

shown that they have little difficulty in sticking to this sort of diet provided that they eat at home, a finding similar to that of Thaler *et al* in New Zealand.<sup>18</sup> Long term compliance is helped by the fact that once patients have overcome the first few weeks of the change in the diet, in which food may taste bland, salt sensitivity of the taste receptors in the mouth changes.<sup>19</sup> Most patients then find that the foods with a high salt content that they used to like are unpleasant and much prefer foods with less salt. Our results also suggest that once the formation of angiotensin II is blocked more severe sodium restriction would be even more effective in decreasing blood pressure. For patients to reduce sodium intake below 80 mmol/day, however, requires the provision of special foods, particularly salt free bread, that are not yet generally available in the United Kingdom and the avoidance of most processed foods.

We think that in patients with mild to moderate essential hypertension who are prepared moderately to reduce their sodium intake such a reduction, together with other non-pharmacological advice, should be the first step in their management. If this fails to control their blood pressure additional treatment with an angiotensin converting enzyme inhibitor such as captopril is particularly effective. This approach overcomes the objections of lack of efficacy that have been made both against moderate salt restriction alone and converting enzyme inhibition alone.

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# Are we drinking our neurones away?

CLIVE HARPER, JILLIAN KRIL, JOHN DALY

## Abstract

**A quantitative neuropathological necropsy study of the human cerebral cortex showed that the number of cortical neurones in the superior frontal cortex in chronic alcoholic patients is significantly reduced compared with that in controls matched for age and sex. The number of neurones in the motor cortex did not differ significantly between the controls and alcoholics, but in both cortical regions there was evidence that alcoholic patients had smaller (shrunken) neurones than controls.**

**Further studies are necessary to identify other regions of the cerebral cortex that are selectively damaged in brain damage associated with alcohol.**

## Introduction

Alcoholic beverages have been available in almost every civilisation known to man. Today, particularly in Western countries, they are an accepted part of society.

The popular belief that excessive alcohol (ethanol) intake, particularly "binge" drinking, causes damage to nerve cells and death in the cerebral cortex is not based on any reliable scientific data. Pathologists have commented on a "patchy loss of cortical neurones" in the brains of alcoholic patients,<sup>1</sup> but this has not been quantified, and similar subjective observations in other diseases have often been shown to be invalid. There are experimental data, however, that show a loss of neurones in animal models of chronic alcohol intoxication.<sup>2</sup> In a recent collaborative study we documented the loss of neurones in the cerebellar vermis of alcoholics.<sup>3</sup>

With regard to the cerebral cortex neuroradiological evidence shows that chronic alcoholic patients<sup>4</sup> and even heavy social drinkers<sup>5</sup> suffer from brain "shrinkage," and there is some correlation between this change and clinical and neuropsychological deficiencies. In recent quantitative neuropathological studies we have shown that the weights of the brains of chronic alcoholics are significantly reduced<sup>6</sup> and that the pericerebral space (the space between the brain and the skull) is increased.<sup>7</sup> Stereometric measurements of the volumes of cortical grey matter, white matter, and basal ganglia in the brains of controls and alcoholics have shown that the loss of cerebral tissue in alcoholics is largely from the white matter.<sup>8</sup> This shrinkage of white matter could be caused by loss of neurones with subsequent degeneration of axons. The purpose of this study was to investigate such a hypothesis.

Department of Pathology, University of Sydney, and Royal Prince Alfred Hospital, Sydney, Australia

CLIVE HARPER, MB, FRCPA, professor of neuropathology

Department of Pathology, University of Sydney

JILLIAN KRIL, BSC, research assistant

Department of Neuropathology, Royal Perth Hospital

JOHN DALY, BSC, research assistant

Correspondence to: Professor C Harper, Department of Pathology, University of Sydney, Sydney 2006, Australia.

## Materials and methods

Two hundred and forty brains obtained by necropsy were studied in collaboration with the forensic pathologists of the Perth city coroner's department. Forty four brains were selected for this study. One of us (either JK or JD) attended the necropsies to carry out the procedures described below.

The selection of brains from alcoholic and control subjects was based on a compilation of clinical and pathological data. The data included clinical notes available from previous admissions to the teaching hospitals of Perth, the results of detailed questionnaires on alcohol intake and nutritional state provided by relatives of the dead subjects, reports about the circumstances of death, and the results of a complete necropsy and microscopical examination of the tissues including the liver and brain. Patients who had had a history or showed pathological evidence of neurological diseases, other than those associated with alcoholism, were excluded. Many of the 240 patients had to be omitted from this study because the information on their drinking habits was inadequate to allow them to be classified satisfactorily as a control or an alcoholic. A third group of moderate drinkers was also identified, and these cases will be studied at a later date.

Cases of Wernicke's encephalopathy, which is caused by vitamin B<sub>1</sub> (thiamine) deficiency, were included if the patients had a history of heavy drinking because previous studies have shown that Wernicke's encephalopathy is closely associated with alcoholism.<sup>9</sup> Any patient with evidence of head injury was excluded.

The brains of 22 alcoholics and 22 controls were included in the study. The controls were matched for age and sex to the alcoholics. Four of the control patients had been teetotal, and the remainder drank less than 20 g alcohol a day, which is considered to be well within the safety limits of alcohol intake.<sup>10</sup> Seven of the alcoholic patients had had pathologically confirmed Wernicke's encephalopathy, and nine had had cirrhosis of the liver. Three had had both, and nine patients had had neither.

The fresh brain volume, fresh brain weight, and intracranial volume were measured at necropsy in each case. The cerebellum and brain stem were removed by sectioning the midbrain. The hemispheres were divided in the sagittal plane, and the right hemisphere was frozen to -70°C for neurochemical analyses, which will be used in other studies. The weight and volume of the left hemisphere were measured, and then it was fixed in 10% formal saline for exactly two weeks and the weight and volume remeasured. It was then embedded in 3% agarose with the occipitofrontal axis at a right angle to the face of the block. After the agarose solidified it was cooled in the refrigerator for several hours before the hemisphere was cut into 3 mm coronal slices with an electric meat slicer. Each slice was photographed with a scale, and black and white prints of actual size were prepared. Each brain produced about 60 slices, and the mean thickness of each slice was determined by dividing the number of slices into the occipitofrontal length of the fixed cerebral hemispheres. The photographs were overlaid by a transparent square grid system, each square having an area of 0.4 cm<sup>2</sup>, and a standard point count was carried out.<sup>11</sup> Three separate regions were counted: the cerebral cortical grey matter, the white matter, and the diencephalic structures, including the basal ganglia, thalamus, hypothalamus, and amygdala. We shall refer to the last group as the basal ganglia throughout this paper. Calculations were performed according to the Delesse principle,<sup>11</sup> and areas and volumes were calculated for each of the four regions in the cerebral hemispheres.

Blocks of the brain were taken from the superior frontal gyrus (area 8 according to Brodmann's classification<sup>12</sup>) and the motor cortex (Brodmann's area 4). The areas of these blocks were measured with a Quantimet 900 (Q900) image analysis system (Cambridge Products). After the blocks were embedded in paraffin wax with standard procedures sections of 10 µm thickness (checked using the "focus through the section" technique) were cut and stained with haematoxylin and eosin, Nissl/haematoxylin, and Weil's stain for myelin.

Blocks were also taken from the medial temporal lobe (including Ammon's horn), thalamus, hypothalamus (including mammillary bodies), basal ganglia, midbrain, pons, medulla, and cerebellar vermis and lateral hemisphere. These were stained as described above and examined microscopically to identify other pathological lesions such as Wernicke's encephalopathy or Alzheimer's disease. Cases of Alzheimer's disease were excluded from the study.

The Nissl/haematoxylin stained sections of the superior frontal and motor cortex blocks were used for studies of the numbers and sizes of cortical neurones and glial cells. The area of this section was measured with the image analysis system so that a correction factor for shrinkage during processing could be calculated. The Q900 image analysis consists of a Leitz Orthoplan microscope fitted with a Plumbicon television camera linked to an automatic detector. This allowed the projected image that was seen on a monitor screen to be analysed. The microscope was fitted with a mechanical stage controlled by a computer. All readings were made at a magnification of 250. The area and number of detected features were

recorded simultaneously. The detection threshold for each field was adjusted so that the binary image (that which is measured) most closely represented the area of the cells. We did not perform any manual editing with the light pen. Though Terry *et al* have emphasised the importance of video editing in automated image analysis studies of the cerebral cortex,<sup>13</sup> we believe that because this was a direct comparative study of paired controls and alcoholics the methods as described were satisfactory.

Features (cells) were sorted into two categories by size, based on measurements of the surface area of the cells. Features with a surface area of 5-40 µm<sup>2</sup> corresponded to glial cells (astrocytic and oligodendroglial nuclei) and those of 41-350 µm<sup>2</sup> to neurones (nuclei plus cell bodies). These categories correspond to those selected by other authors who have used image analysis techniques,<sup>14 15</sup> and the results correlate well with manual counts. Features with a surface area of less than 5 µm<sup>2</sup> were not counted. This eliminated electronic noise and fragments that were too small to be recognised reliably as cells.

Strips of cortex one frame wide and perpendicular to the surface of the cortical gyri were used. There were usually 10-12 fields per strip. The strips were selected randomly from each section until roughly 65 fields had been counted. After each field had been measured the results were printed out, and the microscope stage automatically moved to the field immediately adjacent.

The data from each field included counts of neurones and glial cells and the sums of the areas occupied by these two groups of cells. Histograms (count *v* area) were also prepared. The numbers of neurones and glial cells per unit volume were determined according to the following equation: cell count/mm<sup>3</sup> = (cell count × 10<sup>9</sup>) / (field area (µm<sup>2</sup>) × number of fields × section thickness (µm)). The counts were corrected for shrinkage caused by tissue processing as discussed previously. The counts of glial cells in the deep white matter were performed on the same histological sections in a similar fashion to that described above. Twelve fields were measured for each case.

The data were analysed and comparisons made between the neurone counts, glial counts, and neuronal size (area) in the control and alcoholic groups. Statistical analyses were performed with Student's *t* test on an HP 9825 desktop computer.

## Results

The table summarises the pathological data and the measurements performed in the controls and alcoholics (all men).

*Pathological data and measurements performed during study. (Values are expressed as means (SD))*

	Controls (n=22)	Alcoholics (n=22)	Significance*
Age (years)	59 (14.3)	58 (12.0)	NS
Weight of brain (g)	1429 (105)	1315 (108)	p<0.001
Pericerebral space (as % of intracranial volume)	9.2 (3.7)	16.0 (4.3)	p<0.001
Volume (as % of cerebral hemisphere volume)			
of:			
Cerebral cortex	67.8 (5.9)	63.9 (5.5)	p<0.05
White matter	47.5 (4.4)	42.5 (4.4)	p<0.01
Basal ganglia	5.0 (0.3)	5.1 (0.4)	NS
Neuronal counts (× 10 <sup>3</sup> /mm <sup>3</sup> ):			
Motor cortex	27 (4.0)	29 (5.6)	NS
Superior frontal cortex	27 (4.2)	21 (6.3)	p<0.001
Neuronal areas (µm <sup>2</sup> ):			
Motor cortex	127.1 (11.9)	117.6 (11.2)	p<0.01
Superior frontal cortex	125.5 (10.6)	103.0 (15.4)	p<0.001
Glial cell counts (× 10 <sup>3</sup> /mm <sup>3</sup> ):			
Motor cortex	72 (8.2)	77 (7.9)	p=0.05
Superior frontal cortex	71 (8.6)	80 (9.3)	p=0.0017
Motor white matter	132 (23.8)	147 (26.5)	p=0.0622
Superior frontal white matter	168 (26.9)	169 (32.3)	p=0.8998

\*Student's *t* test.

The ages ranged from 21 to 85 in the controls and from 23 to 81 in the alcoholics. There was no significant difference between the alcoholic and control groups with regard to age, number of hours between death and necropsy, or percentage shrinkage of the brain tissues during processing. Anderson *et al* have confirmed that some changes in the neuronal content of the cerebral cortex are related to age,<sup>16</sup> but as our two study groups were matched for age this factor was not considered further.

The results obtained from the brains of alcoholics who had had Wernicke's encephalopathy or cirrhosis of the liver and from those who had had neither were also compared with those from the control population. Alcoholics who had had Wernicke's encephalopathy or cirrhosis showed

a significant reduction in the number ( $19 \times 10^3$  neurones/mm<sup>3</sup> and  $21 \times 10^3$  neurones/mm<sup>3</sup>, respectively) and area ( $113.0 \mu\text{m}^2$  and  $97.4 \mu\text{m}^2$ , respectively) of the neurones in the superior frontal region. No significant differences were seen between the Wernicke's encephalopathy and cirrhosis subgroups and the controls in the neurone counts in the motor cortex, but the neurones were shrunken in the alcoholic subgroups. The counts of glial cells in the cortex and the white matter were increased. Those alcoholics who had not had Wernicke's encephalopathy or cirrhosis showed changes similar to, but not as severe as, those of the whole alcoholic group.

## Discussion

Despite the expectation of these findings by many doctors and pathologists we believe that this is the first objective documentation of loss and shrinkage of neurones in the cerebral cortices of alcoholic patients. Though many other regions of the cortex need to be similarly studied, it is interesting that the anterior part of the frontal lobe (superior frontal gyrus) seems to be more severely damaged than the motor cortex in such patients. These anatomical sites were chosen deliberately for examination because both neuroradiologists<sup>17</sup> and clinical neuropsychologists<sup>18</sup> have suggested that in alcoholic patients the anterior frontal lobes are more severely damaged than other parts of the cerebral hemispheres. Selective damage of specific neuronal populations has been found in animal models of ethanol toxicity.<sup>19, 20</sup> In these studies workers documented the loss and shrinkage of hippocampal pyramidal neurones<sup>19</sup> and cerebellar Purkinje cells.<sup>20</sup>

The highly significant increase in the counts of glial cells in the superior frontal cortices of the alcoholic patients is consistent with the concept of progressive degeneration and death of cortical neurones and subsequent proliferation of astrocytes as a repair phenomenon. The cerebral shrinkage noted in alcoholic patients by neuroradiological<sup>5, 17</sup> and, more recently, pathological studies<sup>7</sup> may also be explained by such a mechanism. The loss of neurones and the subsequent degeneration of axons will cause cerebral shrinkage, particularly of the white matter. Such a change, however, is irreversible, and several neuroradiological studies have shown that brain shrinkage in alcoholic patients is at least partially reversible after several months of abstinence from alcohol.<sup>21, 22</sup> The reversibility, however, is often incomplete and is most dramatic in younger patients who have a shorter drinking history.<sup>17</sup> The brains selected for our study were those from patients who had had the longest and greatest intake of alcohol. The mean age of the subjects was 58, so they had probably been drinking heavily for at least 30-40 years. The reduction in the volume of white matter may have two components—an irreversible component, caused by the death of neurones and subsequent degeneration of axons, and a reversible component. The irreversible component should predominate in long term drinkers such as those studied for this report. The pathogenetic basis for the reversible component has yet to be characterised.

It might be argued that as the measurement gates for neurones ( $41\text{-}350 \mu\text{m}^2$ ) were not changed throughout the study and as the neurones in the alcoholic group showed significant shrinkage the reduction in the number of neurones in the frontal cortex was caused by a shift of neurones into the glial cell compartment. A study of the pattern of distribution and mean glial and neuronal surface area measurements, however, does not support this explanation.

The shrinkage of neurones in the cerebral cortex of alcoholics is not totally unexpected. As Cragg and Phillips noted in their review of the toxic effects of alcohol on brain cells: "One reason that cellular diameter might be reduced is that prenatal exposure to alcohol can reduce the amount of dendritic branching. . . ."<sup>23</sup> Other animal studies<sup>24</sup> and one human study<sup>25</sup> have reported changes in the dendritic arborisation of Purkinje cells after long term consumption of alcohol. Much has been written recently about "neuronal plasticity," and it is becoming evident that considerable changes can occur quite quickly in the dendritic arborisation of neurones in the central nervous system.<sup>26</sup> Such changes are usually associated with alterations in the size of the neuronal cell body, as shown in this study.

A high proportion of the alcoholic patients in this study had complex medical conditions, including Wernicke's encephalopathy and cirrhosis of the liver. These cases showed more severe loss and shrinkage of neurones than the purely alcoholic group. The greater severity of these changes may reflect either the severity of alcohol abuse or that other factors such as thiamine deficiency or hepatic dysfunction, or both, play an important part in the pathogenesis of brain damage in alcoholics.

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## 100 YEARS AGO

At this season of the year, stewed apples, pears, and plums are favourite articles of diet. For breakfast or luncheon, in the dining-room or in the nursery, there are few table dishes more wholesome and more delicious than well-stewed fruit, served up with cream or custard. There are many persons, however, who cannot eat it, on account either of the acidity of the fruit or the excess of sugar necessary to make it palatable. Sugar does not, of course, counteract acidity; it only disguises it, and its use in large quantities is calculated to retard digestion. The housewife may, therefore, be grateful for the reminder that a pinch—a very small pinch—of carbonate of soda, sprinkled over the fruit previously to cooking, will save sugar, and will render the dish at once more palatable and more wholesome. (*British Medical Journal* 1887;ii:521.)