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Organic Psychosyndromes in Chronic Alcoholics

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Summary
The different organic psychosyndromes that are encountered in chronic alcoholics are reviewed, and their clinical features and investigation discussed.

Alcohol is capable of disturbing brain function in both acute and chronic stages of abuse, and can produce a wide range of clinical syndromes. The confusional stages of acute intoxication, the alcoholic 'blackout', and the different withdrawal states occur frequently, are reversible, and are easy to recognise. There is a second group of organic psychosyndromes associated with alcohol abuse that are characterised by a more chronic and often irreversible cognitive impairment. This group encompasses the Wernicke-Korsakoff's psychosis and a variety of related cognitive deficits present in a large number of those referred to alcoholic units.

An attempt will be made here to describe the clinical features, frequency, prognostic and therapeutic implications of the second group of syndromes.

The Size of the Problem

How often are these organic psychosyndromes encountered in the every day clinical practice of an alcoholic unit? Surprisingly, not many systematic studies have tried to answer this question. Horvath [1] found that in an Australian University Centre for the treatment and research into alcoholism, out of 1,100 patients seen in the course of five years, 9 per cent had a chronic organic brain syndrome as defined by progressive failure of memory, loss of intellectual ability and personality deterioration. These cases were obviously among the most severe and no comments were made about the rest. Of the 100 patients with organic psychoses 20 suffered from a typical Korsakoff's psychosis following Wernicke's encephalopathy; the rest presented a combination of memory failure, behavioural disturbance, apathy, depression, lack of motivation and, in some cases, more specific neurological deficits such as dysphasia or dyspraxia.

The classical picture of the Wernicke-Korsakoff's psychosis is easy to recognise. An initial confusional state with ocular palsies, ataxia and often peripheral neuropathy is followed by an amnesic syndrome with severe disturbance of memory for past events (retrograde amnesia) and difficulty in acquiring new information (anterogade amnesia). In typical cases the prognosis is poor, and Cutting [2] found that only 14 per cent of those who had been admitted to the Maudsley and Bethlem Royal Hospitals were able to leave hospital. Although males predominated in this group, the females had a shorter drinking history.

It has recently become clear that the classical picture, which is relatively uncommon, represents only part of a much larger problem. Harper [3] has found

the typical neuropathological lesions of Wernicke's encephalopathy in 1.7 per cent of all the post-mortem brains examined in a general hospital serving a well defined geographical area. Only a minority of these cases had been correctly diagnosed during life despite the fact that the majority were alcoholics. In a proportion of cases the neuropathological lesions were chronic or acute on chronic. It is therefore possible that the symptoms and signs of thiamine deficiency may sometimes pass unnoticed, and that a syndrome with some of the features of a Korsakoff's psychosis could appear insidiously without the early features of Wernicke's encephalopathy. Cutting [2] has found that this picture, sometimes called 'alcoholic dementia', is more likely to affect older individuals than the abrupt onset variety, is commoner in women and in those with a longer drinking history. In the slow onset group the prognosis was much better, and two-thirds were well enough to live independent lives at follow-up.

If the two forms of Wernicke-Korsakoff's psychosis together account for the majority of Horvath's 9 per cent with organic syndromes, over 90 per cent remain who at first sight might be considered 'intact' alcoholics.

Brain damage in the 'intact' alcoholic

The great majority of those patients going through an alcoholic unit do not suffer from overt cognitive deficits, and after a period of detoxification and treatment are discharged often without detailed psychometric or neuroradiological investigation. As a consequence subtle cognitive and structural brain changes that could have serious implications in potentiating addiction or in making rehabilitation arduous could be missed.

There is evidence that a substantial proportion of these patients have neuropathological lesions and poor intellectual performance as a result of chronic drinking. This area has been reviewed elsewhere [4] and only the salient points will be mentioned here.

The cognitive impairment

Alcoholics have IQs similar to those of the general population [5, 6, 7] and there is no reason to believe that brain damage due to other causes antedates alcohol abuse in all but a small minority of cases. However, standard intelligence tests are not appropriate tools with which to demonstrate psychological impairment in alcoholics, and a clearer pattern of deficits emerges with the use of specific tests for the detection of brain damage, particularly those sensitive to frontal lobe lesions.

Chronic alcoholics perform badly on tests of abstract thinking, problem solving ability, psychomotor speed, and memory tests particularly non-verbal memory [8, 9]. These studies have all been carefully controlled and have clearly demonstrated that even in alcoholics with normal IQs the performance in these specific tests is often similar to that of patients with established brain damage due to other causes. Interestingly the deficits can also be demonstrated in alcoholics with superior intelligence [10] and in moderate social drinkers who have never been referred to an alcoholic unit [11].

Improvement in cognitive performance occurs rapidly during the first two

weeks of abstinence and perhaps more slowly for a few weeks thereafter, but deficits of the type already described can be detected after a year of abstinence [12].

The deficits are more severe in older patients with longer drinking histories, giving some indication that the impairment is probably progressive [13].

Although the severe amnesic defect of the Wernicke-Korsakoff's psychosis is not marked in these patients, the other cognitive deficits do not appear to be qualitatively different [14], and it is possible that the periventricular lesions typical of thiamine deficiency account for the loss of memory, while the cortical damage, perhaps directly related to alcohol, accounts for the poor performance on other tasks.

The neuropathological and neuroradiological lesions

The neuropathological lesions of Wernicke-Korsakoff's psychosis have been carefully described by Victor et al. [15]. Other lesions in alcoholics who do not suffer from the syndrome have also been studied. Courville [16] described cortical atrophy and ventricular enlargement; the damage was widespread, but affected the frontal lobes more severely. The microscopic picture was of loss of neurones and glial proliferation. Although post-mortem studies are based on elderly populations with long histories of alcohol abuse and superadded physical illness and therefore not representative of the alcoholic population as a whole, Courville felt justified in saying that alcoholism was the commonest cause of cerebral atrophy in the fifth and sixth decades of life.

Cerebral atrophy has been demonstrated in chronic alcoholics during life by the use of different neuroradiological techniques, pneumoencephalography (PEG) and, more recently, Computerized Axial Tomography (CAT). Brewer and Perrett [17] used PEG to demonstrate cerebral atrophy in 31 of 33 patients with a mean age of 50, and who drank a minimum of six pints of beer or its equivalent per day. These patients were by no means typical 'skid-row' alcoholics; most of them were considered to be moderate or heavy drinkers. Cortical atrophy occurred in 30 patients; frontal atrophy was found to be common, followed by parietal atrophy. Ventricular enlargement was present in 24 cases. Intellectual impairment correlated positively with the presence of cerebral atrophy. Similar findings have been reported in young alcoholics in whom other causes of brain damage were carefully excluded.

Due to the morbidity and discomfort inherent in the PEG only selected and uncontrolled populations could be investigated, and the true incidence of these abnormalities in alcoholics as a whole could have been easily overestimated. CAT has solved some of these problems and a number of studies are now available. In our series [18] 49 male alcoholics admitted to a rehabilitation unit, aged from 30 to 60, and apparently clinically 'intact', have been investigated. The results were compared with an age-matched group of normal volunteers who were abstemious or who drunk very little. The patients had an average drinking history of 19 years and peak drinking during a heavy drinking period of approximately two bottles of spirits per day. A drying out period of at least two weeks elapsed before testing.

Only 17 patients had normal CAT (35 per cent). Cortical atrophy was pres-

ent in 55 per cent and ventricular enlargement in 35 per cent. The presence of cerebral atrophy was positively correlated with the age of the patients and the length of the drinking history. Other clinical features such as the drinking pattern during the previous year, the peak per day consumption, and the frequency and severity of withdrawal symptoms failed to show any significant correlation.

Although the original IQs of the patients were similar to that of the controls they performed significantly worse in tests of verbal and non-verbal memory and in tests indicative of frontal lobe damage. These cognitive and radiological abnormalities occurred in patients with normal liver function.

The clinical features

The evidence so far suggests that the frontal lobes in particular are implicated although the damage is clearly widespread. Lesions in these areas may impair the critical abilities of the individual, and are often accompanied by affective changes of apathy or euphoria. Moreover, the cognitive changes that accompany frontal lobe lesions make it difficult for the patients, when faced with a choice, to analyse information and select possible alternatives, choosing as a result the first available possibility, perpetuating an inappropriate type of behaviour, failing to learn from previous mistakes.

Affective changes, poor judgement, and relapses into drinking are so common in chronic alcoholics that their presence is often taken for granted, and this is also true of 'inability to abstain' and 'loss of control'. It would be naïve to believe that these features are entirely due to the presence of brain damage, but the importance of its role should not be understimated.

Little is known about the amount of drink, the number of years, or the drinking pattern necessary to produce pathological changes, but it is alarming that mild cognitive impairment can be recognised in moderate social drinkers. It appears that those alcoholics with less psychiatric disturbances, better work records, and more stable lives show more cognitive impairment than those with psychiatric illness and more irregular life styles, probably because as a result of their stability they are able to ensure a higher and steadier alcohol consumption [19].

As with other physical illness related to alcohol there are some indications that after a certain level of consumption intellectual decline becomes a function of the amount of alcohol consumed. This threshold is difficult to predict accurately but Eckhard et al. [20] have suggested that it is in excess of a life consumption of 400 gallons of alcohol, a drinking history of 15 years and a peak consumption in the preceding six months greater than 2.5 G/Kg/occasion (three-quarters of a bottle of spirits for a man of average weight). At lower levels cognitive impairment can still be detected, but its presence is less predictable and not clearly related to the amount of alcohol consumed.

Conclusions and Implications

It appears that a substantial proportion of those referred to an alcoholic unit suffer from alcohol related brain damage. Nine or ten per cent will present with severe organic psychosyndromes of abrupt or insidious onset; most can be con-

sidered as suffering from different clinical forms of Wernicke-Korsakoff's psychosis. Of the remaining 90 per cent neuroradiological cerebral atrophy can be demonstrated in over half of those who appear clinically 'intact'. Cognitive impairment is also present, and problem solving ability, abstract thinking, psychomotor speed and memory appear particularly vulnerable. The intellectual deficits and the severity of cerebral atrophy increase with age and length of drinking history. Other clinical features such as the frequency and severity of the withdrawal symptoms do not appear to be closely correlated with the presence of brain damage. We know very little about the aetiology and mechanism of production of the damage but it is likely that the effects of alcohol per se together with thiamine deficiency will account for the best part. Clinically there is evidence that the damage found in the 'intact' chronic alcoholic is on a continuum with the Wernicke-Korsakoff psychosis. The abnormalities described here are demonstrable in patients with normal liver functions, making a causal link unlikely, and suggesting that the brain may be more sensitive than the liver to the damaging effects of alcohol, at least in a proportion of patients.

There are still large areas where our knowledge is only rudimentary. The prognostic implications and the natural history of these lesions are but examples. Only a careful follow-up study can tell us whether the 'brain atrophy' detected neuroradiologically is permanent and progressive or, at least in part, reversible with prolonged abstinence. Profound metabolic changes in the early stages of withdrawal or reversible histological lesions could account for some of the demonstrable 'atrophy' [21].

For the clinician immersed in the everyday care of patients only tentative guidelines can be suggested for the investigation and management of the 'intact' alcoholic. It would seem reasonable that those who after two or three weeks of abstinence still appear to be clinically suffering from even minor disorientation, loss of memory, and inability to learn new material on clinical testing, should be investigated fully, especially if over forty and after a long drinking history. The investigation of an alcoholic patient in whom cognitive impairment is suspected should not only include standard intelligence tests, but tests specifically designed to detect brain damage, and in particular tests of verbal and non-verbal memory with immediate and delayed recall and tests sensitive to frontal lobe lesions. CAT scans, when available, should also be done, not only to confirm the presence of cerebral atrophy but to exclude other possible causes of persistent confusional states, e.g. subdural haematoma, etc. And if Harper's [3] findings of underdiagnosed Wernicke's encephalopathy are considered, a plea should be made for routine early administration of thiamine particularly in those who show even the slightest suggestion of cognitive impairment.

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