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POST TRAUMATIC AMNESIA AND ALCOHOL DEPENDENCE*

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SUMMARY

Post-traumatic amnesia was found to be prolonged in those who were alcohol dependents prior to the head injury. A comparison with controls who were matched for age and severity of head injury indicated that the PTA in the alcoholics was significantly longer. The pathophysiologic changes in the alcoholic brain that could explain such variations are discussed in the light of recent findings.

Introduction

Post traumatic amnesia is considered as the best predictor of the physical and mental restitution after head injury. Its predictive value with respect to several aspects of recovery has been commented by many authors. Lishman (1968) in his study of penetrating head injuries found that PTA was correlated to the amount of brain tissue lost and that a longer PTA was associated with higher incidence of psychiatric disability. Mayer-Gross et al (1969) used it as a guide to vocational restitution. Brooks (1976) pointed out that various aspects of memory recovery could be correlated to the length of PTA. Mandleberg (1976) showed that PTA indicated the rate of intellectual recovery. The better predictive ability of PTA in comparision to the duration of coma is attributed to its accounting for individual variations in neurological recovery.

Alcohol abuse is the commonest complicating factor in head injuries. Field (1976) observes that chronic alcoholics constitute as many as one fourth to one half of the injuries rated as being as severe. Twenty nine percent of males in Field's study (1976), 62% of males in Galbraith et al's study (1977), half of fatal motor accidents in U.S.A. (National Safety Council, 1978 quoted by Cooper, 1982) and 47% of head injury patients in Finland (Honkanen and Visuri, 1976 quoted by Cooper, 1982) had consumed alcohol at the time of head injury. In India, Natarajan et al (1987) report alcohol abuse in one fifth of their head injured patients and Chandrasekaran (1985) attributes more than 15% of the road accidents to drunkenness.

Recent findings indicate that even those alcoholics who do not show overt neurological complications, neuropathological changes are demonstrable (Ron, 1983). Considering the concept of cumulative brain damage in head injury, such subclinical changes may have an adverse prognostic significance. Becker et al (1982) point out that pre-existing brain damage need not be traumatic and can be due to other causes also. The present study aims to understand if the pretraumatic neurological changes in the alcoholics contribute to an impaired recovery as denoted by the length of PTA.

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Methods

The study was conducted in the Trauma Ward, Dept. of Neurosurgery, Government Rajaji Hospital, Madurai. Patients admitted between September 1984 and March 1985 were included in the present study. All the patients were followed-up prospectively by the psychiatrist along with neurosurgery team from the time of admission. They were seen daily till the day of discharge and subsequently at regular intervals as outpatients. History of alcohol addiction was gathered during the initial history-taking and was later verified during home-visits by the trained social workers.

Duration of coma was assessed using Glasgow Coma Scale. The injury was classified as mild, moderate or severe if the time taken to reach GCS score of eight was below 1 hour; 1 to 6 hours and beyond 6 hours respectively. Though blood alcohol levels were not assessed, it is known that levels upto 200 mgs do not alter the coma score. PTA was measured prospectively. It was defined as 'the lapse of time taken between the injury and the return of continuous memory' (Jennett, 1977). The assessment was done till there was concurrence between two successive measures. Care was taken to rule out fallacies such as memory islands, while measuring PTA. Patients who satisfied the following criteria were chosen.

1) They should satisfy the DSM III criteria for alcohol dependence.
2) Glasgow Coma Scale should have been applicable during recovery.
3) Patients with psychotic disturbances, frontal lobe damage, amnestic syndrome, and delirium tremens were excluded.
4) Only patients with definite history of cerebral concussion were included.

Age and duration of coma are known modulants of PTA (Carlsson et al 1968). Hence in the choice of the patients age-matched populations were chosen. There were eight alcoholics each with the mild, moderate and severe injuries and the patients were included by age-matching. Non-alcoholic controls were chosen similarly in each category of severity. The study involved forty eight patients. Two way analysis of variance was done using severity of coma along the columns and alcohol abuse along the rows.

Results

The eight patients in each cell were comparable to those in other cells with respect to their age. The duration and quantity of intake of alcohol were not considered because of poor reliability. Yet the dependent status of the alcohol group assures that neurological consequences of alcohol abuse assumed a comparable status. The results of two way analysis of PTA are summed up in Table 1.

Table 1
Two-Way ANOVA of PTA duration

	Mild	Moderate	Severe	Row Means
Alcohol Dependents	3.625	11	20.5	17.625
Non-Alcoholic Controls	2.5	3.625	8.125	7.125
Column Means	3.0625	7.3125	14.375	

	df	SS	MS	F
Rows	1	588	588	10.763*
Columns	2	1044.875	522.4375	9.563*
RXC	2	259.625	129.8125	2.3762
Within Cells	42	2294.5	54.631	—

– The figures in the cells represent the mean length in days of PTA.
– Number of patients each cell(n) is 8. Total n = 48
– * $P < .01$.

The analysis indicates that PTA shows significant variance with respect to both factors i.e., duration of coma and use of alcohol. No significant effects were attributable to the effects of interaction, between alcohol dependency and severity of the coma.

Discussion

The unitary concept of PTA is questionable, at least in severe head injuries. (Sabhesan and Natarajan, 1987). The various factors that could prolong the PTA have been excluded in the present sample selection. Though the retrospective selection of the patients may not be very welcome, given the nature of the present naturalistic study, such a method might be justified (Millon and Diesenhaus, 1972).

Recent studies into the nature of neurological and neuropsychological deficits in the alcoholics have established sub-clinical neuopathological changes in many of them. Ron (1983) in a psychometric and radiological study of alcoholics reports that the size of ventricular system is positively correlated with the discrepancy between immediate and delayed recall of verbal material. The same author contends that even in abstinent alcoholics the "lack of correlation between memory impairment and the duration of abstinence suggests that these deficits may be permanent or, if reversible that the process of recovery is extremely protracted". Bergman et al (1980) in a comparison of alcoholics and non-alcoholics make out that ventricular enlargement is correlated significantly with the poor performance on tests of learning ability. These studies indicate that the memory system in the alcoholics is already compromised even if they do not show any clinical evidence of memory problem.

The preinjury status of the brain and the level of function set the limit of recovery of mental and somatic neurological function (Miller and Becker, 1982). The cumulative deficits of memory due to alcoholic damage and due to head injury either summate or potentiate each other. The effects of pre-existing damages due to alcoholism delay the neuropsychological recovery from head trauma. The longer PTA is reflective of such changes.

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