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EDITORIAL

Chronic Post-Traumatic Stress Disorder: implications of recent epidemiological and neuropsychological studies[1]

It is made abundantly clear by a number of recent epidemiological studies that chronic Post-Traumatic Stress Disorder (PTSD) exacts a heavy toll in psychosocial disability. Such findings were anteceded by a report to the US Congress (Laufer, 1981) which disclosed a high toll in suicide, alcoholism, divorce and unemployment particularly among combat veterans of Vietnam. Later studies have confirmed this earlier report (Center for Disease Control, 1988). Of particular interest is the unusual epidemiological analysis of mortality rates among young men chosen by the draft for military service during the Vietnam war era compared with those who were not chosen (Hearst *et al.* 1986). The long-term mortality rate of the first group was higher than the latter; the causes of death reflected in this rate were suicide and motor vehicle accidents. Hearst *et al.* concluded that they had identified a 'large group of young men at risk for preventable death'.

In the past data on prevalence of PTSD have been lacking both in the general population and among military veterans. This gap in our knowledge is now partly filled by the publication of several papers, as well as a printed report to the US Congress. Both the Center for Disease Control (CDC) (1988) and the National Vietnam Veterans Readjustment (NVVR) Study (Kulka *et al.* 1988) focus on the prevalence and social disability of those with PTSD as a consequence of their wartime service in Vietnam. The lifetime prevalence reported in the CDC and NVVR study are remarkably close.

The NVVR study group estimates that 15% (±2·6%) of all male theatre veterans are current cases of PTSD. In numbers this is 470000 of the estimated 3·14 million Americans who served in the Vietnam theatre between 5 August 1964 and 7 May 1975. Among the estimated 7166 American females who served the prevalence estimate is 9% (±2·8%). These findings show that for males and females those exposed to higher levels of war zone stress have rates of PTSD three to five higher than their counterparts with moderate or low exposure. The report describes in detail the methodologies used to determine the diagnosis, the levels of combat exposure and the presumed differences between this study and the CDC report. The study group examined a sample of 1500 Vietnam theatre veterans and 730 other non-combatant veterans who served in the US Armed Forces between 4 August 1956 and 25 March 1973 from over 25000 personnel records. In depth face-to-face individual interviews of 3–5 hours duration were also carried out.

These prevalence rates for combat induced PTSD are much higher than those found by Helzer *et al.* (1987), who conducted an American nation-wide general population survey of psychiatric disorders. The latter study has been criticized as flawed (Keane & Park, 1988) in that the first criterion for diagnosis of PTSD, namely, that of exposure to a traumatic event, was insufficiently addressed, and very small subgroups of veterans were included.

I have personally examined over 300 cases of chronic PTSD in Vietnam veterans and previously had worked with acute cases in both World War II and in the civilian population. I am convinced that in civil life, as well as among military personnel, many cases go unrecognized by both the medical and psychiatric communities. Recognition of PTSD is important, as treatment may prevent the long-term psychosocial impairments mentioned earlier (alcoholism, divorce, suicide, violence, difficulty in holding employment) as well as exposing the sufferers to improper therapy. PTSD is often confused with other anxiety disorders, alcoholism and various depressive reactions. It may co-exist with any of the above or with other psychiatric disorders.

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What are the reasons for this diagnostic insensitivity (Kolb, 1987)? Certainly medical and psychiatric educators have paid scant attention to the condition except in its acute form when forced upon them during wartime or following a major catastrophe. But the problem also stems from the nature of the psychopathology. Most sufferers wish to avoid all enquiries concerning their traumatic experiences. They seldom spontaneously offer information; when questioned they often evade with generalities or denial. Both conscious and unconscious mental processes are at work: fear of shaming by examiners and others, suppression of reports which arouse strong emotional responses, desire in former soldiers to avoid any display of any behaviours which might impugn their masculinity.

Yet for the experienced the diagnosis is easy and the clinical reports of symptomatology are remarkably stereotypic. At one time psychiatrists enquired routinely as to whether their patients had undergone a violent experience/s in which they perceived their life was threatened. This question seems to be seldom put in the USA – even in the Veterans Administration hospitals. If the response is affirmative it should immediately alert the questioner to the possible presence of the constant symptoms of PTSD. Questions then asked about sleep, dreaming and nightmares, as well as dream content, usually and quickly will uncover the repetitive frightening repetition of the traumatic event/s. Questions about the content of daydreams generally uncover the intrusive rethinking of the terror ridden experience often associated with startling fantasies (flashbacks) of the experience/s. Later ruminations about the meanings of the experience/s reveal the stuff from which arise the continuing guilt, fear, anxiety and shame. A few will report the occurrence of brief psychotic-like dissociative episodes in which they seem to re-enact the event. If questioned, others will advise that on exposure to sharp sounds or sights reminiscent of the event, they startle, suffer palpitations and other indicators of physiological disturbance and may even attempt defensive fleeing or hiding. Further query usually reveals the development of various avoidance behaviours to escape situations arousing memories of the trauma. One may also detect the associated reactive dysthymias which impair daily functioning. These develop as the healthy self concept is shaken. The astute examiner will detect the variety of attempts at restitution of personality function in the use of whatever personality assets or defences the victim has available to him.

Even more interesting than the prevalence data on the chronic cases are the results of the psychophysiological and neurochemical investigations of those with chronic PTSD. These have recently been summarized in several reviews which suggest that certain of the symptoms of PTSD are the consequence of neuronal change (Kolb, 1987; Watson *et al.* 1988). The best substantiated data is that related to the evidence of unusual physiological arousal in combat veterans of PTSD exposed to stimuli reminiscent of wartime experiences in whom there occur immediate increases in cardiac rate, blood pressure, muscle tension and galvanic skin resistance.

Similar findings have been reported in World War II veterans (Dobbs & Wilson, 1960) and Vietnam combat veterans. Later replicative reports come from three separate sources (Kolb & Mutalipassi, 1982; Blanchard *et al.* 1983; Malloy *et al.* 1983). The clinical experiments differ only in the nature of the stimulus challenge. In one, only auditory stimuli were used, others used both visual and auditory stimuli. Years ago Kardiner emphasized that the PTSD sufferers were the victims of extraneous stimuli – that they startled in a way others did not – in fact, that they had a conditioned emotional response (Kardiner *et al.* 1947). Pitman *et al.* (1987) added a new dimension to the understanding of arousal in chronic PTSD as they observed physiological arousal by exposing their subjects only to short scripts of read materials purporting to arouse fearful, pleasurable or indifferent imaginations. Only the scripts related to combat experiences led to physiological arousal stimuli. Another study on combat aviators (Wenger, 1948) showed that a high proportion of them gave physiological evidence of abnormal sympathetic arousal in comparison to cadets in training for armed force duty.

Neuroendocrine and neurochemical abnormalities also have been reported in PTSD but are as yet unverified. I have hypothesized (Kolb, 1987) that the permanent neuronal changes in PTSD come about through the same neuronal processes concerned with learning, described in the electrophysiological studies of sensitization and habituation in the animal Aplysia (Kandel, 1982).

Kandel suggested that cyclic AMP is implicated in these changes. Whether the depressed levels of stimulated cyclic AMP in lymphocytes and platelets in patients with PTSD translates into central neuronal abnormality (Lerer *et al.* 1988) is open to question, but it gives some support to the Kandel suggestion. The hypothesis advances the notion that exposure to highly intense sensitizing stimulation (emotional stimulation) over long duration induces a permanent neuronal change in the temporal amygadaloid complex. Central inhibition of lower centres is impaired, leading to excessive discharge of hypothalamic ventromedial nuclei and the locus caeruleus. These, in turn, through their extensive cortical feedback generate the expressed constant symptoms of the condition.

Perhaps the most interesting aspect of the chronic form of PTSD is the often acute onset or rearousal of symptoms in the face of some immediate emotionally disturbing event. Deaths of close ones, exposure to another life threatening incident, exposure to dramatic events of civil violence, personal illness or illness of a close one, and even identifications with others who expose their traumas to their counsellor may lead, and have led, to major personality breakdowns. At high risk are those with chronic PTSD employed in services with frequent exposure to violence and its aftermath: police, firemen, other security officers and mental health counsellors.

To me this clinical phenomenology obviates the arguments that liken chronic PTSD to the addictive states (van der Kolk, 1987; Watson *et al.* 1988). The majority of sufferers exhibit avoidance behaviours, only those with mild to moderate symptomatology re-expose themselves to stimuli rearousing their fears and do so, as I have observed it, to test themselves. The predominant affect is anxiety/fear – not euphoria. At the moment there is an absence of research findings to support the addictive analogy.

From these recent investigations on the PTSDs many implications emerge for psychiatry. There exists from the clinical research the early potential of a diagnostic tool for chronic PTSD of high specificity and sensitivity.

Many of the arousal symptoms of PTSD are similar to those of other anxiety syndromes. They differ in that the stimulus is definable and idiosyncratic to the identified trauma situation. The PTSD syndrome may well be the nuclear disorder of this group in the APA's Anxiety Disorders category. I am of the opinion that many of those who suffer panic attacks and phobic states are unrecognized cases of PTSD. Two patients whom I had seen over four years for such symptoms eventually uncovered such events in their early life.

A similar diagnostic issue exists in the clinical assessment of those with acute schizophreniform or hysterical psychosis. Some men with PTSD dissociate episodically in the face of severe intercurrent emotional stress. We have seen some dissociate briefly following startle, induced during psychophysiological testing. 'Flashbacks' as well as panic attacks have been experimentally induced in PTSD sufferers by intravenous injection of sodium lactate (Rainey *et al.* 1987).

There are significant medical implications associated with this condition. Combat veterans with PTSD are at high risk for alcoholism and its complications. Many of our severely affected Vietnam combat veterans with PTSD had basal blood pressure levels above those of the controls. Of these men, I have known nine diagnosed on the medical services as hypertensive or having coronary artery disease. Others become chronic alcoholics and suffer the complications thereof.

My clinical experience over a period of time discloses the fact that the basic symptoms of PTSD are beyond modification by any of the social or psychotherapeutic approaches (Kolb, 1986*a*). Such efforts do assist the reactive affective and personality organized expressions of pathology. For those with the physiologically induced somatic symptoms there is a rationale in prescribing those drugs which attenuate discharge of the locus caeruleus (Kolb, 1986*b*).

Finally, as one reflects upon the aetiology of PTSD there arises the question of the nature of predisposition. Clearly, some exposed to intense and long enduring emotional stress in the face of mortal threat suffer little. Genetic predisposition has been suggested. But, there are hints that early life exposure to physical and emotional abuse by others hypersensitizes the neural system and establishes the neurophysiological groundwork for more permanent change in the face of massive terrifying exposures in adulthood.

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