Psychosocial Stress Series

When THE PAST Is Always PRESENT

Emotional Traumatization, Causes, and Cures

RONALD A. RUDEN

ROUTLEDGE

This book is part of the Psychosocial Stress Series, edited by Charles R. Figley.

Routledge
Taylor & Francis Group
270 Madison Avenue
New York, NY 10016

Routledge
Taylor & Francis Group
27 Church Road
Hove, East Sussex BN3 2FA

Routledge is an imprint of Taylor & Francis Group, an Informa business

Printed in the United States of America on acid-free paper
10 9 8 7 6 5 4 3 2 1

International Standard Book Number: 978-0-415-87564-6 (Hardback)

Library of Congress Cataloging-in-Publication Data

Ruden, Ronald A.
When the past is always present : emotional traumatization, causes, and cures / Ronald A. Ruden. -- 1st ed.
p. cm. -- (Psychosocial stress series)
Includes bibliographical references and index.
ISBN 978-0-415-87564-6 (hardcover : alk. paper)
1. Psychic trauma. I. Title.

BF175.5.P75R83 2010
616.85'21--dc22 2010007844

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http://www.routledgementalhealth.com

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Disrupting a Traumatization

Traumatization can be avoided if, during a potentially traumatizing moment, an escape is perceived. If an event is encoded as a traumatic memory, upon recall of the event, providing the individual with a safe haven should disrupt the trauma-induced linkages and erase the emotional response to related stimuli.

Avoiding the Encoding of a Traumatic Memory

A gang of thugs is chasing you after you inadvertently insulted the leader's girlfriend. They are going to beat you to a pulp after they catch you and you are literally running for your life. You are getting tired and there is no place to hide. The thugs are gaining and your heart is racing as fast as it can to keep you going. They are just about to catch you and all of a sudden you wake up. Sweating, eyes wide open, and heart pumping, you realize it was just a dream and you laugh to yourself, but it's a little hard getting back to sleep. Awakening just in time from a scary dream is a great way to escape the danger and avoid traumatization. Escape is when the danger has permanently passed. Hollywood understands this concept. How many endings of scary movies have you seen where the predator, thought to be killed, somehow manages to survive and look the audience in the face? Just when you thought you were safe, that you escaped—it is the stuff nightmares and traumatizations are made of.

At the beginning of a potentially traumatizing event, when specific pathways are being created, it is unclear whether the criteria for inescapability will be met. At this moment, dopamine, norepinephrine, and cortisol are elevated, preparing us to do what needs to be done to survive. If we survive and find a haven we calm down. To calm down, we need to inhibit the release of norepinephrine from the locus

coeruleus (LC) and inhibit the central nucleus (Ce) from further activating our physiology. In the LC, serotonin via its effect on GABA neurons prevents the release of norepinephrine.[1] The prefrontal cortex, on perceiving the threat has passed, inhibits the Ce via GABA interneurons. The amygdala is now quiet and the event fades. But things are different if an event is encoded as a trauma.

Disrupting an Encoded Trauma

Once an event has been encoded as a traumatization, the subsequent finding of a haven does not disrupt the pathways. How, then, do we de-encode a traumatic memory? The answer is to disrupt the synaptically encoded glutamate-specific pathways in the basolateral complex (BLC). To do so, we must seek the event that leads to their activation, for as we have mentioned earlier, once activated, the glutamate receptors become vulnerable to disruption. Then, once activated, fool the brain into thinking a haven has been found. To promote this, we must also inhibit the cognitive component from further activating the amygdala. The path to a cure is outlined below:

Retrieval of emotional component → Working memory → Hippocampus → Activate BLC → ↓ Sensory input of the event to the amygdala by distraction → Havening → Disrupt the encoded emotional BLC pathway by depotentiation of glutamate receptors → De-link the components of the traumatization → Traumatization cured

Preventing the Passage of a Retrieved Component From Working Memory to the Amygdala

Conscious/subconscious retrieval of a traumatized component en route to the amygdala passes through a system known as **working memory** (WM) (Figure 7.1). The working memory system (generally considered to be part of the prefrontal cortex) receives the stored memories and sends this information to the hippocampus. If this information has been encoded as part of a traumatization, it is forwarded to the

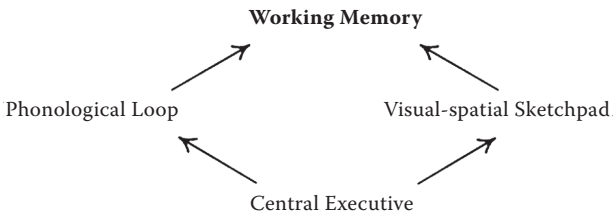

Figure 7.1 Working memory. (Courtesy of Ronald Ruden and Steve Lampasona.)

amygdala. The WM system is a limited-capacity store for retrieving and retaining information over the short term that allows for performing mental operations on the contents of the store. According to Baddeley,[2] working memory has, at minimum, two components, a **phonological loop** that is concerned with auditory and speech-based information, and the **visual-spatial sketchpad** that maintains and manipulates visual and spatial information. His model also postulates a **central executive** that directs what working memory pays attention to and supervises these two components. The central executive's role is to regulate attention, and it does not readily allow working memory to hold dissimilar items simultaneously.

Displacing From Working Memory a Stimulus That Activates the BLC

Working memory is the system to which retrieved components of the traumatic memory are first brought. In order to keep the component in working memory, it must be rehearsed or augmented by an emotional feeling. The ability of an emotion-producing stimulus to sustain the item in working memory is the reason feelings can overwhelm rational thought. However, even emotion-producing stimuli that enter the working memory system can be displaced if the mind is distracted. Displacement can be accomplished by simultaneously attending to other cognitive or physical tasks. Using Baddeley's model, after entry into the working memory, having the individual attend to verbal commands that activate the visual-spatial sketchpad (having them imagine walking downstairs while counting the steps) or the auditory loop (hum "Take Me Out to the Ball Game") can displace the component. It is nearly impossible for the mind to sustain two different items in working memory. Try for yourself

by adding two 3-digit numbers in your head while humming the "Star Spangled Banner." For the moment, this displacement stops the retrieved traumatic memory from activating the amygdala and producing a response. However, it requires a concentrated effort to do so.

Working memory can only hold one item.

Traumatic component in WM → Displacement from WM
→ → /////// Hippocampus → ////// BLC activation → No response

The displacement of the traumatic component from working memory temporarily extinguishes the response.

If the working memory is holding an event that activates the emotional component, it is difficult to dislodge it, and even if dislodged, it returns at another time. Nonetheless, if one can displace the event, activation of the BLC will cease. Thus, for example, for someone who is snake phobic, bringing into working memory an image of a slithering, sliding snake will cause the release of norepinephrine and a fear response. Distraction by thinking of something else stops this conscious activation of the BLC. However, bringing another snake to WM will reactivate the BLC and cause the person to reexperience the fear response.

Shakespeare expressed the idea that retrieval of traumatic memories causes us to reexperience the feelings as if for the first time, and that we can alter these feelings by displacement from working memory:

Sonnet 30

When to the sessions of sweet silent thought
I summon up remembrance of things past,
I sigh the lack of many a thing I sought,
And with old woes new wail my dear time's waste:
Then can I drown an eye, unused to flow,
For precious friends hid in death's dateless night,
And weep afresh love's long since cancelled woe,
And moan the expense of many a vanished sight.
Then can I grieve at grievances foregone,

And heavily from woe to woe tell o'er
The sad account of fore-bemoanèd moan,
Which I new pay as if not paid before.
But if the while I think on thee, dear friend,
All losses are restored and sorrows end.

Activating Traumatic Components So They Can Be Treated

In order to disrupt a traumatic memory, it must first be retrieved and brought into working memory. Then it must activate the BLC. This activation corresponds to the release of glutamate in the BLC-specific pathway that was produced during encoding. It is this ability of stimuli to activate the BLC pathway that must be de-encoded. This prevents a signal from being sent to the Ce (which in turn activates the locus coeruleus and releases norepinephrine) and the other areas of the brain where the linkages are stored. In the case of subconscious stimuli that activate somatosensory and autonomic symptoms, bringing the symptom into working memory followed by havening may rid the individual of the symptoms, but it will not eliminate the synaptic pathway through the BLC that encodes the emotional response. The ability to reencode these nonemotional components exists and relapse remains possible. Sarno[3] has observed this in many of his patients, symptoms returning or appearing elsewhere if the emotional core was not disrupted.

To de-traumatize an event we must search for its emotional origin so that it can be activated. A diagnosis of an amygdala-based disorder should make us seek the encoding event. Chronic pain and other somatic symptoms should cause us to search for a traumatizing event or unresolved anger. PTSD has both cognitive and subconscious stimuli that activate the emotions, and all should be sought. Phobias directly enter into working memory by cognitive processes and activate a fear response. Trying to recall the first time it happened is helpful. Pathological emotions arising from distressing events can be directly activated by conscious effort. If no origin can be found, such as in panic attacks, one can still generate emotions for panic disorder by thinking about the last time it occurred and how fearful we are that it will happen again. Even events that are not cognitively stored, such as

those from early childhood, can be de-traumatized if we can recreate the felt sense, the emotion and/or some sensory feeling. If the traumatizing event can be found and activated, this affords an opportunity to alter the BLC pathway. How can this be accomplished?

Early Successful Trauma Treatments

Early attempts at treating the consequences of a traumatization by talk therapy were generally unsuccessful. Most researchers felt that a traumatization permanently encoded the event, and that cognitive cues or subconscious triggers of the event caused emotional, somatosensory, and visceral responses derived from the original trauma. Professionals in this field thought that a cure was not possible. As we shall see, this has proved to be wrong.

Dr. Roger Callahan first described his tapping approach to cure trauma in 1985.[4] This was followed by eye movement desensitization and reprocessing (EMDR), described by Dr. Francine Shapiro.[5] Both therapies involve imaginal reexposure to the event and followed by various forms of sensory input. Dr. Callahan's approach was to evoke the memory of the trauma, then tap on various acupuncture points. This would be interspersed with a distracting process called a Gamut procedure. EMDR has eight phases. These phases include reexposure and maintenance of the images while attending to other forms of stimulation in the form of repeated sets of eye movements, tones, and taps. The goal is to focus on the information, as it is currently stored. In well-controlled trials, EMDR was shown to cure PTSD in a significant percentage of patients.

Somatic experiencing is a method for the treatment of trauma described by Dr. Peter Levine.[5] He focused his therapeutic efforts on the moments when a traumatizing event is encoded and uses an escape metaphor to describe his theory. It is of interest here because he recognizes that finding an escape is critical for the resolution of a traumatized event. According to Levine,[6] "Traumatic symptoms are not caused by the triggering event itself. They stem from the frozen residue of energy that has not been resolved and discharged; this residue remains trapped in the nervous system where it can wreck havoc on our bodies and spirit. It occurs because we cannot complete the

process of moving in, through and out of the 'immobility' or freezing state." (Dr. Levine and Dr. Scaer (see below) use the term *freeze state* to denote flaccidity.) He uses the animal model of "freeze discharge" to free the individual from the traumatic event. After attempting escape and being caught, an animal becomes flaccid. If somehow the animal survives, it begins to move its legs as if it were running. This is the freeze discharge. After a few moments, the animal is then able to get up and walk away.

Traumatization occurred when the animal could not experience a freeze discharge. For Levine, the animal is psychologically and physically frozen in time. How does one escape from this state? Levine says this is possible by accessing the "felt sense," that which is stored in the procedural memory system. This can be done by a variety of ways, not always requiring the recall of the event. Just sensing the inescapability may be sufficient. Levine[7] uses Eugene Gendlin's term *felt sense*, which "is not a mental experience but a physical one. Physical. A bodily awareness of a situation or person or event. An internal aura that encompasses everything you feel and know about the given subject at a given time—encompasses it and communicates it to you all at once."

This felt sense is the gut feeling, the knowing without knowledge, the experience of correctness or incorrectness; it is somatosensory information without interpretation. It is the physical aspects of emotion without cognition. The same pathway that is activated by cognitive generation of emotions is also activated and experienced as a felt sense.

The first step in somatic experiencing is to retrieve the feeling aspect of the event. The next step is to complete an escape that liberates the undischarged energy. In his seminal story, Levine encourages the patient to run when fear arises. This completes the escape, a freeze discharge has occurred, albeit in the client's imagination, and the person is cured. His discovery story is wonderfully instructive[8]:

> As I began with this patient she began to relax. Suddenly, without warning, she panicked. Terrified, and with no notion of what to do, I had a fleeting image of a tiger jumping towards us. It appeared dreamlike, and at the time, I had no idea where it had come from.

> "There is a tiger coming after you, Nancy," "Run toward that tree; climb it and escape." To my amazement, Nancy's body began to shake and tremble. Her legs started making running movements. After several minutes, she took a few spontaneous breaths. This response, which was scary for both of us, washed over her in waves for almost an hour. At the end she experienced a profound calm, saying she felt "held in warm tingly waves."
>
> Nancy reported to me that during this hour she saw mental pictures of herself at the age of three being held down and given ether anesthesia for a tonsillectomy. The fear of suffocation she experienced as a child—and that she remembered and revisited during her session with me was-terrifying. As a child she felt overwhelmed and helpless. After this one session with me, a whole host of debilitating symptoms improved dramatically, and she felt "like had herself again."

Another mind-body exposure approach is called the sensorimotor approach to psychotherapy. Here the somatosensory component is brought to awareness and then treated. Pat Ogden and colleagues[9] describe this process in their book *Trauma and the Body*. In this therapy, talking is not of importance. Neither are the associations, fantasies, narratives, and defenses the individual has. Rather, it is the unregulated body experiences that are the focus of this therapy. For traumatized individuals, although the narrative of the event may be dissociated, the somatic experience is available. Using this approach, the memory can be safely reevoked and empowering actions are executed. These exposure methods use emotions and body sensations to activate the specific glutamate-encoded pathways, causing them to be subject to disruption.

Are there other ways that we can disrupt this encoding?

Disrupting the Amygdala Component of a Traumatic Event: A Neurobiological Mechanism

Early researchers such as Janet and Freud[10] felt that traumatization caused their victims to become fixed in the past, in some cases becoming obsessed with the trauma. Janet observed behaviors and feelings that included nightmares, intense reactions to benign stimuli, terror without reason, and grief without relief to reminder cues arising from

the original event. These are people stuck in their past with no escape, for whom the past is always present. These memories do not decrease over time and they elicit responses decades after the event.

Sonia, the daughter of an employee of Homeland Security, heard frightening stories about terrorists and potential threats to the country as she grew up. After getting married, Sonia's husband would be awakened in the night by her screaming. He would find her curled in a fetal position in a corner of the room screaming, yet she was asleep. These are called night terrors (see Appendix D). He couldn't awaken her, and the episode could last several frightening minutes. She didn't recall those moments. Sonia also found that she didn't like to leave the house. She would only go for a walk with her new and very large bulldog. Her life was becoming more and more constricted. It was clear from her history that she could not find a safe place; chased, she could not escape.

A potential model for the disrupting an encoded glutamate pathway comes from Rasolkhani-Kalhorn, Harper, and Drozd, on the mechanism for the efficacy of EMDR and amygdala de-potentiation (see Appendix F). These researchers believed that EMDR disrupted the activated glutamate receptors by a mechanism called de-potentiation. The principal mechanism for depotentiation is the removal, by internalization, of activated glutamate receptors by the production of a low-frequency signal produced by eye movement. These receptors, now internalized within the neuron, cannot transmit a signal and the pathway is disrupted.

Activated BLC glutamate receptor → Eye movements → Induction of low-frequency signal → Depotentiation and internalization of BLC glutamate receptor → Inability to transmit a signal → Traumatic memory disrupted

Are there other forms of sensory input that can accomplish this?

The Extrasensory Response to Touch

The first experiences we have with fear, especially abandonment, seem to respond to touch. What does this touch do? In addition to temperature, vibration, consistency, shape, texture, pressure, and of course pain, touch provides comfort, sensuality, relaxation, and experiences

that have nothing to do with the classic neurobiology of ascending pathways. The consequences of the sensation of touch in mammals must therefore affect pathways that involve cognition and emotion.[11] These are the extrasensory properties of touch.

For example, if I stroke the bottom of my foot, or I have a friend stroke the bottom of my foot, the ticklish response is much more intense when my friend strokes it. If someone you hated stroked your head, the response would be much different than someone you loved doing the same thing. So the context of the touching matters, but in the beginning, right after birth, a gentle soothing touch feels good no matter who is doing it because the context doesn't matter; this touch means we are not abandoned. Studies have demonstrated that infants who were stroked smiled, vocalized more, and cried less than infants who were tickled or poked.[12] Infants preferred stroking to tickling and poking. Positive touch includes stroking, holding, hugging, kissing, hand-holding, and care giving. Lack of positive touch negatively affects growth, development, and emotional well-being. Conversely, soothing massage therapy with preterm infants enhanced weight gain. The areas of the body where massage was found to be most effective were the forehead, the scalp, the back of the head, the upper arms, and the hands.

Touch has meaning not just for humans but for other animals as well. Cats purr when petted. Dogs roll on their backs, I suspect, to get their tummies rubbed. All animals are quieter when held. Touch clearly gives pleasure, and it affects the stress axis. It is not just the individual who is touched that benefits; under most circumstances, the person who touches also benefits. See how good it feels to pet a dog.

There are many ways we touch in our culture. The most common is the handshake. The handshake has many meanings, from everything is all right, to we have a deal, to goodbye. The point is that touch bonds individuals. Its intent is contextual, but its meaning is personal; it creates an attachment. Shaking hands with an enemy is not done until peace is accepted on both sides. Comparisons between preschool-aged children in the United States and France revealed that French children were aggressive to their peers on playgrounds only 1% of the time, compared to 29% for American children. This finding

correlated with the amount of time parents touched their children: the French, 35%, and the Americans, 11%.[13] Our current legal system in this country actively discourages unsolicited touch in this culture. It is impossible to determine what an individual's response is to someone else's touch, so we refrain from touching anybody.

Touch is reputed to have many healing qualities, and these have been organized into therapies. The most commonly used touch therapies include chiropractic, osteopath, cranial sacral therapies and acupressure, massage, Reiki, Rolfing, and so on. Some of these are discussed in more detail later. What is interesting is that when we touch and are touched, we experience sensations that are not directly assigned to the physical act. Even more remarkably, watching someone being touched can be relaxing.

What is the neurobiology of soothing touch? How does this soothing touch, what we call havening touch, produce a feeling of safety and allow us to escape from the inescapable? The technique that most resembles havening is Swedish massage. Swedish massage techniques include long strokes, kneading, friction, tapping, percussion, vibration, effleurage, and shaking motions:

Effleurage—Gliding strokes with the palms, thumbs, or fingertips.
Petrissage—Kneading movements with the hands, thumbs, or fingers.
Friction—Circular pressures with the palms of hands, thumbs, or fingers.
Vibration—Oscillatory movements that shake or vibrate the body.
Percussion—Brisk hacking or tapping.

Studies from the Touch Research Institute in Miami, Florida, have shown[14] that massage therapy enhances attentiveness, alleviates depressive symptoms, reduces pain, and improves immune function. Patients in the intensive care units of hospitals describe touch as critical to their feeling safe. There are measurable physiological changes associated with touch. Cortisol secretion, the stress hormone, is diminished with a soothing touch such as massage. There is an increase in dopamine (thought by some to also act as a reward

chemical) and serotonin, as well as a decrease in norepinephrine, during massage. While these studies looked at peripheral concentrations of these chemicals, it is not unreasonable to assume that they are also altered centrally in the brain. If, as described earlier, depotentation occurs because of the production of a low frequency wave, is there a relationship between the neurochemicals released and the electrical activity in the brain? There is an abundance of data to support that serotonergic modulation of GABA neurons[15,18] and increased GABA release is associated with an increase in low-frequency (delta) waves in the amygdala.[16,17]

Can havening touch, the touch that tells us we are safe, be used to create a neurobiological equivalent of a haven, and also produce a depotentiating signal? If so, then we will have found a powerful method for treating a traumatization.

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