The Acute Stress Response Following Motor Vehicle Accidents and Its Relation to PTSD

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This study aimed to identify acute biological stress responses in trauma victims and to determine their contribution to the development of psychiatric illness, especially posttraumatic stress disorder (PTSD). Abnormalities of the hypothalamic-pituitary-adrenal (HPA) axis have been noted in PTSD, with evidence of heightened sensitivity in the negative feedback loop. However, it is not known if some abnormality of stress response occurs at the time of the traumatic event or if the disruption of the HPA axis develops during the course of the illness. An understanding of the acute stress response in accident victims who go on to develop PTSD may provide important information about biological vulnerability to psychiatric illness and may provide a marker of "at risk" individuals.[1]

The HPA axis is of particular interest because cortisol is one of the primary modulators of the stress response and both cortisol and ACTH modify the consolidation of memory. In PTSD, the laying down of memories of the trauma and their recall is a central component of the disorder. Therefore, abnormalities of this axis may provide critical information about the aetiological process in this disorder. The sensitisation of the HPA axis and suppression of the dexamethasone suppression test in PTSD appears to be one of the most specific neurobiological abnormalities in PTSD. The findings of this study has general theoretical importance about the etiology of PTSD from the perspective of whether this disorder is a continuum of a normative stress response or whether it is an atypical or abnormal stress response.

A question that has not been answered is whether these HPA abnormalities arise from an abnormal HPA response at the time of the trauma or whether they develop later in the course of the illness. An understanding of HPA responses at the time of trauma in individuals who later develop PTSD may give insight into biological vulnerabilities and perhaps allow identification of at risk individuals. The body of literature concerning HPA abnormalities in PTSD has concentrated on war veterans many years after the initial trauma, raising the strong possibility that intervening factors contribute to the HPA abnormalities. One small study has assessed acute

cortisol levels in rape victims.[2] Acute cortisol responses did not predict the development of PTSD, but those subjects with a history of prior trauma showed attenuated cortisol responses and were at greater risk of developing PTSD. This raises the possibility that prior trauma may "sensitize" an individual's biological response to later trauma which leaves them at greater risk of PTSD. This study was only carried out on 26 subjects, and a larger number is needed to make more definitive statements. This is a central rational for conducting the proposed study.

METHOD

Subjects. The study group consisted of 40 individuals (30 males, 10 females) ranging in age from 15 to 77 years. They were recruited from a larger study of 200 motor vehicle accident victims who had been admitted for at least one night to a major teaching hospital. By law, all motor vehicle accident victims must have a blood sample taken for estimation of their alcohol level. Cortisol levels and lymphocyte glucocorticoid receptors were then determined by the hospitals pathology service.

Subjects were interviewed on the day following admission (Day 2), 10 days, and 6 months after the accident. During the Day 2 screening, after receiving appropriate consent, subjects were given the IES,[3] Brief SASRQ,[4] 36-item DSQ,[5] and a concentration questionnaire. The IES, SASRQ, and concentration measure were repeated on Day 10, along with the DISSI (Diagnostic Interview Schedule Screening Interview)[6] to account for preexisting psychiatric disorder. These measures were also repeated at a 6-month follow-up, in conjunction with the CAPS,[7] STAI–Anxiety,[8] Beck Depression Inventory,[9] and SIP.[10]

RESULTS

The 6-month follow-up of these subjects determined that seven had PTSD, seven from MDD and 12 had no disorder. The remainder of the subjects had a range of diagnoses and were excluded from analysis for this very reason. The mean of the time the accident occurred was 1330 hours, whilst the mean time a subjects blood sample was taken was 1400 hours. The serum cortisol results were examined for differences between these diagnostic grouping. Group means for cortisol results are presented in FIGURE 1. A one-way ANOVA showed that there was a difference in cortisol levels after the accident ($F = 3.65$, $df = 2,25$, $p < 0.05$). Post-hoc testing confirmed that the mean cortisol levels of subjects who had PTSD 6 months after the accident were significantly lower than those with MDD. This result were not significant when the effects of time the accident occurred and time the blood samples were taken were partialled out.

This result contrasts to the measures of psychological distress on Day 2, which had very little ability to predict the diagnostic outcome at 6 months (TABLE 1). There no differences on Day 2 between any of the diagnostic groups on either the IES or theSASRQ. On Day 10 the only significant difference on either of the measures of psychological distress was on the Flashback subscale of the SASRQ ($F = 9.85$, $df = 2,25$, $p < 0.001$). Post-hoc testing revealed that the PTSD group scored higher on this subscale than both the No diagnosis and MDD groups.

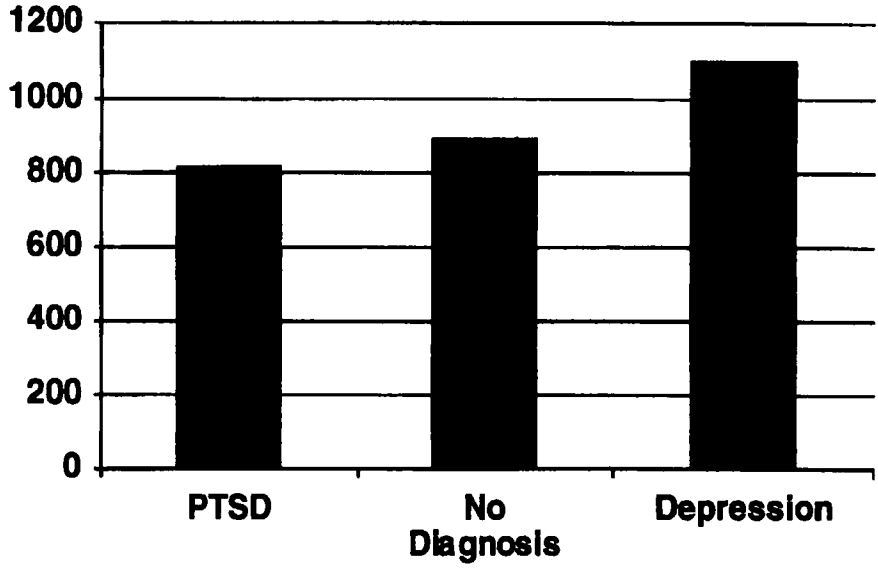

FIGURE 1. Cortisol means by diagnostic grouping.

TABLE 1. Mean and Standard Deviation of IES and SASRQ Subscale Scores on Day 2 across the Diagnostic Groups

	No Diagnosis		PTSD		MDD		*p*
	Mean	SD	Mean	SD	Mean	SD	
IES							
Avoidance	9.75	7.30	10.14	13.42	16.50	6.34	NS
Intrusion	12.67	5.05	12.66	5.05	17.33	9.58	NS
Total	22.41	8.14	20.71	14.87	33.83	13.21	NS
SASRQ							
Anxiety	3.92	1.83	4.29	3.25	5.33	2.58	NS
Dissociation	4.00	2.92	2.86	1.86	3.83	3.25	NS
Intrusion	1.25	0.97	1.43	1.13	1.67	1.63	NS
Avoidance	0.58	0.67	0.86	0.69	1.00	0.00	NS
Hyperarousal	2.58	1.68	2.57	2.15	3.17	1.94	NS
Flashback	0.08	0.29	0.57	0.98	1.00	1.10	NS

DISCUSSION

This study examined the acute psychological stress response and its relationship to the cortisol response amongst motor vehicle accident victims. Given the severity

of the trauma in these subjects, all of whom were admitted to hospital for a minimum of 2 days, a significant rise in serum cortisols occurred. In this pilot study, the rise in serum cortisol was greatest in those with major depressive disorder, and lowest in the PTSD group. The no disorder group were in the mid-range. This is contrary to the idea that in PTSD there is an exaggerated acute stress response. These biological data are of particular note because of the absence of any significant differences between three subject groups on the psychological measures at 24 hours.

These data require replication. However, it suggests that the abnormalities of decreased hippocampal volume associated with PTSD may not be explained by an excessive cortisol response in these subjects at the time of the traumatic stressor. Secondly, it raises questions as to what has modified the cortisol response in these subjects. Resnick *et al.'s*[2] data suggests that prior traumatisation might be one issue modifying this vulnerability.

In contrast, the psychological stress response on Day 1 does not differentiate the PTSD subjects from those with major depression or no disorder. In particular, there were no differences in the dissociative symptoms recorded in the first 24 hours. This is in contrast to the differences that emerged by Day 10. These data suggest that PTSD may represent an abnormality of transition occurring in the immediate post traumatic period, rather than there being a significant difference in the psychological response. The neurohormonal response provoked by the traumatic stressor may be one important factor modulating this response. It also raises the question as to the relationship between these data and the enhanced negative feedback which has been identified by the work of Yehuda.

Further research into the relationship between the acute neurohormonal response to the stressor in PTSD is of particular interest in terms of prevention. The current psychological interventions which are offered do not appear to be particularly potent modifiers of the long-term course of the traumatic stress response. Better characterization of the acute stress response and investigation of its relationship to PTSD may provide other avenues for intervention.

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