

# Forensic Significance of the Limbic Psychotic Trigger Reaction

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During the “decade of the brain,” competent expert testimony should encompass widely neglected, even novel, neurophysiologically plausible explanations for otherwise unexplainable acts. In the case presented here, a sudden, out-of-character, motiveless, unplanned homicidal attack was committed by a patient who demonstrated flat affect, preserved consciousness, and memory of the episode. Transient autonomic hyperactivation and psychosis were suddenly experienced when the victim happened to move his mouth while eating. Following a sudden memory revival of repetitive but moderate bodily stresses, the patient suffered visceral hallucinations of his entire body being cut into pieces with the delusional belief that he was about to be “cannibalized.” The patient’s sudden and very transient symptomatology is characteristic of 13 interrelated symptoms and signs (including autonomic, e.g., visceral, hyperactivation and psychosis) proposed as a new subtype of a partial seizure, called “limbic psychotic trigger reaction,” which has been consistently delineated thus far in 18 white social loners (14 homicidal men, 3 fire setters, and 1 bank robber), who ruminated about past, moderately painful, but repeated events. This rendered them liable to seizure kindling, particularly of the limbic system. Apparently a post-ictal transient frontal lobe deficiency is secondary to the limbic storm. The forensic impact of seizures on cognition (appreciation of the quality of the act) and on volition is discussed.

The goal of this study is to demonstrate the ways in which the use of detailed clinical techniques of differential diagnosis can lead to increased clarity in forensic evaluation. Too often we rely on vague classifications (e.g., atypical psychosis, organic delusional syndrome, not otherwise specified) of inexplicable be-

haviors. These broad classifications are not specific enough and often not “beyond the ken of the average layman” (*Ibn-Tamas v. U.S.*).<sup>1</sup> By contrast, expert witnesses can propose a specific and plausible explanation of deviant behavior to aid the court or jury—more so since *Daubert v. Merrell Dow Pharmaceuticals*.<sup>2</sup>

The sudden and transient symptomatology of the homicide case presented herein is consistent with a simple partial limbic seizure with psychosis (hallucinations and/or delusions, frequently of grandeur), autonomic hyperactivity, and bizarre acts

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committed with flat affect and without quantitatively altered consciousness. This process is described as “limbic psychotic trigger reaction” (LPTR).<sup>3-17</sup> Apparently post-ictal frontal lobe dysfunctioning with inefficient behavior is secondary to the limbic storm (see *Addendum*).

Nine out of the 18 LPTR cases had some positive objective test (such as scalp electroencephalogram (EEG), pneumoencephalogram (PNEG), computerized axial tomography scan, or magnetic resonance imaging) at some time during their lives; 10 had a known history of brain injury. The latter may facilitate seizure kindling, although brain injury has not been a prerequisite for experimental kindling to occur.

### Neglected Issues with Forensic Implications

The literature on violence<sup>18-20</sup> neglects the unsuspected kindling of seizures evoked by minor to moderate stresses. Any type of seizure impairs volition and certain aspects of cognition. In “complex” partial seizures (with impairment of consciousness), such as temporal lobe epilepsy (TLE), evaluators may miss such impairment because routine behaviors (but rarely homicide) are still possible.

By contrast, potentially lethal acts can be committed during “simple” partial seizures (without quantitatively altered consciousness), such as kindled seizures (Table 1), proposed to occur during a LPTR.<sup>3-17</sup> The high degree of dangerousness (especially to others) present during simple partial seizures, as in LPTR, may result from the absence of significant impairment of consciousness and also from

the seizures’ unsuspected occurrence, in association with an insidious repetition of only mild to moderate stresses. Further, the kindling mechanism<sup>21-24</sup> observed in nonhuman mammals, and in a few humans,<sup>25-27</sup> is evoked by just one more subthreshold stressful stimulus which resembles past ones.

*Kindling*<sup>21-27</sup> was first experimentally demonstrated in mammals by Goddard<sup>21</sup> and later by Goddard and McIntyre.<sup>22</sup> An intermittently (not continuously) applied subthreshold electrical, chemical, or experiential stimulus was found to elicit seizures (on EEG recordings), some, but not all, of which progressed to include motor convulsions. The limbic system, particularly the amygdala, was noted to be most susceptible to kindling.<sup>21</sup> Later, a few human cases of accidental kindling were recognized.<sup>25-27</sup> It is important to note that convulsions during kindled seizures occur mostly in lower mammals, less frequently in higher nonhuman mammals, and rarely in humans.<sup>28</sup>

The following case, hopefully, will illustrate the gain in forensic insight that results when vague classifications are eschewed and replaced by increasingly specific ones. XR’s symptomatology had previously been published as a puzzling case of a person who had chronic schizophrenia, which did not satisfactorily explain his sudden, out-of-character homicidal act.<sup>17</sup> This is a reexamination (after two decades) of a case with an episode of out-of-character, sudden, homicidal violence within a novel, first-time psychotic context. Previously, I had diagnosed “Mr. X” (here re-named XR) as a puzzling case

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**Table 1**  
**Differences Between the Proposed Kindled Partial Seizures of LPTR—Affecting Action and Cognition—and the Traditional Conceptual Models for Epilepsy**

	Kindled Partial Seizures: LPTR	Nonkindled Seizures (any type)
Trigger	External or internal memory-reviving stimulus necessary to evoke seizure	Not necessary
Acts	Devastating; involve basic limbic drives: killing, global destruction (fire); acquisitive behavior; reduced socialized behavior	Nondirected (e.g., automatisms)
Consciousness	Quantitatively intact	Impaired
Amnesia for seizure	None	Common for acts
Hallucinations	Unformed or formed; can include commands	Same, if present
Delusions	Often of grandeur	Persecutory, mostly grandiose, if present
Scalp EEG	At times positive in past	Same or negative during psychosis (forced normalization)
Other symptoms shared with schizophrenia	Flat affect Social loner	Not a required criterion Not primarily

of “chronic schizophrenia, paranoid type.”<sup>17</sup>

I had previously published this case (with the patient’s full consent) long before LPTR had been conceptualized. Because of the puzzling symptomatology, I had preserved my extensive notes of three to four hours of interviews per week over a three-months period, as well as notes from interviews with the patient’s older sister. I also had examined the patient’s past hospital and police records, including accounts from several witnesses present at the crime scene on a busy street. Differential diagnoses of LPTR have been discussed previously.<sup>1-16</sup>

## Case Presentation

XR, a strong, physically well appearing, white, 40-year-old man a socially isolated commercial painter, had been admitted to an urban university-affiliated maximum security ward promptly after his arrest at the crime scene for “felonious physical assault” on a stranger. The young, physically strong, male victim had survived thanks to the interference of several witnesses, but required lengthy hospitalization and intensive care.

All routine examinations, including physical, laboratory, neurological, and scalp EEG were reported as within normal limits. His psychological examina-

tion report was "paranoid symptoms," consistent with schizophrenia as well as with some organicity.

The mental status examination shortly after XR's well remembered violent episode, revealed a physically well appearing, strong, white man with flat affect, and a moderately impaired ability for abstract thinking. Otherwise, his formal thought processes were intact and coherent. He revealed first-time delusional beliefs and olfactory hallucinations ("the sick smell of scurvy," associated with loss of teeth) and multiple, bizarre somesthetic hallucinations.

XR was fully oriented to time, place, and person. His concentration and memory functions for immediate, recent, and remote recall were all intact, as was his memory of his attack on the victim and of the circumstances around it. His insight and judgment were grossly distorted.

XR's history was supplemented by interviews with his older sister and by records from five previous psychiatric hospitalizations, which noted a total of three prior outbursts of violence. These latter, however, had never reached homicidal proportions, so that his present arrest was also his first (and apparently his last) one.

XR had been "born in a taxi," the youngest of four children of an intact middle class family. He was trained as a commercial artist, but his older brother was much more accomplished in this field. This brother had recently suffered a stroke, which had left him paralyzed. During his early childhood, XR's mother had "intensively massaged his 'bow legs'" for years, until they were "straight-

ened out." When at age seven or eight XR had "rheumatic fever with a heart murmur," his "overly anxious mother" ordered him to "sit still all the time."

At age 18 he had begun to date several much older women, who left him after he and these women had noticed "a lack of pressure in the penis." Two years before admission to the hospital, he had been briefly married (for about six months) to an older woman, the widow of his building superintendent, who had suddenly died of a heart attack. XR was worried about also dying the same way, linking this fear to his "rheumatic fever with heart murmur" that had forced him to sit still.

*Circumstances Surrounding the Homicidal Violence* XR had suddenly and repeatedly stabbed a male stranger (with the pocket knife he routinely carried) upon seeing the victim "moving his mouth," while offering food and drink to XR on the street.

While seeing the "mouth movements," XR experienced extensive cutting sensations moving through his entire body. These somesthetic hallucinations meant to him that he was going to be eaten by the victim and "his wife" who were cutting his body "into pieces" in preparation of "cannibalizing" him. XR, a commercial artist, had drawn pictures showing his "dismemberment."<sup>17</sup>

XR hallucinated the victim's cutting knife moving through his body, beginning with cutting of the flesh of one hand (he had been bitten in a finger), then cutting off both lower arms, cutting out his heart and his genitals (he had had an inguinal hernia operation under local anesthesia). The cutting of the genitals was

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felt as "going all the way to the back," followed by both his lower legs being "sawed off" while he depicted himself as lying passively on a table. He also depicted himself as quietly "sitting still on a chair, not moving" until "everything" was being cut off and out of his body, making him into a "skeleton" (wearing a vest), with his brain exposed.<sup>17</sup> Note that XR had such hallucinations for the first time during this episode.

## Results

*Specific History, Summarizing Repetitive Mild to Moderately Stressful (Bodily) Experiences (a Prerequisite for Kindling)* XR's symptoms meet the criteria for LPTR kindled seizures (see *Addendum* and Table 1).

1. XR's experiences involved virtually his entire body: he repeatedly feared that he was "getting a heart attack," beginning in childhood, when he had a "rheumatic heart" with fear of dying from a heart attack. The latter experience was also linked to prolonged immobilization, contributing to his all-pervasive passive attitude.

2. His mother "intensively massaged his bow legs" for years until they were "straightened out."

3. As a child, he had been bitten on a finger by his brother.

4. Moreover, recently XR had undergone an inguinal hernia operation under local anaesthesia, after which he had believed that pieces of his testicles had been cut out. (Furthermore, he believed that all his women friends had left him due to "lack of pressure in the penis".)

Thus, the history is remarkable in two

aspects: there is a specific link of his somesthetic hallucinations to his previously injured or malfunctioning body parts (as listed above); and his "feeling" of the cutting knife moving through his body is linked to his belief of being about to be "cannibalized."

His visceral sensations are particularly associated with food intake, which is congruent with such association shown in neurophysiological experiments.<sup>29</sup> Also linked to food intake is the trigger stimulus of "seeing" the victim's "mouth movements," further signifying to XR that he was "about to be cannibalized."

## Discussion

*Specific Forensic Implications: Neurophysiological Differences Between PTSD and LPTR* The literature on the behavioral effects of stress has focused almost exclusively on unusual, extreme stress linked to posttraumatic stress disorder (PTSD).<sup>30-36</sup> In many cases the existence of severe stressors has been viewed as an excuse for antisocial conduct associated with PTSD. Due to the often overlooked essential distinction between PTSD (without seizures),<sup>30-36</sup> as against those with the proposed LPTR (with kindled seizures),<sup>3-17</sup> much of the reasonably raised criticism of forensic defenses under PTSD,<sup>33</sup> which differ essentially from those under LPTR,<sup>3-17</sup> could unjustly be extended to criticism of LPTR as well.

From a forensic viewpoint, a defense based on LPTR is not based on blaming any stress created by an abusing situation or person. By contrast, all 18 identified LPTR patients have voluntarily confessed

their acts and have assumed subjective responsibility for them (some patients to a degree that drove them to serious suicidal attempts).

The essential neurophysiologically proposed difference between PTSD and LPTR is that PTSD is "an overshoot to an evolutionarily adaptive mechanism" to deal with life-threatening trauma.<sup>32</sup> Stress-related neurohormonal activation of epinephrine, adrenocortical, and vasopressor hormones are implicated in PTSD. By contrast, the proposed LPTR symptomatology implicates limbic seizure kindling with potential extremely regressive consequences.

***Neurophysiological Differences Between Stress Responses and Seizure Kindling*** Post<sup>35</sup> recently differentiated kindling from stress responses (which can involve sensitization and conditioning). Post pointed out that, even though both stress responses and kindling are context-specific, only kindling is associated with seizures.

Further, the latter two mechanisms (stress response and kindling) have opposite neurophysiological effects on the same system, which involve neural growth factor and neurotrophic factors 1 to 4.<sup>35</sup> Such opposing effects of stress response compared with kindled seizures appear to be related to "figuring out the meaning of a stimulus," or whether to grow a synapse in response to a stimulus.<sup>35</sup>

Memory flashbacks may occur in both PTSD and LPTR (which includes kindling). Flashbacks represent "paroxysmal spontaneous discharges of memory circuits."<sup>35</sup> (Recall that seizure kindling oc-

curs initially only after repeated, intermittent exposure to a seemingly harmless stimulus. It is only later that a kindled seizure can reoccur spontaneously upon reexposure to a globally similar stimulus.)

In PTSD, where past trauma dominates, Post<sup>35</sup> postulated a secondary deprivation of other environmental sensory input and an "emotional numbing" to such input. Thus, Post does not link PTSD with fleeting seizure kindling. Rather, he considered the persisting symptoms of PTSD as "a specific cognitive disorder" where a generalization of flashbacks can be linked to false memories.

The mechanisms implicated in the above model of PTSD<sup>30-36</sup> differ from the plausible mechanism in the typical, mostly quite brief, kindling effect in LPTR. The neurophysiological distinction between PTSD and LPTR<sup>3-16</sup> decreases in Post's<sup>35</sup> extended hypothesis of PTSD: repeated re-presentation of intrusive memories are replayed in PTSD. Such a replay may also occur in LPTR,<sup>3-16</sup> albeit only during the precursory aura of a kindled seizure. Such replayed memories are associated with changes of neural network circuits.<sup>35</sup> Such processes, Post continued, involve dopamine and noradrenergic systems as well as the hippocampal long-term (declarative) memory storage, with its diverse connections to cortical areas. Here, Post's extended hypothesis of PTSD mechanisms could also be postulated as a potential kindling mechanism, but only during the phase precursory to full LPTR symptomatology.

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In a review of PTSD, Bremner *et al.* (p 200)<sup>34</sup> have recently implicated virtually the same brain systems as those implicated in LPTR since 1981 (particularly the amygdala and hippocampus). The similarity between PTSD and LPTR, however, is mainly limited to a partial overlap, given that in both PTSD as in LPTR “simple sensory phenomena related to traumatic events” can “result in specific intrusive memories and flashbacks.” One difference between PTSD and LPTR lies in what follows the intrusive memories: seizure kindling in LPTR.

***Proposed Model of LPTR, Implicating Specific Limbic Structures and Mechanisms*** The model of LPTR is congruent with the following experimental findings: (1) lack of “habituation”<sup>37, 38</sup> (i.e., “non-forgetting”) of past moderate, although repeated, stressful experiences (hippocampus<sup>39</sup>); (2) stresses attain exaggerated meaning (amygdala<sup>27</sup>); (3) a specific, individualized external stimulus (analogous to the effect of a “primer,”<sup>40, 41</sup> only globally resembling the past stresses) revives such experiences; and (4) a kindling mechanism sets in,<sup>21-27</sup> capable of evoking a (5) limbic seizure with (6) secondary, reciprocal<sup>42, 43</sup> frontal lobe system dysfunction (reflected on the Trail-Making Test B<sup>44</sup> and on the Narratives Test<sup>45, 46</sup>) enabling (7) release of homicidal violence or other regressive behaviors,<sup>47</sup> some of which are reminiscent of prey or defensive killing (amygdala<sup>47, 48</sup> and/or hypothalamus<sup>47</sup>).

***Forensic Implications*** As Diamond<sup>49</sup> emphasized, forensic evaluators are ethically called upon to respect the defendant’s rights. Such respect acquires

a new meaning when the defendant’s out-of-character and otherwise unexplainable pattern of behavior calls for an excuse based on partial seizures. Furthermore, the forensic evaluator’s awareness is particularly taxed in cases where there is a preexisting chronic psychosis, which in itself does not explain sudden, first-time felonious acts.

XR’s sudden, first-time homicidal act, committed without apparent provocation, is a case in point. XR’s long-time chronic schizophrenia, with its associated cognitive impairments would not be a basis for a successful insanity defense. Any excuse in this situation would be based on XR’s lack of volitional capacity. Currently, the use of a volitional excuse is controversial and out of favor. Yet there are situations in which lack of volitional capacity might negate responsibility. Failure to consider volition was characterized by Quen<sup>50</sup> as “nonmedical thinking.”

In *People v. Gorshen*,<sup>52</sup> Diamond<sup>49</sup> testifying successfully for the defense, stated that Gorshen acted “as an automaton” in the fatal shooting. As reported by Bromberg,<sup>53</sup> “the trial court was impressed” by Diamond’s testimony, without which there was no explanation of why this crime was committed by this chronic paranoid schizophrenic man.

In line with these forensic concerns is Halleck’s<sup>51</sup> emphasis on the severity of stressors. In the context of our discussion of LPTR,<sup>3-16</sup> it may be revealing to consider not only the severity of the stressor itself, but also certain nonsevere, albeit repetitive, stressful experiences with their insidiously pernicious potential to kindle seizures.

## Conclusion

Two novel sets of heuristic and of forensic implications are proposed by the case presented here:

*Heuristically*, the sudden, fleeting, motiveless homicidal acts with flat affect suggest a model of a brief limbic partial seizure with aura and atavistic regression, such as predatory or defensive killing.<sup>3, 4, 11, 23</sup> Such a model would avoid the common fallacy of certain violence research, which links all violent acts to displeasure and negative emotions, and/or to severe trauma,<sup>33</sup> overlooking the potentially seizure-kindling effect of repeated minor stresses.<sup>21-27</sup> An animal model for this specific kind of violence is proposed including the study and treatment with an anticonvulsive (e.g., carbamazepine) of certain domesticated mammals (dogs) who suddenly attack or kill their owners.

*Forensically*, the careful evaluator has to rule out a partial seizure,<sup>54-60</sup> such as implicated by the specific 13 symptoms and signs (see *Addendum*) of the proposed subtype of simple seizures without quantitative impairment of consciousness and of memory for the acts.<sup>3-16</sup>

### Addendum: Symptomatology of LPTR

It is essential to note that the proposed LPTR<sup>3-17</sup> is not just a haphazard assembly of a dozen symptoms and signs. Understandably, loose assemblies without neurological interconnectedness have been under critique by Restak<sup>18</sup> and would also be liable to such questions as raised by Zonana.<sup>19</sup>

By contrast, LPTR constitutes a neurophysiologically linked proposed syndrome, in which all parts are interrelated. Further, the specific set of 13 criteria of LPTR has been consistently found reproduced in 14 homicidal cases, as well as in three

cases of firesetting<sup>12</sup> and one case of bank robbery.<sup>14</sup> Moreover, the neurological underpinning of the proposed LPTR is congruent, at least in principle, with Cummings'<sup>20</sup> emphasis on specific, circumscribed brain areas and their neurotransmitters subserving specific behaviors. As examples he cites the mediation of motivation by frontal and limbic structures, that of volition by frontal ones.

The proposed syndrome has been called "limbic psychotic trigger reaction" (LPTR).<sup>3-17</sup> LPTR consists of the following 13 symptoms and signs (which may vary by degree, but not in essence): (1) lack of drive motivation; (2) lack of pre-planning of the act and of its concealment; (3) typically no prior history of (homicidal) violence; (4) flat affect around the time of the act; (5) history of frequently known closed head injury (typically implicating temporo-limbic structures); (6) specific, individualized trigger stimuli are implicated in an evoked out-of-character act (the stimulus constellation acutely revives repeatedly experienced past stressful experiences, each one by itself not severe); (7) typically, some automatized action sequence culminates in motiveless acts—evoked (not emotionally provoked) by the trigger stimuli; (8) typically, an aura-like puzzlement ("cognitive mismatch"; lasting about five minutes), followed by ictus-like symptomatology (about 20 minutes); (9) transient autonomic arousal (e.g., nausea, vertigo, "ice cold" sensation or profuse sweating, "tingling," urinary incontinence, erection, or ejaculation); (10) transient hallucinations and/or delusions (frequently of grandeur); all such psychotic symptoms are first-time occurrences, or if superimposed on a preexisting psychosis, they take on a different psychotic content; (11) no significant quantitative alteration of consciousness, therefore no memory impairment, enabling virtually full recall of the acts; (12) indications of transient frontal-lobe system dysfunctioning around the time of the act, secondary to implicated limbic hyperactivation, and potentially persisting (for some hours) post-ictally; and mostly, (13) self-confession of the act, with subjective feelings of responsibility and remorse (unless there is underlying schizophrenia with habitually flat affect).

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