POST TRAUMATIC STRESS DISORDER AND CRIMINAL DEFENCES

JUDITH THOMSON*

A conceptual no-man's land seems to lie between psychiatry and the law and those who attempt to pick their way across it are probably as foolish as those who, in former times, sailed over uncharted seas marked on maps with the comfortless words "Here be Dragons". It is useful that some attempt be made to deepen understanding of one discipline by considering concepts developed in another. With this in mind, the author undertakes an investigation of the biochemical aspects of Post Traumatic Stress Disorder ("PTSD") to assist in understanding the nature of mental state defences and to examine the appropriateness of the currently bifurcated (sane/insane) automatism plea. It is the author's view that this inter-disciplinary investigation will contribute to increasing flexibility in thinking about an area characterised, at present, by unnecessarily rigid categorisations.

I. INTRODUCTION

On the last day of December, 1984, Roger Gordon Radford shot Mrs Nancy Grugan seven times, thus causing her death. Four years later, Mary Sandra Falconer blasted her estranged husband Gordon Falconer to death with a shotgun fired at close range. What distinguishes these from any other sordid suburban murders (apart, in Radford's case, from the belief that Grugan was his ex-wife's lesbian lover, and the cause of his marital breakdown) is the fact that, at trial,[1] both parties claimed that the shootings occurred while they were in a state of "dissociation" or "derealisation" caused by exposure to traumatic stresses. For Radford the relevant trauma was alleged to have occurred during military service in Vietnam some fifteen years earlier. Mrs Falconer's personality was said to have fragmented as a result of excessive emotional stress caused by the discovery of her husband's incestuous relations with her daughters, her fear of reprisals for the ensuing

* BA(Hons) BJuris(Hons) LLB(Hons) LLM(Distinction) (UWA).

1. *R v Radford* (1985) 20 A Crim R 388; *R v Falconer* (1990) 66 ALJR 20.

criminal charges brought against him, and the revelation that he may have been sexually molesting a young child who had been in her care. Considerations of the validity of these pleas aside, the cases represent an interesting development in the area of mental state defences, raising questions of automatism and the insanity plea, and the importance to the latter of external and internal causative factors.

II. HISTORY AND DEFINITION OF PTSD

PTSD was recognised as a medically distinct phenomenon in 1980, with the publication of the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders ("DSM-III").[2] Although an anxiety neurosis connected with a stressful event had been recognised since earliest times, its reported physiological and psychological manifestations had been so variously described as to be unhelpful.

As one of the primary symptoms of stress neurosis was tachycardia,[3] it was labelled "Irritable Heart Syndrome" by Da Costa in 1871,[4] and, because of its frequent occurrence in the troops, was labelled "Soldiers Heart", by Sir Thomas Lewis in 1919.[5] With the development of psycho-somatic theories and the recognition of a psychological component in the soldiers' malaise, came a change of nomenclature to "Traumatic Neurosis", evolving, in 1967 (as peace time victims were also recognised) to "Post-Accident Anxiety Syndrome".[6] This latter term had the merit of identifying anxiety as the basis of the disorder, thus differentiating it from a host of other neuroses, and as further constantly recurring symptoms were noted, the classification became more reliable and specific.

The list of thirteen criteria published in DSM-III are subsumed into four major symptom groups, but evidence of evolution in the direction of greater

2. American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* 3rd edn (Washington DC, 1987).
3. A conspicuous increase in heart rate. The physiological response to exercise, excitement, fear or emotion.
4. J M Da Costa "On Irritable Heart: A Clinical Study of a Form of Functional Cardia Disorder and its consequences" in C B Scrignar *Post Traumatic Stress Disorder Diagnosis, Treatment, and Legal Issues* (New York: Praegar Publishers, 1954) 2.
5. T Lewis "The Soldiers Heart and the Effort Syndrome" in Scrignar supra n 4, 3.
6. Scrignar supra n 4, 4.

precision is provided by the diagnostic criteria listed in the 1987 Revision of the Manual, known as DSM-III-R[7] and reproduced below.

A. The presumptive PTSD sufferer has experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone, for example serious threat to one's life or physical integrity; serious threat or harm to one's children, spouse, or other close relatives and friends; sudden destruction of one's home or community; or seeing another person who has recently been, or is being, seriously injured or killed as the result of an accident or physical violence.

B. The traumatic event is persistently re-experienced in at least one of the following ways:

   (i) recurrent and intrusive distressing recollections of the event (in young children, repetitive play in which themes or aspects of the trauma are expressed);
   (ii) recurrent distressing dreams of the event;
   (iii) sudden acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative [flashback] episodes, even those that occur upon awakening or when intoxicated);
   (iv) intense psychological distress at exposure to events that symbolise or resemble an aspect of the traumatic event, including anniversaries of the trauma.

C. Persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness (not present before the trauma), as indicated by at least three of the following:

   (1) Efforts to avoid thoughts or feelings associated with the trauma.
   (2) Efforts to avoid activities or situations that arouse recollections of the trauma.
   (3) Inability to recall an important aspect of the trauma (psychogenic amnesia).
   (4) Markedly diminished interest in significant activities (in young children, loss of recently acquired developmental skills such as toilet training or language skills).

7. American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* 3rd edn, revised (Washington DC, 1987) 250.

(5) Feeling of detachment or estrangement from others.
(6) Restricted range of affect, for example unable to have loving feelings.
(7) Sense of a foreshortened future, for example does not expect to have a career, marriage, or children, or a long life.

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by at least two of the following:
(1) Difficulty falling or staying asleep;
(2) Irritability or outbursts of anger;
(3) Difficulty concentrating;
(4) Hypervigilance;
(5) Exaggerated startle response;
(6) Physiologic reactivity upon exposure to events that symbolise or resemble an aspect of the traumatic event, for example a woman who was raped in an elevator breaks out in a sweat when entering any elevator.

E. Duration of the disturbance (symptoms in B, C and D) of at least one month.

Criteria C(1), (2), (3) and (7), D(1) and E are not included in DSM-III and one diagnostic indicator ("guilt about surviving when others have not, or about behaviour required for survival") which was present in the earlier Manual has been omitted from the 1987 Revision.

III. AETIOLOGY OF PTSD

What sort of people develop PTSD, thereby becoming potential claimants for civil compensation, or alternatively able to plead the condition as a defence to a criminal charge? Scrignar, a psychiatrist who has extensively researched the disorder, contends that the sine qua non of PTSD is the occurrence of a discrete traumatic event, subjectively interpreted as a threat to oneself or to "significant others" in one's life.[8] Actual physical injury to the victim need not occur, and there is no demonstrable correlation between the intensity of psychological symptoms and the physical harm sustained.[9] The threatening event must precipitate strong autonomic[10] arousal of the victim,

8. Scrignar supra n 4, 22.
9. Ibid, 37.
10. Refers to the automatic bodily functions, such as breathing and circulations.

characterised by a typical high-adrenalin "fight or flight" response; but whereas in most individuals this response attenuates once the stimulus is removed, in victims of PTSD the natural panic reaction mutates into a pathological anxiety. The victim is unable to dismiss from his mind recurrent intrusive thoughts about the near disaster he has experienced, and these morbid images trigger further anxiety, which in turn renders the depressed and helpless sufferer more prone to similar thoughts. If any physical injury was sustained in the initial trauma, this may complicate the picture since the pathological anxiety exacerbates and sustains pain sensations even after physical healing has occurred, so that patients become convinced that they are suffering from an incurable disorder which, in a cyclical manner, reinforces their anxiety and reminds them of the precipitating event.

Victims of PTSD may suffer constant anxiety at levels varying between three (moderate) to five (panic) on a five point scale,[11] with periodic attacks of symptoms including sweating, dizziness, chest pain, dyspnea (inability to breathe), feelings of impending loss of physical and mental integrity and dissociation or detachment from reality and from one's personal identity so that one's physical actions are no longer seen as belonging to oneself or being under one's own control. Probably at level four, and certainly at level five, anxiety ceases to be under the control of the victim, who can no longer voluntarily distract him or herself with more positive thoughts, but is caught in a self-perpetuating vortex of negativity and fear. That the anxiety is not a carefully nurtured consequence of the trauma, cultivated only for compensatory purposes, has been well established by Sprehe[12] and Valliant,[13] - 78 per cent of patients with significant psychological illness were "no better" ten years after the event for which compensation had been paid and 58 per cent were unable to work, because of their continuing disability. As Scrignar wryly observed, "the greenback poultice is more fantasy than fact."[14]

The symptoms of chronic PTSD must have endured for at least six months before a diagnosis can be made. It has been reported as a result of a variety of stimulus events. Historically its connections with war are well documented – today this source of PTSD is rivalled by motor vehicle and plane crashes.

11. See Levels of Anxiety Chart, Schedule 1.
12. D J Sprehe "Follow-up of Five Hundred and Ten (510) Consecutive Workers Compensation Cases With Significant Psychiatric Disability" 8th International Congress of Law and Psychiatry, Quebec 1982.
13. G E Valliant "Natural History of Male Psychological Health X: Work as a Predictor of Positive Mental Health" in Scrignar supra n 4, 50.
14. Scrignar supra n 4, 50.

Industrial accidents (for example, a fire at a petroleum refinery, or the capsizing of an oil rig) also take their toll upon some of the individuals involved, as do criminal assaults and hijackings. The apprehension of danger need not be precisely coincidental with the trigger event but must occur close to it, either immediately before or afterwards. Thus a nurse with renal failure was undergoing dialysis when she noticed a taste of formaldehyde in her mouth. This was reported and it was discovered that formalin had accidentally been mixed with the ordinary dialysate - information which later precipitated the nurse into PTSD when she realised the potentially fatal consequences for herself.[15]

Scrignar has argued that the danger to oneself need not, on an objective analysis, be very great so long as it is subjectively assessed at the time as being horrific and beyond the capacity of the sufferer to manage, thus causing hyper-arousal of the autonomic system. He reports two cases of enduring PTSD triggered by almost laughably small incidents. In one instance a computer operator bent down to pick up groceries at a supermarket and was hit on the head twice by 4lb bags of carrots sliding off the vegetable counter.[16] Severely shaken, she returned home and developed an intense anxiety attack, even to the point of suffering a dissociative reaction which disabled her for more than 12 months.

The second case is fascinating because it could throw new light upon May Donoghue, the well-known ingester of part of a rotting snail which lurked in a ginger beer bottle and precipitated the case of *Donoghue v Stevenson*.[17] Mrs Donoghue's legal triumph was a Pyrrhic victory - after the snail incident she was inadequately compensated and ended her life in a mental home. Had she lived fifty years later, perhaps she could have claimed compensation for PTSD, as did a 31 year old secretary in America who ordered a sandwich from her local lunchbar and happily consumed part of it before noticing that she had eaten her way through half of a large cockroach along with other sandwich fillings.[18] Greatly distressed, the secretary immediately vomited and subsequently developed pathological anxiety, combined with insomnia, phobias of bugs and nightmares - PTSD symptoms which drove her to distraction for more than a year.

15. Ibid, 19-20.
16. Ibid, 21.
17. [1932] AC 562.
18. Scrignar supra n 4, 21-22.

Some rescuers at the scene of disasters are also potential sufferers from PTSD even though the danger is not to themselves. It appears that if they identify closely with a disaster victim, or alternatively see a close resemblance between a family member (objectively known to be safe) and a victim, rescuers may undergo strong autonomic arousal with accompanying symptoms of nausea, tachycardia and dyspnea, and that this may modulate subsequently into self-perpetuating high-level anxiety, with or without dissociation or emotional disintegration.[19]

As indicated the prognosis for PTSD sufferers is not encouraging. The symptoms often intensify within four to six weeks of the precipitating event and persist into an acute phase in which the patient is totally preoccupied with reliving the horrifying event both during waking and sleeping hours.[20] There is loss of normal emotional responsiveness and interest in life as the victim compulsively replays a mental video tape of the stressful situation, although in some cases this is varied by "flashbacks" in which the whole scene is superimposed upon external reality.[21] These flashbacks, apparently similar to those suffered by LSD and psilocybin users,[22] are often accompanied by illusions or hallucinations and feelings of dissociation, and are precipitated by places, events or odours reminiscent of the original traumatic event.

Another quirk of PTSD is that occasionally sufferers will display a total inability to recall a specific aspect of the disaster, although such amnesia must be psychogenic since no organic damage has been sustained to explain memory loss. Following the acute stage of PTSD, the victim may slip into a chronic stage characterised by depression, generalised anxiety (sometimes increasing to acute levels with derealisation and other symptoms), motor tension and, often, drug or alcohol abuse.[23]

Occasionally and for reasons unknown, the onset of PTSD may be delayed a matter of months or even years after the event (sometimes known as "delayed shock"), in which case identification of the precipitating event needs to be cautiously undertaken so that a less obvious second trauma is not confused with a spectacular primary event.[24]

19. Ibid, 20-21.
20. Ibid, 44.
21. Ibid, 48.
22. Ibid, 19.
23. Ibid, 69.
24. Ibid, 51.

IV. LEGAL IMPLICATIONS OF DISSOCIATION

Given that PTSD can be established as a genuine psychosomatic phenomenon following a more or less predictable course, how does this impact upon the criminal law and mental state defences to crime? Traditionally the legally significant aspect of the disorder (whether officially diagnosed as PTSD after 1980 or not) has been the dissociative/hallucinatory phase founding a plea of automatism and raising the cognate question of whether the automatism be sane or otherwise.

Section 23 of the Criminal Code 1913 (WA) recognises that "a person is not criminally responsible for an act or omission which occurs independently of the exercise of his will, or for an event that occurs by accident", whilst section 27 also excuses a person for an act or omission "if, at the time of doing the act or making the omission he is in such a state of mental disease or natural mental infirmity as to deprive him of the capacity to understand what he is doing, or of the capacity to control his actions, or of the capacity to know that he ought not to do the act or make the omission."

Practical differences between pleading the two sections are the onus and standard of proof required. Voluntariness may be established under section 23 if the accused raises a reasonable doubt as to this matter. On the other hand, if the accused wishes to establish a section 27 defence of insanity, it will be necessary to do so on balance of probabilities. Another consequence of pleading one or the other of the two sections is evident at the dispositional phase - a section 23 "unwilled act" plea, if successful, results in complete acquittal, whereas a section 27 insanity plea means that if in fact the accused did the act complained of, the accused will be found "not guilty by reason of insanity" and may be detained at the Governor's pleasure in a mental institution. Not surprisingly, since the abolition of the death penalty in Australia, the insanity plea, substituting as it does an indeterminate non-parolable sentence for a (sometimes) parolable and determinate sentence for wilful murder, murder or manslaughter, has fallen into comparative disuse.

Into which of these two exculpatory categories does unconscious dissociative behaviour fall? Since the case of *Bratty v Attorney-General for Northern Ireland*[25] ("*Bratty*") the answer is that it may be classified as sane (section 23) or insane (section 27), depending upon the nature of the behaviour, whilst *R v Quick and Paddison*[26] ("*Quick and Paddison*") refined

25. [1963] AC 386.
26. (1973) 57 Crim App R 722.

this by suggesting that the decisive question was whether the causative factors of automatism are internal or external. In his discussion of unwilled acts in *Bratty*, Lord Denning also makes the point that neither amnesia nor irresistible impulse are automatic defences to criminal acts, but sane automatism (for example, caused by concussion) justifies an acquittal whilst insane automatism calls for a special verdict.[27] In that case a third option was also canvassed, that of involuntariness arising from intoxication which is self-induced, but this was seen to be relevant only to charges of specific intent, reducing these to the most proximate offences of general intent, but never excusing entirely.[28]

V. CATEGORISATION OF AUTOMATISM AS SANE/ INSANE

A. Theory

It is necessary to determine which sorts of automatism fall in the sane category, and also the rationale behind this classification, so that it may then be applied also to those alleging automatism whilst suffering from PTSD. Specifically, when is an involuntary act the product of insanity? According to section 27:

> [W]hen the person doing the act is in such a state of mental disease or natural mental infirmity as to be deprived of the capacity to understand what he is doing, or of the capacity to know that he ought not to do the act.

The crucial phrases here are "mental disease" and "natural mental infirmity", which are virtually coterminous with the criteria enunciated by their Lordships in *M'Naghton's Case*:

> [T]o establish a defence on the ground of insanity, it must be clearly proved that, at the time of committing the act, the party accused was labouring under such a *defect of reason from disease of the mind*, as not to know the nature and quality of the act he is doing; or if he did know it, that he did not know he was doing what was wrong[29] (emphasis added).

27. *Bratty* supra n 25, 409.
28. Ibid, 410. In Victoria the position with respect to self-induced intoxication was laid down in *O'Connor* (1980) 54 ALJR 349, in which it was held that the defence of self-induced involuntariness arising from intoxication was available to offences of general intent.
29. (1843) 10 Cl & F 200, 210.

Since 1843 the above formula appears to have preoccupied the judiciary despite advances in medical knowledge and psychiatry. Two matters are clear from case law. The first is that a disease of the mind does not necessarily imply any visible organic changes to the brain. As Justice Dixon stated in *R v Porter* ("*Porter*"):

> Disease, disorder or disturbance of the mind ... does not mean ... that there must be some physical deterioration of the cells of the brain, some actual change in the material physical constitution of the mind, as disease ordinarily means when you are dealing with other organs of the body where you can see and feel and appreciate structural changes in fibre, tissue and the like.[30]

Secondly, it is also manifest that the perception of "disease of the mind" is decided by the court, and not by medical science. As Fairall has pointed in out in his article "Irresistible Impulse, Automatism and Mental Disease,[31] any contrary approach would risk a repetition of the fiasco in a United States court where one Friday it was argued by the staff of a mental hospital that psychopathy was not a mental disease, and then, when the hearing resumed on Monday after a weekend change of terminology by the medical staff, the same witnesses testified that psychopathy was now accepted as a mental disease. This provoked the comment from the trial judge, Justice Burgher, that "no rule of law can possibly be sound or workable which is dependant upon the terms of another discipline whose members are in profound disagreement about what those terms mean".[32]

B. Judicial Categorisations

1. Physical Causes

Courts have had little difficulty in deciding that organic psychosis (such as paresis of the brain caused by invasion by the syphilis spirochete, gross dysfunctioning resulting from brain tumours or lead poisoning) and functional psychosis (whether affective, [for example, manic depression] or schizophrenic [hebephrenia, catatonia, paranoia and so on])[33] are mental diseases justifying a special verdict if the patient is involved in criminal charges. Much less easy to categorise has been automatism due to epilepsy,

30. (1933) 55 CLR 182, 189.
31. (1981) 5 Crim LJ, 136. See also *Hodges* (1985) 19 A Crim R 129.
32. (1961) 288 F 2d 853, 860-861 US App DC in Fairall supra 31, 150.
33. P Hill, R Murray and A Thorley *Essentials of Postgraduate Psychiatry* 2nd edn (New York: Grune and Stratton, 1986) 343.

arteriosclerosis, concussion, diabetes and psychological shock. Which, if any, of these are diseases of the mind capable of preventing their sufferers from knowing the nature or quality of their acts, or appreciating the moral wrongness of acts whose physical quality is comprehended? (The High Court has made clear in *R v Stapleton*[34] that it is moral and not legal transgression which is at issue here).

In *Bratty*, a relatively early case decided when psychological notions were still gaining currency, Lord Denning seems to divide sane automatism from the insane variety on the grounds of dangerousness and likelihood of recurrence, which may be empirically useful but is jurisprudentially unsatisfactory. There he stated that:

> It seems to me that any mental disorder which has manifested itself in violence and is prone to recur is a disease of the mind. At any rate it is the sort of disease for which a person should be detained in hospital rather than be given an unqualified acquittal.[35]

Here disposition for an indeterminate period is based on anticipated dangerousness, though courts may not usually take the same matter into account when passing even a determinate sentence upon a repeated sane offender. Denning's definition would mean that psychopathy (normally not seen as a mental state defence)[36] would compel an insanity verdict since it is characterised by repeated violent outbursts. Against his Lordship's view it may be salutary to state the opinion of Justice Dixon in *Porter*:

> A great number of people who come into a criminal court are abnormal. They would not be there if they were the normal type of average everyday people (sic). Many of them are very peculiar in their dispositions and peculiarly tempered ... Nevertheless they are mentally quite able to appreciate what they are doing and quite able to appreciate the threatened punishment by the law and the wrongness of their acts.[37]

Thus dangerousness and repetition are less satisfactory indicators of insanity or mental disease than an alternative criterion of internal/external provenance advanced in the more recent case of *Quick and Paddison*, by Lord Justice Lawton. His Lordship stressed the requirement, for a special verdict, that the malfunctioning be caused by disease, and observed that "a malfunctioning of the mind of transitory effect caused by the application to the body of some external factor such as violence, drugs including anaesthetics,

34. (1952) 86 CLR 358, 367.
35. Supra n 25, 412.
36. *R v Jeffrey* in I G Campbell *Mental Disorder and Criminal Law* (Sydney: Butterworths, 1988) 127.
37. Supra n 30, 187.

alcohol and the hypnotic influences cannot fairly be said to be due to disease.[38]

Of course the problem may have an apparently dual origin as considered in *R v Meddings*[39] where an epileptic fit induced by alcohol resulted in an apparently motiveless shooting. There the judge rejected the view that the effective cause was the external factor, since epileptic fits are usually triggered by external stimuli, whether chemicals, patterns of light, and so forth, and concluded in favour of the underlying cause that the accused was, as an epileptic, suffering from a disease of the mind which precluded an unqualified acquittal. This decision is in line with the earlier Queensland ruling in *R v Foy*[40] ("*Foy*") that epilepsy constitutes a mental disease manifesting itself (sometimes) in insane automatism. By contrast the automatism resulting from hypoglycaemia has been held in *Quick and Paddison*[41] to be sane, since the triggering event is the injection of insulin and the failure to counterbalance this with an adequate carbohydrate intake. However this is a more contentious decision since one could argue that diabetes and epilepsy are both organic problems and the only difference is that in one case the external event was the administration of a physiologically unnecessary stimulus (alcohol/light) whereas in the other case the external event was the administration of a physiologically necessary chemical either in an excessive dose or combined with an unreasonable abstinence from food.

Arteriosclerosis which causes automatism has been held in *R v Holmes* to support a special verdict since its causative factors are internal:

> [P]remature hardening of the arteries ... led to a reduced blood supply to the brain and in the stress of strong emotion there would be a severe restriction of that blood supply which at the time would be only temporary and which would leave [the accused] unable to distinguish right from wrong and unable to control his actions.... [T]hat evidence does support a finding of a disease affecting the mind.[42]

The same conclusion was reached in *R v Kemp*.[43] Consistent with the external/internal stimulus theory, automatism resulting from a blow to the head was held to be sane and to justify an acquittal in *Cooper v McKenna*.[44] In Canada, automatism based on delirium produced by infection was held in

38. Supra n 26, 734-735.
39. [1966] VR 306.
40. [1960] QdR 225.
41. Supra n 26.
42. [1960] WAR 122, 126.
43. (1957) 1 QB 399.
44. [1960] Qd R 406.

R v King[45] to found an acquittal due to the fact that the organism producing the infection originated externally to the body. The Tasmanian decision in *Bedelph v R*[46] also endorsed this theoretical approach. Thus until recently it appeared established that as far as physical stimuli were concerned, the sane/insane automatism dichotomy was predicated upon whether the causative factors were internal, resulting in a disease of the mind and insanity, or external, leading to acquittal, provided always that the external triggering factors have not been self-administered. In the event that the triggering factor *was* self-administered the situation becomes unclear. English common law[47] and Australian Code states hold that no consideration may be given to the question of the voluntariness of the defendant's acts,[48] whilst for Australian common law jurisdictions the High Court has admitted evidence of witting self intoxication as being relevant to determine whether an act was voluntary.[49]

However, one might query the basis of this internal/external categorisation of causes of actions. If germs producing delirium may excuse because of their provenance external to the human body, why not seek the primary cause of arteriosclerosis (causing restriction of blood to the brain) in the high cholesterol food also originating externally to the body? If one adopts this view, the justification for an acquittal in one instance, and a special verdict in the other, seems tenuous and the approach adopted in *R v Falconer*[50] ("*Falconer*"), discussed below, appears preferable.

2. Psychological Causes of Automatism

If externally applied physical shock (such as a blow to the head) which causes a dissociative state of automatism has frequently justified an acquittal, what is the situation in respect of an externally originating psychological shock similarly causing dissociation?

45. (1962) 35 DLR (2d) 386 in R D Mackay "Non-Organic Automatism - Some Recent Developments" [1980] Crim LR 350, 357.
46. (1980) 1 A Crim R 445.
47. *R v Lipman* (1970) 1 QB 152.
48. *R v Kusu* [1981] QdR 136; *R v Palmer* (1985) A Crim R 1.
49. *R v O'Connor* (1979) 146 CLR 64.
50. Supra n 1.

In Canada, the recent trend is adumbrated in *Parnerkar v R*[51] and *R v Sproule*[52] and clearly established in *R v Rabey* ("*Rabey*").[53] There a third year geological student claimed to have become emotionally dissociated when told by another student for whom he had conceived a strong feeling, that she regarded him as "just a friend". He hit her repeatedly with a geological rock sample and then strangled her, after which he appeared extremely upset. The trial judge accepted that a dissociated state *could* be occasioned by external stimulus of psychological trauma which would support a plea of automatism, a view endorsed by the appellate court. However the latter court went further, specifying that sane automatism would be caused by only the most extreme types of psychological shock, and that dissociation provoked by "the ordinary" stresses and disappointments of life which are the common lot of mankind" (for example, rejection by a girlfriend) would only result in automatism in individuals with a predisposed weakness of mind, whose consequently diseased state would restrict them to a plea of insane automatism. Instances of objectively horrific situations capable of producing sane automatism in normal persons were given, such as "being involved in a serious accident without sustaining injury, seeing a loved-one murdered or seriously assaulted, and being the victim of a murderous attack with an uplifted knife."[54]

VI. PROBLEMS WITH JUDICIAL CATEGORISATION OF PSYCHOLOGICALLY-INDUCED AUTOMATISM, AND A CHANGE OF EMPHASIS IN *FALCONER*

The *Rabey* decision represents an extremely restrictive legal view, possibly motivated by a desire not to open the floodgates for acquittal pleas. It is also based on rather curious reasoning. Surely a psychological blow, whatever its strength, must originate externally, and it is the recipient's reaction to this which may be abnormal or disproportionate? And if the relevant criterion for the sane/insane classification were its external provenance, then a psychological blow should, if supported by adequate psychiatric evidence and judged credible by the jury as finders of fact, automatically

51. (1973) 33 DLR (3d) 683 in J McR Herlihy "Non-Insane Automatism - Its Future Under the Queensland Criminal Code" (1981) 12 UQLJ 101.
52. (1972) 19 CRNS 384 in Mackay supra n 45, 358.
53. (1980) 54 CCC (2d) 1 in M Goode "On Subjectivity and Objectivity in Denial of Criminal Responsibility" (1987) vol 11 no 3 Crim LJ 131.
54. (1978) 79 DLR (3d) 414, 430 in Mackay supra n 45, 358.

found an acquittal for crimes committed during the resultant dissociative state. Absent policy reasons, the fact that the psychological blow is sustained by a person with an "egg-shell psyche" should not preclude a sane automatism verdict on the grounds that only a potentially diseased mind will succumb to an (objectively) minor shock, any more than the possession of an egg-shell skull would preclude a similar plea on the grounds that a physically normally-constituted person would not have sustained concussion.

If the mind upon which the shock is inflicted has not hitherto manifested symptoms of insanity, aberrant behaviour following an externally applied shock should, if the jury accepts that there is automatism, be ruled sane, for to do otherwise is to assume that all sane humans have similar mental-stress tolerance - which most natural disaster situations and war show to be untrue.

Indeed research into PTSD claims in civil cases has, as discussed previously, shown that dissociation can be provoked by any situation perceived, rightly or wrongly, as so threatening that it arouses strong autonomic activity. This is true whether the trigger event is one of the objectively horrific situations mentioned in *Rabey*,[55] and catalogued in DSM-III-R, or a subjectively "mind-blowing" situation such as being repeatedly hit on the head by falling bags of carrots or consuming a cockroach. There seems little justification for saying that persons dissociating as a result of a major trauma are suffering sane automatism whereas those with the same symptoms resulting from a trauma which the judge subjectively assesses to be of less importance to the sufferer, are insane. If seeing a "loved one murdered or seriously assaulted" is a major stressor, how does one categorise the viewing of a murder or assault upon a family member or acquaintance whom one actively dislikes? Does it found a verdict of sane or insane automatism? And what of the genuinely psychotic person who is as yet undiagnosed but who sustains a major shock (such as being involved in a serious accident) and in a dissociative episode assaults another person. If he confines his plea to PTSD he may, ironically, be found to be suffering from sane automatism on the Canadian reasoning. Having two possible verdicts for the one plea of PTSD appears unsatisfactory if the *only* justification for this is the subjective judicial categorisation of a trauma as being extremely shocking.

The foregoing highlights, for the area of sane automatism, a tension hitherto mainly considered to inhere in the problem of insane automatism. How should the fate of a morally innocent perpetrator of otherwise criminal

55. Ibid.

acts be balanced against the need to protect the public from the unusually fragile personality which may commit such acts? Should a jury be instructed that, on policy grounds, any psyche which dissociates under an objectively minor shock is likely to do so again and this should be taken as prima facie evidence of mental infirmity? And if this expedient is adopted, can any allowance be made for idiosyncratic response? For example, is a person who eats half a cockroach in her sandwich, and then suffers periods of derealisation because of a horror of insects, to be found insane because of the objectively trivial nature of her experience? The recent decision in *Falconer* would suggest that this might be the outcome. This High Court judgment is important for mental state defences insofar as it appears to alter the emphasis from the cause of automatism to the effects upon the accused's personality. Recognising the tension between the need for individual justice and public protection Chief Justice Mason and Justices Brennan and McHugh said in a joint judgment that "the problem of classification in a case of transient malfunction of the mind lies in the difficulty in choosing between the reciprocal factors - the trauma and the natural susceptibility of the mind to affection by psychological trauma - as the cause of the malfunction".[56]

The solution proposed by their Honours is to "postulate a standard of mental strength which, in the face of a given level of psychological trauma, is capable of protecting the mind from malfunction to the extent prescribed in the respective definitions of insanity."[57] The standard is to be an objective one corresponding to that frequently used to establish provocation. It must represent the standard of an ordinary person, so that minds less resilient than this will be adjudged infirm, those as resilient will be held to be sane. A person raising the defence of automatism will be presumed to be pleading unsoundness of mind or insanity unless he can show sane automatism by demonstrating three things: that the cause of the automatism was: "(1) transient, (2) caused by trauma, whether physical or psychological, which the mind or an ordinary person would be likely not to have withstood, and (3) not prone to recur".[58]

56. *Falconer* supra n 1, 30.
57. Ibid.
58. Ibid.

Whilst this approach finesses the problems raised by the external/internal cause dichotomy, it clearly does not take into account the idiosyncratically-horrific-but-objectively-minor trauma, for example, being hit on the head by falling bags of carrots, or ingesting a cockroach in a salad roll. It is suggested that the psychobiology of PTSD may conceivably assist in this area.

VII. ASPECTS OF THE PSYCHOBIOLOGY OF PTSD

Was the latent reasoning behind the *Rabey* decision a desire to restrict spurious pleas, since the idiopathic effects of shock would be easily counterfeited and used to justify criminal acts committed during specious dissociative episodes? If so, perhaps what is needed is more hard data regarding PTSD to facilitate formulation of the correct plea and disposition. Traditional legal thinking does not offer a means of unravelling this tangle - but possibly biochemistry can sever the Gordian knot and at the same time provide a credible explanation of the pathology of PTSD. In addition, the medical basis of PTSD may also be shown to ground a plea in mitigation for those genuine sufferers who are not subject to dissociative episodes.

Research by psychologists from Yale, Harvard and Massachusetts[59] has revealed a biochemical foundation for PTSD. There appears to be a physical explanation for much of the sufferer's behaviour, including even the compulsive replaying of the disaster scenario which is symptomatic of the disorder. Roger Pitman,[60] has addressed the question of how an external traumatic event can impact upon the biology of an organism to produce memory traces that are highly resistant to extinction. He points to research by McGaugh,[61] and, later by Gold[62] indicating that some hormones (such as adrenocorticotropic hormone [ACTH], vasopressin, epinephrine and norepinephrine) are stress-responsive neuromodulators in that they enhance consolidation of memory traces in the brain. When hormonally enhanced, these memories are very resistant to extinction, confirming Pavlov's early work with fear-connected memories and responses in animals.[63] Pitman also suggests that noradrenergic

59. B van der Kolk, M Greenberg, H Boyd and J Krystal "Inescapable Shock Neurotransmitters and Addiction to Trauma: Toward a Psychobiology of Post Traumatic Stress" (1985) vol 20 no 2 J Biological Psychiatry 314-325.
60. "Post Traumatic Stress Disorder, Hormones and Memory" (1989) 26 J Biol Psychiatry 221.
61. J L McGaugh, K C Liang and C Bennett "Adrenergic influences on memory storage: interaction of peripheral and central systems" in Pitman supra n 60, 221.
62. P W Gold "Stress Responsive Neuromodulators" in Pitman supra n 60, 221.
63. I P Pavlov "Conditional Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex" in van der Kolk supra n 59, 316.

mechanisms are involved in this "superconditioning" of memories, imprinting them on the mind in a way that is extremely intense and resistant to normal decay. He postulates the following steps in the pathogenesis of PTSD.

1. An extremely stressful traumatic event overstimulates endogenous stress-responsive hormones and neuro-modulators.
2. These substances precipitate an overconsolidation of the memory trace of the event.
3. This leads to the formation of a deeply engraved traumatic memory.
4. This manifests itself as intrusive recollections and the conditioned emotional response characteristic of PTSD.[64]

The PTSD sufferer is therefore not simply being a weak-minded malingerer when he claims to be unable to stop reliving the traumatic incident - it has been physically branded upon his mind. These findings accord with the work of van der Kolk, Greenberg, Boyd and Krystal who see in the hormonally-induced long-term potentation of neural pathways an explanation also of the extremely vivid nightmares and flashbacks that haunt the PTSD sufferer,[65] flashbacks which are associated with the dissociative (and criminogenic) phase.

VIII. PTSD AND ADDICTIVE ENDOGENOUS OPIOIDS

A curious feature of these intrusive memories, nightmares and flashbacks is that they may be addictive. Separate research by Maier,[66] Bodner,[67] Lewis[68] and Kelly[69] all suggest that in animals who have suffered from inescapable shock, brief re-exposure to a stressor will produce a form of temporary analgesia related to the release of serotonin, whilst another form of analgesic response to stress in humans is connected with the production of endogenous opioids, for example, beta-endorphin, and corticosteroids (ACTH).[70] In other

64. Pitman supra n 60, 222.
65. Van der Kolk supra n 59, 318.
66. S F Maier, S Davies and J W Gran "Opiate antagonists and long term analgesic reaction induced by inescapable shock in rats" in Van der Kolk, supra n 59, 318.
67. R J Bodner, D D Kelly and M Glusman "Stress induced analgesia: Time course of pain reflex alternation following cold water swims" in Van der Kolk, supra n 59, 318.
68. J L Lewis, J T Cannon and J C Liebeskind "Opioid and non-opioid mechanisms of stress analgesia" in Van der Kolk, supra n 59, 318.
69. D D Kelly "The role of endorphins in stress-induced analgesia" in Van der Kolk, supra n 59, 318.
70. H Anisman "Neurochemical changes elicited by stress: Behavioural Correlates" and J Willes, J Petren and J Cambier "Stress induced analgesia in humans: Endogenous opioids and ralaxone reversible depression of pain reflexes" in Van der Kolk, supra n 59, 319.

words, persons who develop PTSD may have a physiological drive to seek out or create stressful or dramatic situation in order to experience some relief from perpetually high anxiety, caused by the intrusive memories, in the release of natural opioids which occurs in such circumstances. Jaffe and Martin have pointed out that the symptoms of opiate withdrawal are almost identical with the symptoms of PTSD - anxiety, irritability, explosive outbursts, insomnia, hyper-alertness and emotional lability.[71] Lending credence to this theory is the work of Christie and Chesher, which showed that animals who are subjected to prolonged stress develop the same sort of changes in the opiate receptors of their brains as would be expected if they were taking opiates from an external source.[72]

Looked at from the physical aspect, PTSD changes from a mysterious and rather incredible one-off response to a shock, and becomes rather more comprehensible. In some persons excess hormones are triggered by a stressor event, whether large or small, so long as autonomic arousal is present to precipitate the release of neuromodulating hormones. These hormones indelibly fix the horrific (or perceivedly horrific) memories in the brain of the sufferer so that the memories, nightmares and flashbacks consistently recur and provoke high level, chronic anxiety. Cessation of the high stress situation leads to withdrawal symptoms of anxiety and hyperactivity, with outbursts of aggression. Thus the sufferer from PTSD is in the extraordinary situation of being "hooked" on his own natural narcotics, caught in a cycle of unwittingly seeking high stress/trauma situations to relieve the anxiety produced by the eidetic, hormone-enhanced memories, and then experiencing a further anxiety producing "low" after the latest event or outburst. This involuntary addiction also has implications for the individual with PTSD who does not dissociate under extreme stress, but who nevertheless may be able to argue that incomplete self-control should be a mitigating factor, especially since the incapacity was, unlike alcohol or drugs, not self administered or voluntarily acquired.

71. J H Jaffe and W Martin "Narcotic Analgesics and Antagonists" in Van der Kolk supra n 59, 320.

72. M J Christie and G B Chester "Physical dependence on physiologically released endogenous opiates" in Van der Kolk, supra n 59, 319.

IX. IMPACT OF PATHOLOGY OF PTSD UPON ITS LEGAL CATEGORISATIONS

With respect to PTSD as an automatism plea, a salient fact must surely be that the provenance of the original trauma was external. However, if the PTSD is of some duration, that is, the automatism episode is not an immediate response to the stressor event as in *Rabey*[73] - a problem arises in determining the proximal cause of the current criminal behaviour. Given the pathology of PTSD, is the effective cause of an outburst of aggression years after the original shock (as alleged in *Radford*[74]) internal or external? It is submitted that since there is usually an external trigger which raises the sufferer's stress rating to the maximum level and thus precipitates a dissociative episode, the precedents set by *Quick and Paddison*[75] and *Foy*[76] both merit close consideration. In the former case diabetes was identified as the indirect cause of the automatism, which was directly triggered by over-administration of insulin. Lord Justice Lawton there observed:

> [I]n this case, Quick's alleged mental condition, if it ever existed, was not caused by his diabetes, but by his use of the insulin prescribed by his doctor. Such malfunctioning of his mind as there was, was caused by an external factor and not by a bodily disorder in the nature of a disease which disturbed the working of his mind.[77]

On the other hand, in *Foy*,[78] the accused, who murdered his wife with a tomahawk whilst in a state of epileptic automatism, was found not guilty by reason of insanity. Whilst epilepsy often requires an external trigger to precipitate an attack, a more compelling argument for the radical distinction between it and diabetes would appear to be that the latter is a malfunction of the islets of Langerhans in the pancreas which may, upon the application of a stimulus (excess insulin) have repercussions in the brain, effectively separating the act from the will, whilst epilepsy is essentially a malfunctioning of electrical impulses in the brain itself, which produces the same result upon the application of a variety of triggering stimuli. The locus of the underlying cause being different in the two cases, diabetes is seen as not being related to insanity whilst epileptic automatism is categorised as having its roots in a disease of the mind.

73. Supra n 53.
74. Supra n 1.
75. Supra n 26.
76. Supra n 40.
77. Supra n 24, 735.
78. Supra n 38.

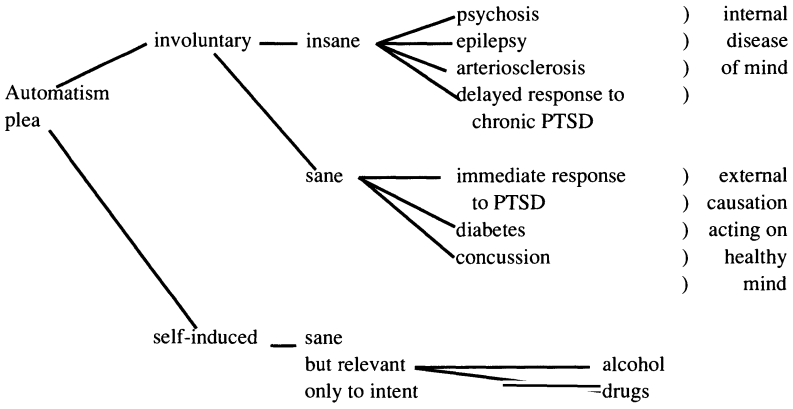
Given knowledge of its psychobiochemistry, where, on the sane/insane spectrum whose boundaries are marked by *Quick and Paddison*[79] and *Foy*,[80] does PTSD fit in? Is the bifurcated verdict, sane if dissociation occurs after a large shock, insane if dissociation follows an objectively small shock, sustainable as suggested by *Rabey*? It is respectfully submitted that a better view would be to assume, in accordance with section 26 of the Criminal Code, and absent any credible evidence of functional or organic disease to the contrary, that the accused's mind was sane when it sustained the initial trauma, whether physical or psychological. If this is so severe as to precipitate immediate or almost immediate dissociation, it is not unreasonable to see the situation as being analogous to *Quick and Paddison*[81] or *Cooper v McKenna*[82] and hold that the automatism, if proven to the satisfaction of the jury, was sane. This would have the merit of being susceptible to objective confirmation - persons trying to explain away criminal activity on the grounds of automatism precipitated by a recent shock should be required to submit to urine and blood sampling[83] to prove the presence of excessive stress hormones, before a sane automatism verdict is considered.

If, on the other hand, there is a substantial delay between the initial external trauma and the subsequent alleged trigger event, following which dissociation occurred, it is suggested that the relevant precedents are set by *Foy*[84] and *Bratty*.[85] It is at least arguable that, albeit mind subjected to the original trauma was sane, the subsequent development in that mind of chronic PTSD has now, with its alteration of the psychobiochemistry, produced a mental disease of a pathological nature more akin to epilepsy than to diabetes or concussion. Indeed Mason, Giller, Kosten, Ostroff and Podd have found that sufferers from chronic post traumatic stress disorder have a low urinary free-cortisol level (indicating lower activity of the adrenal cortex), similar to readings obtained from a control group of paranoid schizophrenics and significantly lower than patients with other major psychoses and depression.[86] Besides being a potentially useful evidentiary tool

79. Supra n 24.
80. Supra n 38.
81. Supra n 26.
82. Supra n 44.
83. Pitman supra n 60, 222.
84. Supra n 42.
85. Supra n 23.
86. Mason, Giller, Koslen, Ostroff and Podd "Urinary free-cortisol levels in post-traumatic stress disorder patients" in G Mendelson "The Concept of Post-Traumatic Stress Disorders: A Review" (1987) vol 10 Int J Law & Psychiatry 45.

to persuade judges that the disorder was present, low urinary free-cortisol levels and other biochemical indications thus suggest that the mental processes of the chronic PTSD sufferer who dissociates upon the stimulus of some subsequent separate event are demonstrably different from those of normal persons. It is the prerogative of the court to decide whether or not this difference is sufficient to ground an insanity plea, but it is to be hoped that objective criteria from medical research will assist in this categorisation. If this is accepted, the automatism defences might be graphically represented thus:

Automatism Defences



X. PTSD AS PLEADED IN CRIMINAL COURTS

In many states of America the subtleties of the *Bratty*[87] and *Rabey*[88] dichotomies have been ignored since the defence of sane automatism is not always recognised. Consequently PTSD has been put before many state courts simply as an insanity defence to enable the accused to escape capital punishment. As such, it was first pleaded in 1981 in *State v Heads*.[89] In that case Heads, a Vietnam veteran, successfully attributed an episode of automatism during which he shot his sister-in-law's husband, to his earlier traumatic combat experiences. A similarly successful plea was entered in 1982 by another ex-Vietnam soldier, Wood, who was accused of attempting to

87. Supra n 25.
88. Supra n 53.
89. 106, 126 (1d Caddo Parrish Nov 10, 1981).

murder a factory foreman after a work-related dispute.[90] In *Pard v US*[91] ("*Pard*"), a PTSD defence was accepted as a criminal defence to two counts of attempted manslaughter and one of attempted murder, but ironically the accused's affliction with the condition was rejected by a civil judge when *Pard* subsequently took the offensive and sought $9 500 000 damages from the government for failure to provide him with treatment for the disorder allegedly contracted as a result of war trauma. In another case, *State v Jensen*[92] ("*Jensen*"), the defendant contended that he suffered an episode of automatism occasioned by PTSD resulting from exposure to Vietnam horrors, and in this state of dissociation, committed two murders. Due to highly dubious evidence, the plea was rejected.

The American experience thus suggests that there are problems in sifting out specious pleas.

XI. ELIMINATING SPURIOUS PTSD PLEAS

The very minimum requirement for those pleading PTSD which caused a dissociative episode, resulting in the commission of a crime, some time after the original trauma, is that the details relating to that trauma be carefully validated. This sounds obvious, but a simple check of the alleged traumatic events in Vietnam in *Pard*[93] and *Jensen*[94] showed that in neither case did the alleged events take place.[95] When PTSD is pleaded, Wilson and Zigelbaum suggest that it should be established that the circumstances surrounding the criminal dissociative episode show strong links with the original traumatic event.[96]

90. *US v Wood* 80-7410 (3d Cook County May 5, 1982).
91. 589 F Supp 518 (1984) in L F Sparr and R M Atkinson "Post-Traumatic Stress Disorder as an Insanity Defence: Medicolegal Quicksand" (1986) vol 143 no 5 Am J Psychiatry 608, 611.
92. CR 75687 (Supra Ct of Arizona, Maricopa County 1985) in Sparr and Atkinson, supra n 91, 611.
93. Supra n 91.
94. Supra n 92.
95. Sparr and Atkinson supra n 91, 609.
96. Wilson and Zigelbaum "The Vietnam Veteran on Trial: the relation of post traumatic stress disorders to criminal behaviour" in Sparr and Atkinson supra n 91, 611.

Blank, investigating flashback phenomena in PTSD sufferers from the Vietnam war, has put forward the following criteria for validating genuine dissociative behaviour:

1. The flashback behaviour is unpremeditated and sudden;
2. The flashback behaviour is uncharacteristic of the individual;
3. There is a retrievable history of one or more intensely traumatic combat events that are re-enacted in the flashback episode;
4. There may be amnesia for all or part of the episode;
5. The flashback behaviour lacks current motivation;
6. The stimulus (trigger) for the flashback behaviour may be current physical or environmental features that are reminiscent of original experiences in Vietnam;
7. The patient is mostly unaware of the specific ways he has repeated and re-enacted war experiences;
8. The choice of victim may be fortuitous or accidental;
9. The patient has, or has had, other symptoms of post traumatic stress disorder.[97]

To these could also be added the more objective requirements of low urinary free-cortisol levels and high beta-endorphin and cortiso-steroid production upon exposure to stressful circumstances, which, it has been suggested, characterise sufferers from chronic PTSD. Once again psychiatric evidence of mental disease will be indispensable.

XII. RADFORD AND FALCONER REVISITED

To come full circle, how does this apply to Roger Radford, whose case began this paper and who was found guilty of murder when his PTSD plea was withdrawn from the jury by the trial judge? Nothing can now be said as to his psychobiology and, unfortunately, little is reported relating to his alleged Vietnam experiences. However, Radford's argument that the dissociation occurred unexpectedly and precipitated him back into a "combat mode" in which he killed Nancy Grugan, sits uneasily with the fact that he had previously provided himself with a long-barrelled rifle to take with him to help persuade his wife to leave Mrs Gurgan's house. Chief Justice King laid stress upon the fact that mental illness "must result from an underlying pathological infirmity of the mind, be it of short or long duration and be it

97. Blank "The Unconscious Flashback to the War in Vietnam Veterans: Clinical mystery, legal defence and community problem" in Sparr and Atkinson supra n 91, 611.

permanent or temporary ... as distinct from the reaction of a healthy mind to extraordinary external stimuli".[98] It is suggested that if Radford did indeed suffer dissociation provoked by extreme circumstances (his wife's relationship with Mrs Grugan), compounding the PTSD from a trauma suffered 15 years earlier, there is a good argument that his mind was not a "healthy mind" but one manifesting a pathological infirmity so that any dissociation should have been categorised as insane.

Nor is it easy to see Mary Falconer's behaviour in terms of PTSD. The alleged precipitating factors in this case were the discovery of her husband's sexual abuse of her daughters, her fear of his reaction to the ensuing criminal charges and her distress at learning of her husband's possible sexual abuse of a child who had lived with Mrs Falconer. The instances of sexual assault could not have formed the basis of a PTSD plea, since there was no threat to herself, and all of the abuses had occurred in the past and were no longer current. The fear of her husband's reaction to learning of the charges appears to have been ongoing - a factor raising her general anxiety level, but lacking the suddenness and terror that characterises PTSD. This is not to deny Mrs Falconer's alleged automatism, but rather to suggest that dissociation may be symptomatic of a variety of conditions, of which PTSD is only one. It may be that some of the physical indicators of PTSD are shared with other conditions, but this as yet is only a tentative hypothesis, awaiting further research.

XIII. CONCLUSION

PTSD is not an easy concept since, being primarily within the mind, its aetiology has idiosyncratic components and its relation to dissociative episodes makes most lawyers at least a little cynical. DSM-III-R has introduced clearer diagnostic criteria and the law has accepted, in theory at least, that automatism may be provoked as genuinely by traumatic stress and psychological blows as by physical blows. The problem remains of how to categorise stress-produced automatism - and here it is hoped that a cross disciplinary look at psychobiology may provide grounds for categorising immediate dissociation as sane and delayed dissociation as prima facie insane - although it is readily acknowledged that much work still remains to be done.

98. Supra n 1, 396.

SCHEDULE 1

Level of Anxiety Chart

(Scrignar, *Post Traumatic Stress Disorder*)

LEVEL	SYMPTOMS OF ANXIETY
Level 5 (panic)	Acute, intense, dysphoric symptoms include feeling of impending loss of physical and mental integrity, depersonalisation, derealisation, and incorrect encephalic statements, eg thoughts of impending doom, going crazy, going out of control, dying, or other thoughts of a cataclysmic nature. Panic attacks occur suddenly, without apparent warning, and are followed by severe anxiety symptoms. Autonomic hyperactivity is a prerequisite for the development of a panic attack.
Level 4 (severe)	Acute, intense symptoms include palpitations, dyspnea, hyperventilation, tightness or pain in the chest, trembling or shaking, sweating, dizziness, vomiting, fainting, tingling sensations in hands and feet, cold, clammy feeling, and hot flashes or flushing. Symptoms usually occur abruptly and unpredictably in the form of an anxiety attack.
Level 3 (moderate)	Chronic, moderate anxiety includes somatic symptoms usually affecting the gastrointestinal, cardiovascular, respiratory, genitourinary, or musculoskeletal systems. When persons are in this level for most of their waking state, they are suffering from a Generalized Anxiety Disorder.
Level 2 (mild)	Subclinical mild symptoms are characterised by statements from persons that they are uptight, edgy, high strung, tense, or nervous.
Level 1 (normal)	Nonsymptomatic.