Contribution of traumatic head injury to neuropsychological deficits in alcoholics

MATTI HILLBOM, LENA HOLM

From the Department of Clinical Alcohol and Drug Research, Karolinska Hospital, Stockholm, Sweden and the Department of Neurology, University of Helsinki, Finland

SUMMARY The contribution of head injuries to neuropsychological deficits was studied in 157 recently detoxified alcoholics and 400 control subjects consisting of age-stratified randomly selected men and women from the same geographical area as the alcoholics. Head injuries had occurred in 41% and 22% of the male and female alcoholics, but only in 15% and 6% of the male and female control subjects. One third of the injured subjects in both groups had been admitted to hospital for treatment of the acute injury. The neuropsychological test results of alcoholics were significantly inferior to those of control subjects. Unexpectedly, alcoholics with head injuries not identified at hospital were significantly inferior in several Halstead-Reitan subtests when compared with uninjured alcoholics with a similar duration of alcoholism and abstinence. By contrast, control subjects who had sustained a head injury not identified at hospital did not show signs of intellectual impairment when compared with uninjured controls. We conclude that traumatic brain injuries that may cause significant intellectual impairment may easily remain unrecognised in alcoholics.

Patients attending accident or emergency units because of acute head trauma are often intoxicated. In fact, several studies have shown that about half of such patients have ethanol in the blood on admission and most of the patients reveal a history of alcohol dependence.¹⁻⁴ Significant brain injuries may, however, easily remain unidentified both by the emergency room physician⁵ and the intoxicated subject himself, and it is unclear to what extent traumatic brain injuries contribute to the intellectual impairment of the alcoholic. We have studied the neuropsychological deficits of alcoholics with and without head trauma in previous history and the results indicate that even previously unrecognised traumatic brain injuries may significantly contribute to the neuropsychological deficits commonly seen among alcoholics.

Patients and methods

Our patient material comprised 98 alcoholics without any head trauma in previous history and 59 others who had had

head trauma(s). Of the latter group 21 had been admitted to hospital because of the trauma, but 38 others who also reported unconsciousness and even other symptoms of brain concussion were not admitted to hospital. Of these 38 subjects 11 had more than one concussion in previous history.

All the subjects were similarly interviewed by a physician to get reliable anamnestic data. The methods have been described elsewhere.⁶ Recent CT scans of the brain were available from every subject and previous records from other hospitals were examined in order to reveal data and diseases not known or reported by the patients themselves. Details of the severity of the head injuries could not be obtained from every subject, since previous records of other hospitals were not always available. Patients with liver cirrhosis, drug abuse, severe neuropsychiatric complications such as the Wernicke-Korsakoff syndrome and brain infarction and those primarily in need of intense medical care were excluded. However, liver biopsies were not routinely taken.

The alcoholics belonged to our inpatient programme and met the World Health Organisation criteria for alcohol dependence syndrome.⁷ The control subjects were an age-stratified random sample of 200 men and 200 women taken from the general population of the same geographical area in Stockholm.⁸ Every fifth control subject was either a current or previous heavy drinker.

Both the groups were identically examined by neuropsychological methods. All the alcoholics (27 women and 130 men) were studied during drug-free periods 10-30 days after admission for detoxication.

Address for reprint requests: Matti Hillbom, MD, Department of Neurology, University of Helsinki, SF-00290 Helsinki 29, Finland.

Received 19 November 1985 and in revised form 13 February 1986.
Accepted 15 February 1986

Fig 1 Mean anterior horn index and 3rd ventricle (maximum transverse diameter) values $\pm$ SEM for alcoholics with identified head injuries (1), alcoholics with unidentified head injuries (2), alcoholics without head injuries (3) and control subjects with identified head injuries (4). A score of 50 represents the mean value of the general population and a score of 10 represents one standard deviation. * $p < 0.05$, ** $p < 0.01$ compared with control subjects (4) with identified head injuries ($n = 14$).

Our neuropsychological test battery included tests for general intelligence (Synonyms, Reasoning, Block Design), learning and memory (Memory-for-Designs, Claeson-Dahl verbal learning and 30 min retention) and the Halstead-Reitan battery. Based on the results of the general population and controlling simultaneously for age and education but not for gender the raw scores were transformed to T-scores,⁹ that is, the mean of each parameter was set to 50 and the standard deviation to 10. Three age intervals and two levels of education were taken into account.

Student's *t* test and Chi square test were used for statistical comparisons.

Results

Clinical and neuroradiological findings. We found that

38% of the alcoholics and 10% of the control subjects had head injuries in previous history ($p < 0.001$). In both groups the injuries had led to hospital care in about one third of the cases. Forty-one and 22% of the male and female alcoholics, respectively, had head injuries in previous history. Corresponding figures for the control subjects were 15 and 6%.

Alcoholics showed profound cerebral cortical and central atrophy in CT scans taken a couple of weeks after they stopped drinking. Only insignificant differences were observed when those with head injuries were compared with other alcoholics but, by contrast, control subjects with head injuries identified at hospital (14 subjects including 5 heavy drinkers) showed significantly less cerebral atrophy (fig 1). It is interesting to notice that 36 and 18% of the head injured and non head injured control subjects were heavy drinkers, respectively. Heavy drinking was associated with increased susceptibility to head injuries even among the control subjects.

Alcoholics with and without head injuries showed very similar duration of subjective loss of control, but the uninjured alcoholics were somewhat older than those who had head injuries (table). There was no difference in maximum alcohol intake per drinking occasion as measured in grams of absolute ethanol ingested per day.

Cognitive performance in non head injured cases. Exclusion of subjects with head trauma showed that alcoholics had many signs of intellectual impairment that were not caused by traumatic brain injuries. Compared with the control subjects the uninjured alcoholics were significantly inferior ($p < 0.001$) in Trails, Category test, Memory-for-Designs, Block Design and Reasoning.

Cognitive performance in head injured cases. The alcoholics with head injuries were more impaired. They were inferior to the control subjects in all the tests used except the Claeson-Dahl 30 min retention test which is sensitive for the Korsakoff syndrome.

When alcoholics and control subjects admitted to hospital because of head trauma(s) were compared no statistically significant differences were observed in neuropsychological test results, but the groups were rather small (21 vs 14 subjects).

In some Halstead-Reitan subtests such as Tactual Performance localisation and Finger Tapping, the alcoholics with head injuries ($n = 59$) were signifi-

Table Mean age of the alcoholics and duration of subjective loss of control. Values are mean $\pm$ SEM

Patient groups	Number of subjects	Mean age (years)	Duration of subjective loss of control (years)
Alcoholics without head injury	98	47.1 $\pm$ 1.2	11.7 $\pm$ 0.9
Alcoholics with identified head injury	21	42.8 $\pm$ 2.2	12.8 $\pm$ 1.4
Alcoholics with unidentified head injury	38	42.3 $\pm$ 1.5	12.8 $\pm$ 1.6

Fig 2 Neuropsychological test results of the alcoholics compared to the general population. The hatched columns represent the mean values $\pm$ SEM of alcoholics with head injuries. The unhatched columns represent respective values of alcoholics without head injuries. *** $p < 0.01$ compared to alcoholics without head injuries.

cantly inferior to those without head injuries ($n = 98$), but no marked differences were found in tests of general intelligence, learning and memory (fig 2). The Halstead-Reitan impairment index which is computed from the scores derived from the six tests of the battery also showed significant impairment in the head injured alcoholics.

Alcoholics who reported head trauma(s) for which they were not admitted to hospital ($n = 38$) also showed poor performance in many neuropsychological tests. Compared to the uninjured alcoholics ($n = 98$) they were inferior in Finger Tapping ($p < 0.001$), Tactual Performance localisation ($p < 0.01$), Trails ($p < 0.05$) and the Halstead-Reitan Impairment Index ($p < 0.01$). A comparison of the test results of alcoholics with identified ($n = 21$) and unidentified ($n = 38$) head injuries showed only

minor inconsistent differences (fig 3) suggesting that many of those alcoholics who had not been admitted to hospital because of the acute head trauma had, however, sustained a significant traumatic brain injury.

Attempts were not made to localise the traumatic brain injuries since we lacked reliable anamnestic data and CT scans of the brain taken acutely after the head trauma. Hence correlations between functional deficits and cerebral morphology were not drawn.

Discussion

Most studies demonstrating neuropsychological deficits in alcoholics have tried to screen out subjects who have traumatic brain injuries. Patients admitted to hospital because of head trauma or treated there

Fig 3 Neuropsychological test results of alcoholics with identified and unidentified head injuries compared to the general population. The hatched columns represent the mean values $\pm$ SEM of alcoholics with unidentified head injuries. The unhatched columns represent respective values of alcoholics with identified head injuries. * $p < 0.05$ compared to alcoholics with identified head injuries.

for a brain injury have been excluded. However, as already pointed out by Wilkinson,¹⁰ such criteria may not have resulted in successful exclusion of subjects with significant traumatic brain injuries. Therefore we used more rigorous criteria, and included alcoholics who, following a head trauma, were not admitted to hospital, but nevertheless had traumatic head injuries with signs and symptoms of brain concussion. However, even our study may have underestimated the contribution of traumatic brain injuries to neuropsychological deficits in alcoholics, since such subjects who did not remember their head trauma events or had not recognised them were included in the uninjured group.

We found that alcoholics reporting traumatic head injuries not identified at hospital performed worse in neuropsychological tests and showed more profound radiological brain atrophy than those who denied ever having sustained clear head trauma, despite an equally long duration of alcoholism. This suggests

that single or multiple less severe head traumas had caused significant brain injuries to these alcoholics. Similar observations were not made in control subjects who reported mild head injuries not identified at hospital, but several control subjects with hospital treatment for head injuries showed clear signs of post-traumatic intellectual impairment and brain atrophy.

Our observations suggest that a considerable amount of traumatic brain injuries of alcoholics remains unrecognised. However, we found that one third of both head injured control subjects and alcoholics were admitted to hospital because of the injury which indicates that alcoholics received hospital treatment for head injuries as often as control subjects. Thus the injuries of alcoholics were not overlooked by physicians. Yet it is unclear whether all the alcoholics who had sustained a significant brain injury had been admitted to hospital.

Alternatively the head injured alcoholics may have been drinking in a pattern that is more likely to cause

brain damage. However, this was not observed, since the drinking habits of the three groups of alcoholics were rather similar.

Such Halstead-Reitan subtests as Tactual Performance localisation and Finger Tapping seemed to differentiate the trauma patients from other alcoholics in our study, but in this respect the injured alcoholics were not consistent. Since all our alcoholics were studied within a month after ceasing to drink, we do not know whether a subacute withdrawal syndrome masked some other differences.

There is experimental evidence to suggest that ethanol potentiates central nervous system trauma.¹¹⁻¹² Brain damage caused by a single concussion may become more extensive if the subject is intoxicated. Accordingly, minor head injuries may lead to more severe consequences among alcoholics than the general population. This may explain why our alcoholics, who had sustained untreated and unidentified head injuries, showed more signs of intellectual impairment in neuropsychological tests than uninjured alcoholics. In nonalcoholics mild head injuries seldom cause permanent impairment of mental functions.¹³

Tarter and Alterman¹⁴ have suggested that the number of head traumas actually suffered may be higher than that reported by alcoholics. Our data support this view and suggest that the prevalence of head injury is 2-4 times higher in alcoholics than in the general population. We believe that posttraumatic epilepsy and encephalopathy may also be more frequent among alcoholics, but this is difficult to prove since alcoholism is associated with seizure problems and encephalopathies of several different aetiologies. The frequency of such subjects who have head injuries in previous history is high among patients brought to the emergency wards of general hospitals because of alcohol-related seizures.^{15,16}

We agree with Grant *et al.*¹⁷ in that a detailed medical history is needed before permanent neuropsychological impairment of alcoholics can be ascribed to neurotoxic effects of ethanol instead of, for example, head injury. Since reversal of neuropsychological deficits can occur,¹⁷⁻¹⁹ further information needs to be obtained, by retesting abstinent patients 1-2 years after the first testing. This policy may reveal subjects who have irreversible deficits because of, for example, traumatic brain injuries.

In conclusion, traumatic head injuries seem to be about two to four times more prevalent in previous history of alcoholics than in previous history of the general population. Many alcoholics sustain single or multiple less severe head injuries which may, however, cause significant brain injuries, although these easily remain unidentified. Contrary to a previous observation²⁰ we have demonstrated that previously unrecognised traumatic brain injuries can contribute

to the intellectual impairment of alcoholics.

We thank Prof C-M Idestrom and Prof H Bergman for support during the study. We are also indebted to the staff of Magnus Huss clinic at Karolinska Hospital and to all the subjects who took part in the study. The work was supported by the Karolinska Project for Research and Treatment of Alcoholic Dependence (KARTAD).

References

- Honkanen R, Visuri T. Blood alcohol levels in a series of patients with special reference to accident and type of injury. *Ann Chir Gynaecol* 1976;**65**:287-94.
- Rutherford WH. Diagnosis of alcohol ingestion in mild head injuries. *Lancet* 1977;**i**:1021-3.
- Holt S, Stewart JC, Dixon MJ, Elton RA, Taylor TV, Little K. Alcohol and the emergency service patient. *Br Med J* 1980;**281**:638-40.
- Brisman B, Engstöm A, Rydberg U. Head injury and intoxication: A diagnostic and therapeutic dilemma. *Acta Chir Scand* 1983;**149**:11-14.
- Patel AR, Jennett B, Galbraith S. Alcohol and head injury. *Lancet* 1977;**i**:1969-70.
- Bergman H. Brain dysfunction related to alcoholism: Some results from the KARTAD project. In: Parsons OA, Butters N, Nathan P, eds. *Neuropsychology of Alcoholism: Implications for Diagnosis and Treatment*. New York: Guildford Press, 1986.
- Edwards G, Gross MM, Keller M, Moser J, Room R. *Alcohol Related Disabilities*. WHO Offset Publications 1977;no 32.
- Bergman H, Axelsson G, Idestrom C-M, *et al.* Alcohol consumption, neuropsychological status and computer-tomographic findings in a random sample of men and women from the general population. *Pharmacol Biochem Behav* 1983;**18**(suppl 1):501-5.
- Gulliksen H. *Theory of Mental Tests*. New York: Wiley, 1950.
- Wilkinson DA. CT scan and neuropsychological assessments of alcoholism. In: Parsons OA, Butters N, Nathan P, eds. *Neuropsychology of Alcoholism: Implications for Diagnosis and Treatment*. New York: Guildford Press, 1986.
- Flamm ES, Demopoulos HB, Seligman ML, Tomasula JJ, DeCrescito V, Ransohoff J. Ethanol potentiation of central nervous system trauma. *J Neurosurg* 1977;**46**:328-35.
- Persson L, Rosengren L. Increased blood-brain barrier permeability around cerebral stab wounds aggravated by acute ethanol intoxication. *Acta Neurol Scand* 1977;**56**:7-16.
- Gentilini M, Nichelli P, Schoenhuber R, *et al.* Neuropsychological evaluation of mild head injury. *J Neurol Neurosurg Psychiatry* 1985;**48**:137-40.
- Tarter RE, Alterman AI. Neuropsychological deficits in alcoholics: Etiological considerations. *J Stud Alcohol* 1984;**45**:1-9.

- ¹⁵ Earnest MP, Yarnell PR. Seizure admissions to a city hospital: the role of alcohol. *Epilepsia* 1976;**17**:387–93.
- ¹⁶ Hillbom ME. Occurrence of cerebral seizures provoked by alcohol abuse. *Epilepsia* 1980;**21**:459–66.
- ¹⁷ Grant I, Adams KM, Reed R. Aging, abstinence, and medical risk factors in the prediction of neuropsychological deficit among long-term alcoholics. *Arch Gen Psychiatry* 1984;**41**:710–8.
- ¹⁸ Parsons OA, Farr SP. The neuropsychology of alcohol and drug use. In: Filskov S, Boll T, eds. *Handbook of Clinical Neuropsychology*. New York: Wiley, 1981.
- ¹⁹ Carlen PL, Wilkinson DA, Wortzman G, Holgate T. Partially reversible cerebral atrophy and functional improvement in recently abstinent alcoholics. *Can J Neurol Sci* 1984;**11**:441–6.
- ²⁰ Lee K, Møller L, Hardt F, Haubek A, Jensen E. Alcohol-induced brain damage and liver damage in young males. *Lancet* 1979;ii:759–61.