

NOTES, TRAUMA AND THERAPY

Trauma, a New Kind of Memory

Synopsis: 1) Definitions and a summary of the extensive clinical research on trauma have been discussed previously in Part 1, Twilight sleep (1.5.3). To the clinicians, trauma is "psychological trauma" that has "narrative" and "intrinsic" components and involves the functions of the amygdala and hippocampus. By contrast, the view presented in this monograph includes a subcortical memory substrate that is not psychological, but purely physical, as seen in the recapitulation of M's skull sensations. The input of analgesia, catalepsy and skull feelings into the open memory substrate at birth was sensory and physical. The importance of this sub-cortical physical domain will be discussed.

2) In this previous part (1.5.3.1) the RaRN model is suggested as a means to explain not only the persistent nature of traumatic PTSD and flashbacks, but as an embodiment of the postulate that hallucinogenic flashbacks and those associated with general trauma are one and the same. It is argued on the basis of M's disappearing flashbacks that they, as well as PTSD symptoms, are indications of healing attempts by the organism to erase the traumatic memory. According to the RaRN model their persistence and lack of resolution are partly a consequence of environmental circumstances during the time of the recall or flashback.

3.01 Corollaries and Postulates of the RaRN Model

The parturient mother and fetus are under hallucinogenic influence

Traumatic memory has reversible aspects amenable to partial or complete permanent cure with the use of 5-HT1a agonists as an adjunct to other therapeutic protocols.

Persistence of flashbacks or PTSD arises from the "algebraic" addition of new sensory information to the old in re-consolidation of traumatic memory. The noxious elements can be reduced, increased or erased.

Nightly REM states produce imagery of flashbacks as dreams and are initiated by secretion of endogenous hallucinogens to heal trauma.

Casual self-experimentation with hallucinogens could diminish the quality of life, long term, by opening long-hidden memory substrates containing intense trauma, leading to PTSD.

Trauma is established by raphe suppression, not by the intensity of traumatic insult. Thus, there is far more sub rosa trauma than generally known .

Trauma memory consolidation occurs even without insult during receptor activation of 5-HT raphe neurons.

As a parasympathetic brainstem process, recall of traumatic memory is involuntary and recapitulative. This is a re-living of trauma elements.

Raphe-mediation of (traumatic) memory consolidation may occur in limbic as well as in sub-cortical storage substrates.

"Psychological trauma" is a misnomer, owing to clinical disregard of the sub-cortical storage of physical and visceral memory.

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3.02 Caveats on Hallucinogen use.

The RaRN model addresses the specific requirement for determining whether or not a

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physical insult will be stored as trauma, namely, reticular activation following raphe suppression. A physical insult becomes trauma only when a certain reticular nucleus (RN) is in the active state. Hitting one's finger with a hammer isn't traumatic, because the normal state of reticular nuclei (RN) is long-term depression (LTD) or inactive. It's not because a hammer blow or slamming a car door on one's hand or being clobbered by a defensive fullback is less impressive. The comparatively less intense skull pressure and anesthesia in M's case qualifies as trauma. In fact, the sensory input for storage via an active on/off nucleus such as RN need not be painful, but a necessary wholesome stimulus to be sequestered during parturition. This aspect will be presented in Part 2, where it is proposed that a natural process of raphe suppression produces an active reticular nucleus awaiting proper and wholesome stimulation of the fetal sensations within a critical window of development during the mother's labor.

As speculated upon previously, the storage of trauma is not necessarily limited to brainstem sites, but could include hippocampal long-term storage as well, to account for sequelae of the more stereotypical trauma involving psychological and emotional aspects. The median raphe nucleus is the primary source of serotonin afferents to the limbic system, where they suppress specific theta oscillations originating in the hippocampus (Freund, et al 1990). As already mentioned, the hippocampus mediates short-term memory and directs it to neocortical storage for long-term memory (Kandel et al., 2007). Thus, certain neuron groups (a nucleus) within the hippocampus that are enervated by the rostral raphe nuclei could be a two-state nucleus in the same way as the putative RN within the brainstem. Also, the dorsal raphe nucleus sends fibers to the amygdala, that part of the limbic which functions in the context of fear-related emotion. Accordingly, the definition and specific requirements of both physical and psychological trauma are embodied in the simple notion of reversible, raphe modulated inhibition of specific two-state nuclei in both the limbic and brainstem areas.

Another perspective on trauma follows the possibility of raphe (Ra) 5-HT neuron suppression as the primary first step in the consolidation and re-consolidation of traumatic memory. The strength or intensity of impulses depends on the rate of neuron firing. Impulses of both weaker and stronger potentials in a continuum of intensities can be stored as this kind of memory, since the only thing that is required is whatever will induce the secretion of the endogenous hallucinogen, or activate the GABAergic system, which responds to stress - to shut down Ra firing. M's LSD recall is evidence of a traumatic memory far less intense than those demanding high profile therapy on PTSD. Undoubtedly the perceptions of fear, horror and the threat to survival from the outside world are those most associated with serious trauma. If it can be inferred that limbic and sub-cortical pathways for impulses from these perceptions will shut down Ra activity, then more subtle perceptual and sensory influences might be evaluated as Ra 5-HT<sub>1a</sub> agonists by methods already developed for recording the firing of their 5-HT neurons. While this question might not send an over-worked scientist to the laboratory, the possibility is raised that trauma defined by the RaRN model is a memory that can be comprised of any number of psycho-physiological forces associated with the nature of the triggering input. All that is needed is the secretion of the endogenous hallucinogen and/or activation of the GABAergic system. The implication is that a staggering number of the world's peoples are walking around with trauma from lesser encounters with hurtful and fearful qualities that are sufficient to interrupt the normal activity of raphe nuclei. The Ra firing can be stopped even without this input, as seen in M's case, where EH secretion was a process occurring naturally at birth. Is this the secret behind the notions of Dr. Freud? Perhaps, if raphe activity were taken into consideration, his approach might have seen some more lasting success. Dr. Grof's extensive LSD work on the psychology of neuroses and their origin during birth provided the evidence on the unconscious effects of hidden memory and conforms to the added proposal of the RaRN mechanism for memory consolidation and release.

Finally, the postulate of an open memory substrate during raphe suppression raises the question of the desirability of hallucinogen use outside the context of well informed therapy or spiritual practice. It has been argued earlier that the intake

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of sensory and emotional data could be additive during, say, a flashback or PTSD. wholesome input under hallucinogenic influence would diminish the re-consolidated trauma, while noxious symptoms of the flashback would reinforce and facilitate the next release of symptoms. This is consistent with the much better outcome of the therapeutic context, as opposed to the recreational and would explain the success of MDMA and its celebrated positive effects. The same would apply to the intake of a hallucinogen, particularly of the indole type that acts to inhibit raphe 5-HT neurons. It would fix deep into an individual's psychic constitution any preoccupation of negative emotions and intentions dominating thoughts when under the influence of the drug. This kind of memory persists to influence thoughts and behavior of the individual for years and decades, as borne out by the discovery of perinatal influences by Dr. Stanislav Grof (Grof, 1980, 1991). The chronic effect of negative influences have been recorded by Carl Jung in his analysis of dreams, particularly those of German people after the first world war (Jung, 1953). These negative influences should be resolved before self-experimentation to avoid the possibility of a lifetime of diminished quality. Indeed, negative preoccupations may be milder symptoms of PTSD; they have the herald earmark of being involuntary. Casual hallucinogen use would be the best method on hand to make it worse.

### 3.1 PTSD: Therapy

Recalling our war veteran, he or she hears the backfire of a car exhaust and the physiology changes abruptly to sky-rocketing blood pressure, tachycardia, confusion and even momentary unconsciousness. Now, the vet walks in front of a car and is hit, taken to the ER and later, doesn't remember the incident. The onset of symptoms is involuntary and recapitulative. Indeed, running into the car, in this case, was part of the memory, inducing sub-conscious re-enactment of the physical actions that led to the original trauma. This tale of post-traumatic stress disorder (PTSD) is more truncated than most, since that of the larger segment of sufferers is prolonged and detrimental to their lives. The phenomenon of PTSD is, of course, very complex involving possible physical damage to many parts of the brain. Actual brain damage is largely outside the context of the RaRN model as a basis for a therapeutic approach.

So far, therapy of PTSD victims has followed other models that are sketchy, incomplete or downstream of the source. Some of these approaches can be viewed vis-à-vis the RaRN model, the latter based on removing traumatic memory by gaining pharmacological access to the actual neural substrate that holds it:

1) The RaRN model provides a scenario for the self-perpetuating nature of severe PTSD. Beginning with the recapitulation of the noxious sensations and excited physiology triggered by some subliminal internal or external signal, the raphe are in a state of EH suppression accompanied by RN activation. At this time, the memory substrate is re-opened according to the on-off, bidirectional nature of the model and the noxious components are further re-consolidated as a memory, now reinforced to increase the sensitivity to the trigger. Here, the alternative view of Price and co-workers is confronted. XXX

2) One therapeutic prediction of the RaRN model for PTSD follows from RN activation by raphe suppression as a first step to release the trauma, just as M's trauma was released with LSD. Conversely, this would be the first step in consolidating the memory. The approach deals with the source of the memory itself and its application might be the indispensable element in concerted approaches to eliminating the memory. The lesson of M's LSD recall of a birth trauma is that, once it begins, slower natural processes were set into action to eliminate the memory altogether. Accordingly, a therapeutic approach based on this paradigm would involve the judicious application of 5-HT<sub>1a</sub> agonists to eliminate, not just treat, the trauma.

In fact, the RaRN model generates specific predictions that have already been borne out. Notably, until the discovery of propranolol as a beta-adrenergic antagonist for the alleviation of PTSD symptoms, the most effective pharmacological treatment of PTSD has been with serotonin cogeners such as sertraline, paroxetine and

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fluoxetine

([www.oqp.med.va.gov/cpg/PTSD/PTSD\\_cpg/content/interventions/ptsd\\_pharm.htm](http://www.oqp.med.va.gov/cpg/PTSD/PTSD_cpg/content/interventions/ptsd_pharm.htm)). Since serotonin neurons are involved in the inhibition of raphe firing (LSD is a  $\alpha$ 5-HT1a agonist), this therapeutic model is supported. Significantly, MDMA (Ecstasy) has been mentioned as the focus of vigorous investigation for the successful alleviation of PTSD disability (see MAPS.com). Like LSD and other hallucinogens, MDMA inhibits the raphe nuclei (Sprouse et al., 1989). The 5-HT1a agonists, 8-hydroxy DPAT or lisuride, might be considered as non-hallucinogenic alternatives for the resolution or release of traumatic memory. However, the use of MDMA may be preferable by replacing the open substrate contents with new, wholesome input. This would be amply provided by the highly positive mental state that has become famous in MDMA inebriation. Other approaches to PTSD can be viewed vis-à-vis the model.

The recent application of beta adrenergic antagonists such as propranolol has shown spectacular alleviation of PTSD symptoms, provided the drug is given within a window of time, depending on the severity. Acetylcholine, secreted by the amygdala in response to the emotional content of the memory, is blocked from hippocampal beta-receptors by propranolol, thereby preventing further memory consolidation of fear and emotion by the hippocampus. However, elimination of the memory itself is doubtful and FDA approval of propranolol is problematical, owing to its hypertensive and pulmonary effects. (cf. [harvardmagazine.com/2004/07/cushioning-hard-memories.html](http://harvardmagazine.com/2004/07/cushioning-hard-memories.html)): Notably, the use of propranolol would be inimical to the RaRN approach, since it is an antagonist of the 5-HT1a receptor (Tricklebank et al, 1985).

Other approaches are based on the manipulation of other palliative systems, notably the  $\alpha$ 2-adrenergic drug, clonidine. Clonidine has been used to treat PTSD symptoms for nightmares in combat veterans and children (Mousavil et al, 2005) and frequently for pain and anxiolytic treatments in children (Lange et al, 2000). As a 2- $\alpha$  adrenergic agonist it fools the brain to disregard the excitatory locus ceruleus. Other drugs of this type include prazosin, and quanafacine. A different approach employs cyproheptadine, one of the strong anti-histamines that promote sleep (Claritin). Interestingly, this drug is a high affinity antagonist for the 5-HT2a receptor.

Some approaches for PTSD treatment are based on trial efforts that have no biological model to use in their effect. While psychological approaches are effective within limited contexts one approach uses willful memory suppression, i.e., deliberate avoidance of the traumatic memories (Depue, et al 2007). This has been questioned, based on an opposite, more successful approach that involves deliberate recall of the trauma (Holmes et al., 2007). The success of this latter approach would be consistent with the pharmacology of raphe suppression and the bi-directional nature of the proposed model. However, this clinical approach per se suffers from the inability to give the patient input strong enough to mimic the terror and helplessness associated with the original incident (Van der Kolk, 2001). Previously, under "Twilight Sleep and Scopolamine" it was suggested that retrieval of hippocampal memory via the hypothalamus could trigger the resurgence of PTSD symptoms. In any case, psychological approaches xxxinvolve treatments downstream of some central neural event that may govern the actual release of traumatic memory, but is not accessible by clinical approaches. While these paradigms are somewhat effective and not unreasonable, the model presented here for memory storage and release offers a different perspective for approaching PTSD treatment.

It is the idea that therapy for traumatic memory would be approached by initial treatment with a 5-HT1a agonist and can be erased, provided the protocol is designed to elicit recall in small increments insufficient to overload the patient. The lesson of M's LSD (gentle) recall of a birth memory is that its activation into conscious re-enactment was the first step leading to its complete stepwise erasure. The flashbacks were repeated activations that eliminated one-by-one the memories of specific skull areas in reverse order of their intensity: the most persistent flashback was the palate area, where the greatest force was exerted during birth. Based on the relatively gentle nature of M's recall of traumatic memory and the notion of putative healing by REM sleep discussed previously, the memory is

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completely erased. As discussed within the context of the RaRN model, re-experiencing the original insults in their totality may only reinforce the trauma and interfere with its natural erasure through the process that produces flashbacks.

Recurring noxious flashbacks or PTSD symptoms are postulated to occur when the memory substrate is open and vulnerable to additional consolidation of the noxious symptoms. Adding to this problem are the reflexes established by the traumatic victim to adapt to these renewed insults.

### 3.2 PTSD Prophylaxis

As a prophylactic for soldiers going into battle, scopolamine or a similar M-3 anticholinergic should be given serious consideration. The rationale for this attractive possibility is presented in this monograph's section, "THE TS MOTHER IS TRAUMATIZED: TWO KINDS OF MEMORY". This suggestion is based on the anticholinergic action of scopolamine to inhibit the hypothalamic connection between trauma stored in the brainstem and its later, more casual associations mediated by the hippocampus, hypothalamus and amygdala. Furthermore, the RaRN model provides a way to look at the inverse of PTSD therapy by investigating the use of antagonists to the 5-HT<sub>1a</sub> and 2a receptors. This would guard against the opening of memory substrates by protecting raphe nuclei from suppression following DMT+ secretion that might occur in response to a dangerous situation or to the shock of injury. In animal research clonidine has been shown to prevent post-traumatic symptoms if given before a painful shock (xxx).

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