose built into every prosymptom position.<sup>9</sup>

That intrinsic resistance to change through extinction is the background against which we now turn to the process that does change these emotional schemas held in implicit memory. We will describe the built-in rules and procedures of the change process, first in coherence therapy's phenomenological terms and then in terms of the neural mechanisms plausibly recruited by those procedures.

### *How Constructs Change: The Phenomenological View*

The native process of the brain–mind–body system for transformational change of personal constructs has been delineated by

Ecker and Hulley (1996, 2000a, 2004) and Ecker (2005, 2006) as follows:

- The brain–mind–body system allows the coexistence of any number of contradictory, incompatible constructions of reality (knowings, schemas, models, ego states, parts), as long as the contradictory constructions are held separately, never enter the same field of attention, and never are consciously experienced together.
- The brain–mind–body system does not tolerate an incompatibility or ontological contradiction between disparate constructions that are juxtaposed and experienced together in the same field of attention. The attentive experiencing of juxtaposed, incompatible constructions, where both are *experienced* as real yet both cannot possibly be true, is a violation of coherence and is therefore the precise condition under which the brain–mind–body system immediately uses its capability to dissolve one of the two incompatible constructions, ending the presence of an ontological contradiction.
- The more limiting and antiquated of the two incompatible, juxtaposed constructions is disconfirmed by the other and is dissolved and ceases to have subjective realness.
- The brain–mind–body system has the capability of dissolving (erasing) any of its previously formed personal constructs at any time, but does so only in response to an *experience* (as distinct from mere conceptualization) of disconfirming juxtaposition.

Simply put, for transformation of the nonverbal, implicit knowledge or prosymptom position driving a therapy client's symptom, the person must (a) attentively reactivate and experience that prosymptom position and (b) alongside and along with it concurrently experience another living knowledge that is logically incompatible with it and disconfirms it—a simultaneous experiencing of the two knowledges juxtaposed in the same field of attention, and both cannot possibly be true.

This process differs fundamentally from extinction learning, which merely competes against but does not alter prior learnings. It recruits the brain–mind–body system's native ability to actually modify or dissolve the reactivated constructs (Ecker & Hulley, 1996, 2000a, 2004). Examples and evidence follow later in this article.



*How Constructs Change: The Neurological View*

The corresponding neural picture is perhaps best understood by considering the neurodynamic life history of a memory. As noted, a newly created knowledge structure achieves a state of long-term sturdiness in memory through a complex neural process known as consolidation, which has several phases, takes weeks to complete, and involves an interaction of specialized brain regions (Alberini, 2005; Izquierdo & Medina, 1997; McGaugh, 1966, 2000). Once consolidated, an implicit long-term memory such as a prosymp-  
tom position consists of a set of synapses that are molecularly locked into an indefinitely enduring, potentiated state by certain proteins manufactured on demand by neural genes.

It was canonical knowledge of neuroscience throughout the twentieth century that once a conditioned response is consolidated in the amygdala's long-term implicit emotional memory, the locking of the synapses is permanent and the encoded memory is unchangeable. The evidence for that indelibility came from studies of animal learning—specifically, the fact that a subsequent *extinction* learning overrides the original conditioned response but does not alter or erase it. Even after complete and successful extinction, the original aversive, fearful response can be reevoked in a number of easy ways, which shows that the initial conditioned, implicit memory continues to exist and was not changed by the extinction training. The latter forms a separate, second learning that competes against, but does not change, the original conditioned response (see, e.g., Bouton, 2004; Milad & Quirk, 2002; Milner, Squire, & Kandel, 1998; Myers & Davis, 2002).

Extinction appeared to be the best one could possibly do to put an end to a response driven by conditioned emotional learnings. One had to create other learnings that successfully counteract and override the unwanted response. That counteractive strategy and mechanism is the basis of behaviorism and of cognitive-behavioral therapy. It was fully recognized that, although this seemed to be the only viable strategy for getting a conditioned response to cease, extinction learning is not highly reliable: A reactivation and relapse can occur at any time because the original learning is still present in memory.

However, from 1968 to 1982 a handful of exceptions were published, observations of a conditioned response disappearing

("retrograde amnesia") as a result of special laboratory procedures (Judge & Quartermain, 1982; Lewis & Bregman, 1973; Lewis, Bregman, & Mahan, 1972; Mactutus, Riccio, & Ferek, 1979; Misanin, Miller, & Lewis, 1968; Richardson, Riccio, & Mowrey, 1982). These anomalies received little attention, and it was another 15 years before researchers revisited the matter. Then, from 1997 to 2000 several articles were published reporting strong new evidence of a type of neuroplasticity, or synaptic change, that is capable of directly revising long-term implicit memory and the conditioned responses that it generates (Nader et al., 2000; Przybylski & Sara, 1997; Roullet & Sara, 1998; Sara, Roullet, & Przybylski, 1999; Sekiguchi, Yamada, & Suzuki, 1997). The recognition of this neural capacity, termed *reconsolidation*, meant that a century of regarding amygdalar memory as indelible was turning out to be wrong. Reconsolidation rapidly then became a focus of study in many neuroscience laboratories.

This dramatic reversal corroborated the phenomenological finding of Ecker and Hulley (1996, 2000a, 2004) that a true depotentiation of long-term emotional implicit memory is observed to occur during coherence therapy. In addition, as described later in this article (and by Ecker, 2006), the procedural details of how this neural mechanism of depotentiation gets activated match the steps of the native process of phenomenological depotentiation identified by Ecker and Hulley.

The fact that the amygdala's implicit memory is changeable after all means that the counteractive strategy of extinction learning is not necessarily the most effective strategy for changing conditioned responses. Actually eliminating a conditioned response would be a more effective and reliable change than merely counteracting it. The implications for psychotherapy are revolutionary. However, reconsolidation was discovered in animal studies in the laboratory using specialized biochemical and behavioral methods, and neuroscientists do not yet know how to induce reconsolidation selectively, safely, and ethically in people. Nevertheless, the clinical evidence described in the next subsection suggests that coherence therapy methodology may already be bringing about reconsolidation in people. In this subsection we briefly review how reconsolidation operates in neural terms.

In the course of experience, when an existing, consolidated, long-term knowledge structure is called into active use by retrieval

and reactivation, under certain conditions a process can occur in which the constituent, locked synapses are molecularly unlocked temporarily and become labile and modifiable, which allows for an experience-dependent alteration (either strengthening, weakening, or revision of content) in the memory's model of reality and in the responses that it generates. Upon deactivation of the memory after the reactivating experience ends, the synapses are relocked, now in their newly altered configuration (Alberini, 2005; Nader, 2003; Nader et al., 2000; Sara, 2000; Walker et al., 2003). This is the reconsolidation of the memory.

Comments in recent research papers sum up current knowledge concerning memory labilization and reconsolidation: Reviewing studies by Debiec, LeDoux, and Nader (2002), Nader (2003, p. 69) stated that "even remote memories, when triggered . . . return to a labile . . . state, and . . . they remain in this state for several days." Frenkel, Maldonado, and Delorenzi (2005, p. 1757) stated, "A considerable body of evidence reveals that consolidated memories, recalled by a reminder, enter into a new vulnerability phase during which they are susceptible to disruption again." Suzuki and associates (2004, p. 4787) stated,

Memory retrieval is not a passive phenomenon. Instead, it triggers a number of processes that either reinforce or alter stored information. . . . Memory reconsolidation after retrieval may be used to update or integrate new information into long-term memories (LTMs). . . . The finding that LTM may be more dynamic and plastic than previously thought may have important clinical implications for the treatment of emotional disorders.

Alberini (2005, p. 54) stated, "the stronger the reactivation of the learned experience, the more labile the memory becomes."

These studies documented reconsolidation of implicit memory of aversive, fear-based conditioned learning, stored in the amygdala. In addition, reconsolidation has so far been documented for two other, nonamygdalar memory systems in the brain: the appetitive, food-seeking memory of honeybees (Stollhoff, Menzel, & Eisenhardt, 2005) and the hippocampal, spatial memory of rats (Rossato, Bevilaqua, Medina, Izquierdo, & Cammarota, 2006). The occurrence of reconsolidation across the brain's memory systems is noteworthy because it means our

hypothesis that coherence therapy recruits reconsolidation does not stand or fall on where in the brain prosymptom positions are stored in implicit memory. That hypothesis, of course, remains to be verified by neuroscientific methods.

It seems necessary to understand reconsolidation in terms of both subjective experience and neural and molecular processes, with the latter driven by the former. This is made apparent perhaps most clearly by the research of Pedreira, Perez-Cuest, and Maldonado (2004). These authors have demonstrated that what induces the labilization (mutability) of a conditioned memory's encoding synapses is not memory reactivation alone but the experience, during reactivation, of a decisive mismatch between what is expected according to the reactivated memory's model of reality and what is perceived to actually exist. (Likewise, reactivation alone, without a mismatch experience, did not produce synaptic reconsolidation in a study by Cammarota, Bevilaqua, Medina, and Izquierdo [2004].) This finding matches well with what coherence therapy defines as the native process through which implicit constructs get transformed, as described in the previous subsection—the experience of a disconfirming juxtaposition, a definite mismatch between what is expected according to a reactivated prosymptom position and according to some other, incompatible living knowledge.

Of course, extinction also results from experiences that mismatch what is expected. The neural research has shown that the duration and/or total number of disconfirmation experiences determines whether extinction or reconsolidation result. For example, Pedreira and Maldonado (2003) demonstrated in animal studies that when reexposure to a conditioned stimulus ended after a relatively short time (5 to 40 minutes) without the occurrence of the expected unconditioned stimulus, the experience functioned as a reminder and induced synaptic labilization and reconsolidation of the original memory (merely preserving the original memory in this case). In contrast, reexposure for a longer time (1 or more hours) instead induced extinction (the formation of second, separate memory). Subsequent studies of various species have confirmed this finding that experiences of disconfirmation (mismatch) produce either reconsolidation or extinction—two very different neurological processes—depending on the temporal structure of the

experience (Pedreira et al., 2004; Rossato et al., 2006; Stollhoff et al., 2005; Suzuki et al., 2004).

It is reasonable to expect that the same will be found true for humans, because neural mechanisms are highly conserved across phyla. We will propose in the next subsection, however, that in humans there is, in addition, a unique, third type of result that can follow from a disconfirmation experience, a result produced by the human capacity for bestowing attention in an inclusive, integrative manner that animals appear to lack.

### *An Endogenous Process of Reconsolidation*

Coherence therapy's methodology of transformation strongly fulfills the condition described above for synaptic labilization of a symptom-requiring implicit memory. The methodology creates for a client the following interrelated experiences:

1. deep, explicit reactivation: attentive, lucid experience of a reactivated prosymptom position with full emotional arousal plus verbalization;
2. concurrent lucid experience of a contradictory, emotionally vivid knowledge;
3. decisive experience of the prosymptom position's model of reality as making incorrect predictions and/or attributions of meaning; and
4. decisive experience of the contradictory knowledge as a correct alternate model of reality.

Experiences 1, 2, and 3 fulfill the condition for labilization of the prosymptom, implicit memory. Experiences 3 and 4 guarantee that the prosymptom position is subjected to a massive weakening, alteration, or dismantling. Depth and intensity of subjective experiencing maximize the effectiveness of each step of this process.

Ecker and Hulley's (1996) formulation of the rule of transformation through disconfirming juxtaposition received significant support from memory experiments conducted neurodynamically and biochemically by Frenkel and associates (2005), who concluded that their "present results constitute the first [neural] evidence that memory can be positively modulated during

**TABLE 1** The Process of Transformation of Knowledge Structures in Implicit Memory

Neurological process (Reconsolidation studies)	Animal learning process (Behavioral studies)	Human phenomenological process (Coherence therapy)
<b>1. Reactivation</b> Firing of the synaptic circuit that encodes the memory	Memory reactivation via presentation of conditioned stimulus	Subjective immersion in prosymptom constructs (discovery/integration work)
<b>2. Labilization</b> Synapses are unlocked and rendered labile by a disconfirmation experience	Termination of conditioned stimulus with no unconditioned stimulus <i>after short period</i> (long period triggers extinction, not reconsolidation)	Experience of an incompatible living knowledge
<b>3. Mutative influence</b> Alters synapses, strengthening, weakening, or remodeling the memory	Blockade or enhancement of molecular pathways by pharmaceuticals, endogenous biochemical effects, or electroconvulsive shock	Attentional, experiential juxtaposition of prosymptom position and incompatible knowledge
<b>4. Reconsolidation</b> Locks in the altered synapses	Irretrievable disappearance of conditioned response or permanent amnesia for a task	Symptom cessation; prosymptom constructs seem unreal and are non-evocable by former triggers

reconsolidation through ... a real-life episode. ... We propose that during reconsolidation it is possible to modify memory strength by the influence of a concurrent experience.” Note that Ecker and Hulley’s “juxtaposition experience” qualifies as a “concurrent experience,” as meant by Frenkel et al. This recent neural discovery that so closely aligns with Ecker and Hulley’s phenomenologically defined rule for transformation is a major point of unification between the neural and phenomenological domains.<sup>10</sup>

The possibility that coherence therapy recruits reconsolidation garners further plausibility in the close correspondences indicated in Table 1 (from Ecker, 2006). The left column de-

scribes the sequence of steps in the neurological process of reconsolidation. The right column describes the corresponding, endogenous/experiential steps in the methodology of coherence therapy. The middle column supplies the steps used in animal studies of reconsolidation. Of course, these correspondences do not constitute proof that reconsolidation is brought about by coherence therapy. However, when taken together with even stronger evidence—the inability of strong reminders (former triggers) to reevoke prosymptom schemas after transformation, as described in the next subsection—our reconsolidation hypothesis seems to merit consideration.

Coherence therapy, first presented to an audience of psychotherapists in 1993, functions entirely as a methodology for accurately finding and transforming the specific implicit memories driving the production of a specific presenting symptom. Now neuroscientists, as a result of discovering the memory-altering neural process of reactivation, labilization, modification, and reconsolidation, also recognize the native capacity of the brain–mind–body system for the selective depotentiation of implicit memory. Among neuroscientists the search is on for a suitable treatment protocol for humans that will reliably depotentiate a reactivated memory while it is temporarily labile. An editorial in the *British Journal of Psychiatry* concludes,

The clinical value of the [research findings on reconsolidation] remains controversial; can a memory trace be removed individually without unwanted disruption to other memories? Is the web of connections in human memory too extensively interlinked to allow for the possibility of therapeutic erasure? (Morrison, Allardyce, & McKane, 2002, p. 197)

The memory depotentiation methods under consideration by neuroscientists are exogenous, such as drugs that block molecular processes necessary for memory reconsolidation or electroconvulsive shock, which has been shown to disrupt a reactivated, labilized memory in many animal studies and one human study, described below.

In contrast, we hypothesize that coherence therapy produces memory depotentiation through endogenous, natural processes of human attention and experiencing. To our knowledge, coherence therapy is the only paradigm of psychotherapy with a

methodology that matches, in detail, all steps needed for bringing about the native neural process of reactivation–labilization–modification–reconsolidation.

During symptom production in the course of living, the responsible implicit memory (prosymptom position) is reactivated, but unconsciously so. It is at such moments that exogenous treatment schemes would intervene—that is, during symptom production, while the prosymptom memory is unconsciously reactivated.

In contrast, coherence therapy's discovery and integration stages turn that unconscious reactivation into a fully felt, fully conscious experience of the specific content of the prosymptom memory, which is a much stronger and more complete reactivation of that memory. The integration process in particular entails keeping attention directly on and in a prosymptom position repeatedly or even continually for many days or weeks.

To our knowledge, the only previous report of a clinical use of reconsolidation describes the effects of applying electroconvulsive shock (ECS) to subjects during florid reactivations of hallucinations or symptoms of obsessive-compulsive disorder (Rubin, 1976; Rubin, Fried, & Franks, 1969). This differs sharply from the standard ECS procedure, in which the patient is anaesthetized and unconscious when the ECS is administered. Rubin's procedure was based on prior animal studies in which reconsolidation was first discovered (Misanin et al., 1968) by applying ECS during reactivation of a long-term memory, resulting in significant weakening of the behavioral expression of that memory, whereas ECS applied when the memory was not reactivated had no such effect.

Rubin surmised therefore that while a person is producing the symptom, the underlying, implicit memory driving its production is in a state of reactivation, making the memory vulnerable to disassembly by ECS. All 28 human subjects in the Rubin studies showed dramatic reduction or elimination of previously severe symptoms for periods of 3 months to 10 years, when the study was published (reviewed in Nader, 2003). The ECS treatment is ineffective for these same symptoms if given when the individual is anaesthetized (the standard form of ECS treatment) instead of during memory reactivation.

Rubin's demonstration of the use of reactivation–labilization–modification–reconsolidation to dispel clinical symptoms is of



momentous significance for the field of psychotherapy. However, ECS is unacceptable for general psychotherapeutic use because it causes large-scale, nonselective brain damage. Coherence therapy's use of native experiential process to bring about presumably the same depotentiation of memory makes it highly suitable for general clinical use.

There are various psychotherapeutic systems that entail the conscious retrieval of unconscious, unresolved emotional themes (implicit schemas), such as psychodynamic psychotherapy (Crits-Christoph, Luborsky, & Barber, 1990), psychoanalysis (Curtis & Hirsch, 2003; Wolitsky, 2003), and existential-humanistic therapies (Schneider, 2003). However, none of them, to our knowledge, directs the clinician to remain entirely non-counteractive toward the retrieved, symptom-generating material and to carry out the specific steps necessary for creating a disconfirming juxtaposition experience, as required (according to coherence therapy) for transformation of the retrieved knowledge structures.

The opposite shortcoming characterizes cognitive-behavioral therapy, which focuses heavily on the creation of a disconfirming knowledge without adequately finding and accessing the unconscious emotional constructs that require the symptom. CBT dismisses the symptom-requiring material as "irrational" and does not recognize or take advantage of its coherence. Again, the experience of true juxtaposition is not built into the methodology (though with some clients it occurs accidentally), so an extinction-like suppression of symptoms is the result, not transformation and reconsolidation.

The enhanced potency of the juxtaposition methodology is most apparent in cases, such as the one described below, in which a compelling, life-organizing prosymptom position that generated symptoms for decades is lastingly depotentiated in seconds once the condition of disconfirming juxtaposition is properly established. The crucial experience of juxtaposition is a higher-order, attentional mediation of mental operations and is by no means an automatic or inevitable result of the holding of an incompatible knowing or of making the prosymptom position conscious and integrated.

## PRAGMATICS OF DEPOTENTIALIZING THE IMPLICIT KNOWINGS

## DRIVING SYMPTOM PRODUCTION

A majority of therapy clients already possess a living knowledge that is incompatible with a newly conscious prosymptom position. Only for a minority must the therapist help create an experience that newly forms a disconfirming knowledge to use for juxtaposition and transformation.

For example, in Carol's next session after realizing that she was presupposing that her sexuality would be the same as her mother's—mortifying and violating to her daughter—she reported a lucid, contrary knowing that her own sexuality could and would differ fundamentally from her mother's. That contrary knowing was already available among her conscious, cortical models of herself and the world, and it came into juxtaposition with her prosymptom constructs spontaneously, with no prompting by the therapist, soon after the prosymptom constructs were also in conscious, cortical representation.

However, even when a contrary knowing already exists, it is not at all automatic or inevitable that a juxtaposition will spontaneously occur in consequence of the prosymptom position becoming routinely conscious. Deliberate prompting of juxtaposition by the psychotherapist is necessary about half the time.

Deliberate prompting and rapid transformation are illustrated in the case of Tina, 33, who described many years of heavy depression, complete with "black cloud," despite her use of an SSRI antidepressant. Her depressed state proved to have several prosymptom positions maintaining it, but the one found in her sixth session was the pivotal breakthrough and demonstrates rapid change of a major, longstanding core construct. (For a more complete account of this case, see Ecker & Hulley, 2002a).

Tina's sixth session focused specifically on her profound, chronic lack of interests and pursuits. The experiential discovery work took the form of a guided imaginal process involving her parents. This led to a moment in which Tina blurted out, "I erased myself!" upon realizing that rendering herself blank had been her own way of protecting herself from her invasive, omnipresent mother, who always took over and took credit for literally every interest or pursuit Tina had ever had. The solution of self-erasure had worked; it ended the wretched experience of being invaded and robbed by Mom. Tina had been carrying out that

self-protective tactic of being blank ever since childhood, with no conscious, neocortical knowledge of it at all.

Consciously she judged herself harshly for her absence of motivation and achievement, disparagingly calling herself “a vegetable.” Now her recognition of purposefully rendering herself blank, completely lacking motivation and interests, made sudden, enormous sense in a whole new way. Her depression was largely a response to that blankness in her life—a combination of anticipating an endlessly dull future and feeling intense low self-worth over her seemingly intrinsic dullness. Her discovery of her own agency and purpose in maintaining that dullness was an immediate, powerful shift. However, Tina soon said that although this illumination was very real to her, it did not yet free her, because her mother was still the same and Tina still felt as vulnerable to her as ever. Consequently her blankness was still needed for safety from Mom and others.

When the purpose (third-order construct) in a prosymptom position remains in force after becoming conscious, as it did for Tina, it means that the core, ontological (fourth-order) construct giving rise to that purpose is still in force and needs to be the target of discovery and change. Therefore the therapist (Ecker) sought next to guide Tina’s attention to that ontological construct. In order to find how to do that, he first thought as follows: “What sort of construct would dictate blankness as the *necessary* solution to Mom’s invasiveness and pillaging? Other solutions are, after all, possible; other children respond very differently to the same dilemma. What made *blankness* the right and necessary solution for Tina?” This was orienting, and he then said to Tina, “Tell me: In what ways do people keep other people from just reaching in and taking away things?”

In that way, the therapist invited Tina’s attention (a) to encounter the possibility of having boundaries and (b) to recognize what her own position is in relation to the possibility of having boundaries. During the next minute, she became conscious of obeying a “no walls rule” imposed by her mother and of her fourth-order construct of herself as a completely exposed being. Simultaneously, she recognized the amazing possibility of “having walls” and of deliberately keeping her personal affairs “behind walls” and unseen by her mother or others. The realness of this new possibility operated at once as a knowing that was

incompatible with her prosymptom knowing. The juxtaposition of the two knowings was immediately transformational. The spell of the no walls rule was broken and the prospect of freedom from her lifelong, self-imposed blankness became very real to her. Her mood became joyous and giddy—appropriately so.

A postsession task of integration, written on an index card, was created to maintain and solidify her new knowings and to guide her to implement them through small experiments in keeping her personal affairs private from her parents. Tina said that if she needed another session, she would call for it. She never did, and in follow-up telephone contacts six months and two years later, she reported being free of depression, off of antidepressants, and excited about a new career, with her vocal tone and inflection congruently expressing vitality.

This example shows that the new experience that will accurately transform a prosymptom position is strictly determined by the content of that prosymptom position and cannot be known or designed without first discovering that content.

Transformation can take longer in some cases due to various complications, such as client resistance (stemming from a separate implicit knowledge of a daunting loss, difficulty, or hardship that would be suffered if the transformation were allowed). However, the fact that a lasting phenomenological depotential is sometimes found to occur in seconds implies that the synaptic circuits of subcortical, emotional implicit memory correspondingly can depotential on this timescale. This indication seems worthy of neurodynamic investigation. Several animal studies have already been reported in which neurons of the basolateral amygdala (the site of long-term memory of fear conditioning, as noted earlier) have been depotential—switched from a turned-on state to a turned-off state (Rammes, Eder, Dodt, Kochs, & Zieglgänsberger, 2001; Rammes et al., 2000; Wang & Gean, 1999).

The observation of permanent symptom cessation following a disconfirming juxtaposition experience does not in itself rigorously prove that transformation of the implicit, prosymptom memory has occurred. However, the failure of reminders and context changes to evoke the prosymptom position and the observation that it does not spontaneously recover in situations that previously always reactivated it into producing the symptom are strong evidence of true depotential.

Following transformation work that seems effective, active pursuit of such proof is best practice in coherence therapy: The therapist probes for any remaining capacity for reactivation through use of vivid reminders. This is done by guiding the client either behaviorally or imaginally to reencounter cues, percepts, and situations known to have strongly evoked the prosymptom position. The prosymptom position can be reevoked only if it still exists. If, however, the prosymptom position has been successfully transformed and no longer exists, the client's response to the reminder is distinctive in that it consists of a report that the previous realness, seriousness, and compelling power of the prosymptom emotional theme and purpose has vanished and was not evoked, and further that that theme and purpose now seem implausible, silly, lifeless, absurd, nonsensical, or even laughable.

A response of that kind was apparent from Carol in our case example in the preceding article of this series (Toomey & Ecker, 2007) when, in the next session, she described her previously serious and troubling prosymptom position as now seeming "silly." In that case, her between-session task of integration—a daily reaccessing of her prosymptom position by reading its verbalization on an index card—first achieved its main goal of integration, then led to a spontaneous, disconfirming juxtaposition, and then functioned as a reminder of the now silly-seeming notions she had held.

If Carol's therapist had felt it necessary to test for transformation during the session, she would have done so, for example, by guiding Carol first to picture her husband making a sexual overture to her, and then to respond (within the visualization) by genuinely feeling and saying to herself, "I've *got* to resist this, because if I go along with it and enjoy sex with him, I'll become like Mom, openly erotic and voyeuristic toward our daughter's sexuality." If this construction still existed, it would, with high predictability, have been reevoked and unmistakably felt through this exercise. If it no longer existed, the lack of subjective realness of the words would have been just as unmistakably apparent to Carol. The client's report of nonrealness in the face of such a strong reminder is, we suggest, phenomenological evidence that reconsolidation has eliminated the implicit emotional constructs.

For a nonimaginal, real-life version of the same type of probe, Carol would have been assigned the between-session task of internally feeling and saying to herself the same things on the next occasion when her husband makes a sexual overture. (For a real-session example of that transformation test procedure on video, in which the client describes a major, lifelong, newly conscious prosymptom position as being extremely funny at the end of one session of therapy, see Ecker & Hulley, 1997).

The same type of response was observed by Rubin et al. (1969) in one of their electroconvulsive shock subjects, a woman obsessed with the urge to stab her mother. After ECS treatment she was asked if she still felt like stabbing her mother. In response she laughed at the idea and said, "Oh, she doesn't deserve anything like that." This woman was then free of symptoms throughout a two-year follow-up.

Probing for reactivation requires a reminder that is designed to evoke the specific content of a prosymptom position. If, in response to such an evoker/reminder, real or imagined, the client experiences a reenlivening and reinhabiting of the prosymptom position as still having some degree of emotional realness, then that prosymptom position still exists and requires more work. Likewise, when a client reports a reactivation that occurred during any real-life situation, the therapist takes this as a cue to carry out further discovery, integration, and/or transformation work in the concrete scene and situation that evoked reactivation. Day-to-day life itself often greatly facilitates the thoroughness of the work by inducing reactivations that reveal exactly where and how the work is incomplete.

Transformation of a prosymptom position is observed to occur all at once or in incremental stages. A prosymptom position consists of several hierarchical constructs, as described in the previous article, and the therapist faces a choice as to which construct to subject to disconfirming juxtaposition. As a rule it is the fourth-order, ontological construct (the most superordinate) that is the most effective explicit target, because the existence and realness of the entire position depends on it, as illustrated in the case of Tina. Often, however, a disconfirmation experience that focuses explicitly on the *purpose* (third-order construct) driving symptom production is adequate, probably because the directly

underlying ontological construct is so clearly implicated that it too gets disconfirmed, albeit implicitly.<sup>11</sup>

The therapist deliberately prompted transformation in work with Tina. However, as already noted, transformation can and often does take place as a spontaneous result of integration of a prosymptom position. This occurs because integration deinsulates and exposes a prosymptom position to a wide range of other knowledges held by the person, some of which prove to be incompatible with prosymptom knowledge and therefore capable of disconfirming it.

We noted earlier that the case example of Carol illustrates this spontaneous form of transformation via disconfirming juxtaposition. Carol's congruent report in a subsequent session that her own compelling prosymptom position, read on an index card between sessions, began to seem "almost silly" is a clear indication that a considerable degree of transformation had followed integration spontaneously. The disconfirming knowledge that had juxtaposed was indicated by Carol's comments to the effect that "finding a new way to understand her sexuality as her own and not her mother's was a freeing experience." A key construct in her prosymptom position was the fourth-order knowing that her mother's sexuality reflected the general nature of sexuality; that any real appetite for sex on her own part meant that her sexuality would be the same as her mother's. However, Carol harbored a contrary knowing that came into spontaneous juxtaposition with this. What felt freeing, actually, was the dissolving of that limiting and anxiety-producing fourth-order model of sexuality that she had formed in implicit memory as a girl, decades before.

This illustrates one of coherence therapy's principles of change: *People are able to change a position they experience having, but are not able to change an unconscious position they do not know they have.* An in-depth therapist who does not know that transformation occurs only as a result of disconfirming juxtaposition is likely to incorrectly construe spontaneous transformation as confirming his or her expectation that implicit memory retrieval and integration alone should be freeing, although it is not, as noted earlier. Retrieval alone does not unlock the hold and the realness of the material if no juxtaposition occurs. Likewise, as Ecker (2005) has pointed out, even counteractive methods

sometimes result fortuitously in an internal, unvoiced experience of juxtaposition of a prosymptom construct and a disconfirming knowledge, yielding a transformation and lasting symptom cessation. In such instances both therapist and client, unaware of the crucial juxtaposition that happened to occur, may infer mistakenly that counteracting in itself produced the profound change.

When transformation does not occur spontaneously following integration of a prosymptom position, the therapist must deliberately orchestrate a disconfirming juxtaposition. A wide range of techniques are useful for this purpose (see Ecker & Hulley, 1996, 2000a, 2004), but techniques per se are not always necessary, as shown by the example of Tina, who was simply prompted to attend to the disconfirming knowings while experiencing her prosymptom knowings.

We emphasize that disconfirmation through juxtaposition is, like the rest of the native-process-matching methodology of coherence therapy, a noncounteractive process. Specifically, as our case examples have shown, the therapist says and does nothing whatsoever directly opposing the prosymptom position. To the contrary, the therapist continues to accept it, to empathize with it, and to guide the client's attention to stay with it, in it, and experiencing it, not to get away from it. At the same time, the client also is guided to attend to and experience some other, contradictory knowledge. The therapist lets the contradiction speak for itself and scrupulously expresses nothing that comes across as indicating which of the two incompatible knowings is right and which is wrong, instead trusting the client's native process to carry out the depotentiation of the prosymptom knowings and synapses. The slightest indication of a right-wrong preference from the therapist subverts the juxtaposition experience, prevents it from occurring, and replaces it with a counteractive, cognitive experience that will have little if any effectiveness.

If, as we have been arguing, juxtaposition is indeed the mind's and brain's critical condition for profound change in the experiential realness of personal knowledge structures, it is reasonable to ask: Why is that so? The critical role of juxtaposition seems to imply the operation of a meta-level rule or self-ordering principle that strictly requires a well-knit consistency among all of



the constructs, or knowings, that are copresent *within any one field of awareness*. Inconsistency evidently is not tolerated among constructs being experienced together. In contrast, knowings that are dissociated—held separately and never co-attended—are allowed to be wildly incompatible. However, once constructs are brought together into the same field of awareness/attention, the brain and mind appear to be strict about maintaining compatibility, either by decommissioning one construct out of existence or by resplitting them back into separate compartments.

Finally, the client's moment-to-moment steps of experiencing a disconfirming juxtaposition are noteworthy. When attention first subjectively encounters and holds the two-sided, disconfirming juxtaposition, there is a sudden, lucid recognition that the prosymptom constructs, which until this moment had seemed to be the given reality of the matter, are actually notions that had been formed by oneself. There is a surprising recognition that one was only imagining it to be so. One's awareness is suddenly now separate from, and superordinate to, the prosymptom constructs. One recognizes oneself as the author of those now obviously erroneous notions, and in the next moment one deauthorizes them by withdrawing a kind of life-giving, imaginal consent and participation. This leaves the constructs withered and lifeless (as is also happening, presumably, to the corresponding synapses). These experiential events are nonverbal, direct knowings that happen very quickly, silently, and implicitly. We believe these experiential elements to be noteworthy because they suggest a different kind of evidence—phenomenological evidence, the intrapsychic perception of the dissolution of constructs—that is consistent with the view that a disconfirming juxtaposition depotentiates the implicit memory requiring the symptom.

### **Conclusion: Toward Unifying the Phenomenology and Neurobiology of Psychotherapy**

Our aim in this article has been to capitalize on strides in neuroscientific research by attempting to map the methodology of coherence therapy onto known mechanisms of synaptic change in the brain. We see this exercise as a new step in the unification of the reductionistic and holistic viewpoints on human experience and change.

Based on the specific neurodynamics we have identified in this article as being the plausible neural correlates of coherence therapy methodology, we believe that the following tentative inferences can be made, pending direct, rigorous confirmation through neurological study of therapy clients undergoing coherence therapy:

- The methodology of coherence therapy appears to depotentiate the specific implicit memories generating a given symptom, resulting in symptom cessation.
- The rules of change of the brain–mind–body system, as defined by coherence therapy, appear to be corroborated by the rules and mechanisms of neuroplastic change.
- Subjective attention is a powerful agent of change, neurally as well as experientially, if used in accordance with the brain–mind–body system’s rules of change.
- The distinction between transformative change and counteractive change is of fundamental importance to psychotherapeutic effectiveness, because counteractive change fails to remove the cause of symptom production and is therefore inherently unstable and susceptible to relapse.
- Coherence therapy, by virtue of (a) procedurally fulfilling the requirements for neurological reconsolidation and (b) rendering longstanding, symptom-generating implicit learnings nonevocable, appears to recruit reconsolidation, the brain–mind–body system’s inherent neural process for rapid, transformative change.
- Built into coherence therapy methodology is the neural integration of brain regions harboring implicit and explicit memory—that is, the neural integration of unconscious, non-verbal, emotional–perceptual–somatic knowings and conscious, verbal knowings.
- Coherence therapy appears to fulfill our proposed criteria for optimal psychotherapeutic use of neuroplasticity: It apparently recruits reconsolidation, the most potent mechanism of synaptic change; it does so in the brain regions and circuits causing symptom production; and it can proceed as rapidly as neuroplasticity allows synaptic change to occur.

The phenomenological and neurobiological models and methods set forth in this article are based on extensive clinical observation of thousands of psychotherapy clients across two decades, as well as our scrutiny of pertinent research in neuroscience and psychotherapy. However, neither of the authors is a trained neuroscientist, and controlled studies have not yet been conducted for the models and processes we have described.

Toward that end, we envision the following research agenda for determining the veracity of most of the inferences listed above.

1. *Controlled outcome study of efficacy as a function of degree of methodological adherence.* We plan to develop measures of qualitative process adherence that will quantify the degree to which the defining features of coherence therapy are carried out. This adherence grid would then be applied to a controlled outcome study to yield plots of efficacy versus level of adherence. Key dimensions of adherence to be rated will include these descriptors of therapeutic process: coherence-focused, non-counteractive, nonpathologizing, noninterpretive, experiential, phenomenological, depth-focused, process-directive, and specificity of unconscious material accessed. The arguments in this article predict that with complete adherence, coherence therapy should yield significantly higher efficacy than has been measured to date for other therapies (exceeding the Dodo bird verdict). As adherence drops below 100%, we expect efficacy to drop rapidly down to the usual level.
2. *Identification of process markers critical to efficacy.* Coherence therapy entails several major milestones of methodology that are deemed necessary for full efficacy. Qualitative studies are needed to test the recognizability and the necessity of these events, which include (a) experience of discovery of prosymptom position, (b) experience of previously unconscious purpose and agency in generating the symptom, (c) experience of juxtaposition of key prosymptom constructs with a disconfirming knowledge, and (d) experience of depotentiated prosymptom position as lacking emotional realness and nonreevocable by former triggers.
3. *Identification of differing brain processes subserving coherence therapy versus counteractive and other noncoherence-focused psychotherapies.*

Our expectation that coherence therapy is capable of a significant enhancement of efficacy rests on the hypothesis that it operates through a fundamentally different process of change, including a different neural process in the brain, than do all therapies previously subjected to controlled trials. These differences may be apparent in fMRI brain images that would compare the brain systems and circuits active during key processes of change in coherence therapy (see previous paragraph) versus key points in other therapies.

We have emphasized the neuroscientific verification of the role of subjective attention in bringing about mental change because this is of particular importance for coherence therapy. Coherence therapy is most essentially a coherence-guided use of attention so as to create experiences that bring about psychological and neural transformation. From the neural viewpoint, a person undergoing coherence therapy is guided first to use attention to create experiences that locate the neural circuits driving a particular symptom (the discovery process), then to use attention to create experiences that connect those neural circuits to the neocortex (the integration process), and finally to use attention to create experiences that depotentiate those neural circuits, ending symptom production (the transformation process). Coherence therapy skillfully applied can often have a high degree of accuracy in locating, accessing, and depotentiating the implicit memory driving symptom production.

The distinction between transformative and counteractive change is so important, in our view, that it justifies this final emphasis: We reiterate the view of Ecker and Hulley (2000c) that the “common factors” that cause over a dozen seemingly different forms of psychotherapy to score the same modest level of efficacy—apparently, the same level as properly designed placebos (Baskin et al., 2003)—are not any of the factors often cited, such as empathy, trust, attunement, and a good client–therapist working alliance (see, e.g., Hubble, Duncan, Miller, & Hubble, 1999). Rather, as understood from the vantage point of coherence psychology, the common factors responsible for the efficacy barrier are these (Ecker, 2006): (a) a counteractive methodology, (b) the lack of a decisive coherence focus within a noncounteractive methodology (that is, failure to address adequately the

implicit schemas actually causing symptom production), and/or (c) nonutilization of the built-in process of change (disconfirmation of implicit, symptom-requiring schemas through a juxtaposition experience).

In other words, the usual common factors analysis, which holds that specifics of therapeutic methodology and technique have little influence on effectiveness, may well be true within the universe of counteractive and noncoherence-focused methodologies, but not beyond it (Ecker, 2006). Indeed, neuroscience suggests why such methods are fundamentally limited, as discussed in this article. In contrast, in the domain of *noncounteractive*, coherence-focused, transformative therapies, (a) effectiveness seems to be dramatically higher and (b) methodology matters greatly. For example, details of methodology determine whether or not the work fulfills the specific conditions required for the neural reconsolidation of an implicit memory.

Coherence therapy by design can be effective only when the cause of a symptom presented in therapy is an implicit memory, a set of unconscious, subcortical personal constructs, or prosymptom position(s), that were formed previously in response to experience. Thus, if a given symptom can be dispelled by coherence therapy, we may reliably infer that the cause of this symptom was an implicit memory—a purely psychological cause—rather than a genetic defect or neurochemical imbalance (causes that will be considered in the next, third article in this series; Toomey & Ecker, in press). The effectiveness observed by coherence therapy practitioners for a broad range of symptoms<sup>12</sup> is itself evidence that implicit memory is the cause of many more so-called clinical disorders than is now recognized by the therapy field.

A qualitative yet empirical proof of symptom coherence (symptom causation by a well-knit implicit schema) is built into coherence therapy methodology properly carried out with each client: With no counteractive, symptom-opposing measures ever applied (as could be verified through qualitative analysis), symptom cessation nevertheless occurs, immediately following depotentiation of the discovered, symptom-requiring schema in implicit memory. Depotentiation is verifiable in the manner described in this article.

Although coherence therapy has been used effectively for a wide range of clients and symptoms, it is not applicable to certain

client populations. The main counterindication is a profound, chronic avoidance of interior experiencing of vulnerable feeling, to a degree that is refractory well beyond even a significant level of “resistance.” Profound avoidance of interior process tends to be the case for persons with extreme degrees of several types of “character disorder,” including persons who are active addicts (of alcohol, drugs, sex, gambling, etc.) and some persons with significant bipolar or psychotic symptoms. Also, persons currently suffering extreme degrees of depression, anxiety, or dire situational crisis are likely to need some degree of relief of symptoms or situation through other methods before they have enough free attention to participate usefully in coherence therapy. And, of course, any symptoms caused not by personal constructs in implicit memory but by true biochemical, genetic, or neural irregularities, or by organic damage, will not respond to coherence therapy. In the next article in this series, we argue that this is likely to be the case for a very small minority of therapy clients.

As depicted through most of this article, neuroscientists are in the position of being the bestowers of knowledge and psychotherapists are in the position of receivers and appliers of this knowledge. There are a few exceptions, however. Several areas for future neuroscientific research have been identified:

- A key element of clinical phenomenology documented by Ecker and Hulley (1996, 1997, 2000a, 2000b) is the depotentiating of a long-term, emotional implicit memory, sometimes in seconds, by an experience of disconfirming juxtaposition. This degree of endogenously induced neuroplasticity—this native human capacity for full depotentiation of synapses storing long-term, implicit emotional memory—has not, to our knowledge, been studied by neuroscience.
- Our neurodynamic analysis of coherence therapy’s use of disconfirming juxtaposition to achieve transformation has yielded indications that humans, unlike animals, can, through deployment of attention, put an extinction learning (a contradictory knowledge) into neural and experiential contact with an acquired memory to drive reconsolidational (transformative rather than counteractive) change of that acquired memory. This contrasts with the almost century-old tradition in

neuroscience and learning theory of regarding all extinction learning as strictly dissociated and counteractive, as in animals.

- In the first article of this series (Toomey & Ecker, 2007), we reviewed Ecker and Hullely's (1996, 2000b) phenomenological delineation of the hierarchical, four-level structure of emotional implicit memory, which implies a correlate structure in the neural architecture of emotional implicit memory systems, although to our knowledge, this particular hierarchy appears not to have been identified by brain science as yet.

For psychotherapists it should be welcome to know that among prominent neuroscientists there are signs of receptivity to a two-way flow of knowledge. For example, Eric Kandel (2001, p. 605) stated toward the end of his Nobel address, regarding a range of challenging unknowns in brain science:

These systems problems of the brain will require more than the bottom-up approach of molecular biology. They will also require the top-down approaches of cognitive psychology, neurology, and psychiatry. Finally, they will require a set of syntheses that bridge the two approaches.

We share that spirit. In a unified model, there are two complementary ways to understand the cause of symptom production: phenomenological and neural. Phenomenologically we can view the cause of most (not all) symptoms of clients as being the unconscious emotional-perceptual-somatic knowings, the unattended themes, purposes and tactics that necessitate producing the symptom. Neurally, we can regard the cause as being the subcortical neural circuits of implicit memory and procedural implementation that encode those knowings and that are firing when symptom production is occurring.

Those two dimensions of symptom production—subjective and objective, holistic and reductionistic—do not exist separately from each other. Each exists through the other. If we truly transform the underlying emotional themes driving symptom production, we also transform the implicated neural pathways.

We caution against concluding that developments in neuroscience imply the need for a reductionist view that sees consciousness and selfhood as mere epiphenomena of purely physical processes. In preparing this article we have closely studied many

dozens of research articles by neuroscientists, in which not once was any neurodynamic process or property referred to as a *cause* of sentient experience, but only as a “substrate” or “correlate.” Neuroscientists have made impressive progress in identifying physical mechanisms that translate physical perceptions into a physical pattern of synapses, but they have nothing to say about the source and ultimate nature of sentient, subjective experience. In this we subscribe to Albert Einstein’s cautionary guideline, “Things should always be made as simple as possible, but not simpler.”

### Notes

1. The name used from 1993 through 2005 was depth-oriented brief therapy, or DOBT. The change to “coherence therapy” and “coherence psychology” is intended to reflect the central principle of the approach.
2. Ecker previously worked in experimental physics research for 14 years.
3. Ecker and Hulley (1996) refer to the discovery process as *radical inquiry* to denote both its focus on root material and its swiftness.
4. Upon perceiving an action done by another, one’s mirror neurons fire as if one had performed that action oneself. They are thought to help create a model of the internal states of others, facilitating empathy and the ability to reliably infer the presence of certain subjective states in others (sometimes termed *mindsight*). Mirror neuron dysfunction has been linked to autism.
5. An opposite effect, the disruption of explicit memory formation, is caused by the yet higher hormonal levels produced during experiences of extreme emotional arousal or stress, as during trauma (McGaugh, 1989; Payne, Nadel, Britton, & Jacobs, 2004; van der Kolk, 1994). Under these conditions the hippocampal memory formation system is rendered inoperative, and memories are stored instead as separate, unintegrated affective and perceptual components, which tend to be exceptionally vivid and enduring. Thus, as noted by van der Kolk (1994), the effect of trauma on memory is bimodal: An absence of explicit, narrative memory (amnesia) is accompanied by a sharp enhancement of perceptual and affective memory (*hypermnesia*).
6. Hippocampal synapses that encoded a mouse’s spatial memory of a newly encountered neutral environment, which attracted little attention, lasted 3 to 6 hours, but when an added, specific feature of the same environment was of special significance to a mouse, attracting strong attention, the same synapses mapping spatial memory lasted for days, or about 20 times longer.
7. Homosynaptic plasticity: A particular synapse becomes stronger (“facilitation,” “potentiation”) or weaker (“depression”) because of activity in the presynaptic and postsynaptic neurons of that very synapse.
8. Heterosynaptic plasticity: Change in a synapse’s strength occurs due to activity in one or more modulatory interneurons that act on the presynaptic and/or postsynaptic neurons of the synapse, and not due to activity in the presynaptic or postsynaptic neurons themselves.



9. Other possible reasons for nonextinction of prosymptom positions include a variable ratio schedule of learning, which is the learning schedule most immune to extinction (Field, Tonneau, Ahearn, & Himeline, 1996); higher-order conditioning (Gewirtz & Davis, 2000), which could be largely immune to perceived disconfirmation; and redundant conditioning, in which multiple percepts, each a conditioned stimulus, tend to occur concurrently, again creating immunity to perceived disconfirmation.
10. It should be noted that there are two differences between the memory-changing procedure used by Frenkel et al. (2005) and that of coherence therapy: (a) The procedure of Frenkel et al. was designed for reconsolidation of a positive alteration (up-regulation, strengthening) of a conditioned response memory by a concurrent experience, whereas the procedure in coherence therapy is designed to use the same mechanism of reactivation–labilization–concurrent experience–reconsolidation for a major down-regulation or weakening of the strength of a prosymptom implicit memory. Our unified model awaits demonstration of a down-regulatory version of the Frenkel et al. use of a real-life episode. (b) The concurrent experience used by Frenkel et al. (2005) was entirely unrelated to the target memory in both content and context, and operated to strengthen the target memory purely through endogenous biochemical effects. In contrast, the concurrent experience used in coherence therapy is, by design, a phenomenological experience strongly related to the content and context of the target memory and is change-inducing through experiential disconfirmation of the target memory.
11. In a small minority of cases, the client's prosymptom position does not require transformation because, upon becoming conscious, the client recognizes that it serves his or her best interests just as it is. For example, the unconscious prosymptom position generating a graduate student's serious procrastination of course assignments was a refusal to pursue a program and a career that had been chosen by his parents and was not his true calling. He did not change this position (other than to make it conscious); rather, he resigned from the program. Ecker and Hulley (1996) term this *reverse resolution*.
12. Symptoms dispelled by coherence therapy include depression, anxiety, panic, agoraphobia, low self-worth, attachment problems, sequelae of childhood abuse, sexual problems, food/eating/weight problems, rage, attention deficit, complicated bereavement, fidgeting, codependency, underachievement, procrastination, and a wide range of interpersonal, couple, and family problems.

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