

EDITORIAL / VAN DIE REDAKSIE

Alcohol and brain damage

Alcoholism may constitute the major health problem in many parts of the world, with its effects on morbidity and mortality grossly underestimated in world health statistics.¹ Alcohol-related neuropsychiatric disorders constitute a particularly large and incapacitating subset of the medical complications of alcoholism.² There are a number of well-described neuropsychiatric syndromes associated with alcohol withdrawal (e.g. withdrawal delirium, hallucinosis, seizures) or concomitant nutritional deficiencies (e.g. Wernicke-Korsakoff syndrome, polyneuropathy, pellagra).³

Alcoholic brain damage has traditionally been viewed in straightforward terms. Thus, Wernicke-Korsakoff syndrome has long been regarded as the classic form caused by vascular lesions in di-encephalic structures as a result of thiamine deficiency. Wernicke's encephalopathy is the acute component, manifesting clinically as impairment of consciousness, accompanied by ataxia and ophthalmoplegia, while Korsakoff's syndrome is the chronic sequel of the same pathological process and is characterised by profound retrograde and anterograde amnesia.⁴ Research has shown that memory deficits of patients with Korsakoff's syndrome are not entirely pure and that other cognitive functions, especially visuo-perceptive and problem-solving capacities, are also impaired. There appears to be marked variability in the degree of cognitive impairment, with female patients particularly severely affected.⁵ Wernicke-type pathology is far more common than is generally recognised, and may develop surreptitiously. In a neuropathological study in Western Australia, morphological evidence of the disorder was found in 1,7% of all autopsies performed, only the minority of whom had been diagnosed as such during life. The Wernicke-Korsakoff syndrome was, until fairly recently, thought to account for the majority of alcoholics who suffered lasting cognitive impairment, and those who escaped this development were regarded as being essentially 'intact'.⁶

Evidence from neuropathological and neuroradiological studies was, however, rapidly accumulating to show that diffuse brain damage can occur in alcoholics.³ This brain damage is not always clinically obvious, but refined psychological tests have been able to demonstrate that a large proportion of alcoholics actually do have impaired cognitive functioning.⁶ Heavy consumption of alcohol results in the increasing danger of cognitive impairment. In fact, there is some evidence to suggest that such impairment may be detectable even in the so-called social drinker!⁷ The frontal lobes appear to be most severely affected by chronic, excessive consumption of alcohol.⁸ Frontal lobe dysfunction may manifest with symptoms such as decreased ability to make abstractions, lack of insight and impaired impulse control — functions that are of critical importance in any attempt to rehabilitate the alcoholic. In this way the development of even subtle brain damage may be a significant factor in the perpetuation of the alcoholic's dependency problem.

Computed tomographic (CT) studies have shown that alcoholics have enlarged ventricles and widened cortical sulci.⁹ These changes are broadly associated with cognitive impairment.⁵ A great number of alcoholics — perhaps the majority of severe alcoholics — impair their brains quite early on. This impairment, although structurally demonstrable, appears in most cases to remain relatively benign over many years and, to some degree, is potentially reversible by abstinence.¹⁰ As the alcoholic gets older, however, perhaps as the result of the alcoholism's interaction with other pathological conditions (e.g. ageing, trauma, hepatic dysfunction, vascular changes), he may develop a more severe global impairment of cognitive function — so-called 'alcoholic dementia'.⁴

It has been proposed that two separate pathological processes may contribute to brain damage in alcoholics: (i) severe thiamine deficiency because of dietary neglect, poor absorption and utilisation for the metabolism of alcohol, with consequent di-encephalic damage and memory impairment; and (ii) cortical shrinkage caused directly by alcohol neurotoxicity, which leads to widespread cognitive impairment.¹¹ Thus, Korsakoff's syndrome may in fact represent only the tip of an iceberg; many other alcoholics may be affected to a lesser degree by the same pathological processes.⁶

Unfortunately, the causes of alcoholism are still largely unknown, and its treatment remains unsatisfactory. Priorities are primary prevention and early identification and treatment of those at risk. The public at large needs to know of the hazards involved. Therapeutic intervention needs to take place before physical dependence and cognitive impairment compound an already difficult task.

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