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Alcohol related hepatic lesions in young victims of road traffic accidents in Australia

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There is considerable evidence that the advanced stage of alcoholic cirrhosis results from alcohol abuse over 15–20 years (1, 2). The natural history of alcoholic liver disease (ALD), especially in the early phases, however, is not clear (1, 3). Tissue damage as seen in liver biopsy specimens is therefore still one of the more reliable indices of alcoholic liver damage (2).

As ethical considerations preclude liver biopsy in a healthy population who drink alcohol, our knowledge of the tissue effects of alcohol ingestion in healthy young adults is poorly documented (4). A high proportion of the victims of fatal road traffic accidents in Australia are young and apparently healthy. The association between drinking and road traffic accidents is high (5). The liver morphology in young victims of road traffic accidents was therefore examined to study the possible early tissue effects of alcohol in an otherwise healthy, mainly young population.

Materials and methods: Evidence of ALD was sought during coronial post mortem examination on 50 consecutive, apparently healthy, road traffic accident victims in the age group 15–35 years. All had died within 24 h of the accident. There were nine females and 41 males (Figure 2). Over 95% of the subjects were under 30 years of age. Blood alcohol level was tested in 43 cases by standardised gas liquid chromatography (6). The nutritional state, height, weight and general body stature was recorded. According to these criteria, none of the subjects was classified as being grossly obese. At post mortem examination there was no evidence of other major systemic disease in any of the fatalities. The liver of each victim was examined both macroscopically and microscopically. Specimens of liver were processed for light microscopy by standard methods. Alcohol associated histological changes were graded according to the criteria recommended by an international panel (2).

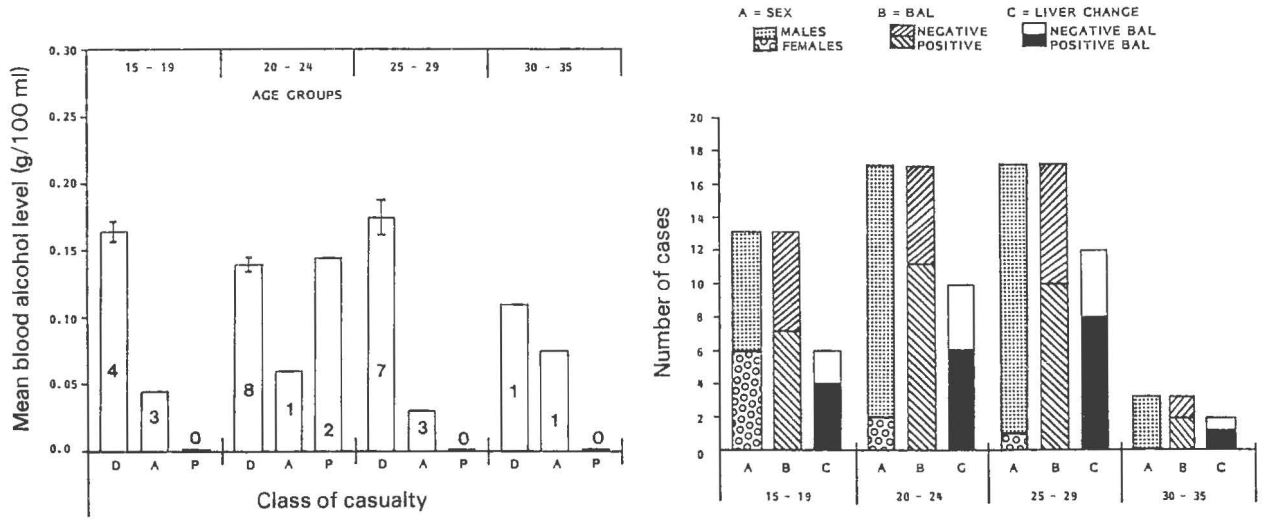

Figure 1 (left): Histogram of mean blood alcohol level in a particular age and class of casualty (± SEM if there were four or more in the group). Total no. of casualties = 30; blood alcohol level for all casualties = 0.124 ± 0.013 g/100 ml (mean ± SEM). D = driver; A = passenger; P = pedestrian. Figure 2 (right): Histogram of age and sex distribution (A), numbers with positive blood alcohol levels (BAL) in that group (B) and numbers with histological evidence of fatty change of the liver (C). Total number of casualties = 50. The data are grouped by age category.

Results: The blood alcohol level was raised in 30 of the 43 subjects tested (Figure 1) with an overall mean of 0.124 ± 0.013 (SEM) mg/100 ml of blood. The mean blood alcohol level in each age group indicates at least periodic alcohol abuse in the majority of those who had been drinking. Although a high single blood alcohol level is not a true indicator of drinking habit it is still a useful marker of possible excessive drinking (4). A random negative blood alcohol level, however, cannot be regarded as identifying abstainers. Information regarding drinking histories was not available for correlation with either blood alcohol level or liver history.

There were no obvious lesions on gross macroscopic examination of the liver in any of our cases. However, the mean liver weight of the study group ($n = 50$) was 1634 ± 46 g and this was significantly higher ($p < 0.001$) than that stated for a normal population (7). The nature and severity of the histological changes are summarized in the Table. There were no cases of cirrhosis. Fatty change had a random rather than a lobular distribution with fat present in hepatocytes mainly in macrovesicular form. Occasional lipogranulomas were identified in association with fatty cysts in those with moderate and severe fatty change. The two cases of alcoholic hepatitis had central ballooning degeneration, occasional liver cell necrosis with focal polymorphonuclear leucocyte infiltration and early pericellular fibrosis. Mallory bodies, cholestasis, excess iron deposition and portal fibrosis were not encountered.

There was no correlation between the blood alcohol level and hepatic histology (Figure 2). Liver histology was normal

in 24% with raised blood alcohol level, whilst, conversely, 52% of those with no blood alcohol had evidence of fatty change. As other common causes of fatty change could be excluded at post mortem examination the cause of fatty change in those with no blood alcohol was most probably related to alcohol consumption. Overall, therefore, 62% of cases had evidence of liver abnormality, the most probable cause of which could be attributed to alcohol consumption. Comparison between the sexes was not possible due to the small number of females in this study.

Nature and severity of alcohol-associated liver changes in road traffic accident fatalities

		Fatty change			Alcoholic hepatitis			
Age (years)	Normal	Mild	Moderately severe	Severe	Minimal	Fully-developed lesions	Total	% Abnormal
15–19	7	6	0	0	0	0	13	46
20–24	7	7	3	0	0	0	17	59
25–29	4	8	2	1	1	0	16	75
30–35	1	0	1	1	0	1	4	75
Total	19	21	6	2	1	1	50	–
%	38		58			4	100	62

Discussion: The road traffic accident victims in this study can be regarded as healthy young adults. Hepatic enlargement and fatty change was present in 62% of what might generally be regarded as a healthy young population of 'social drinkers'. Fatty change is not a specific feature of alcohol abuse. However, other known common causes of simple fatty liver were excluded at post mortem examination. Whilst it has been shown that fatty change is common in known chronic alcoholics (1, 2), its prevalence in a general population of social drinkers is unknown (2). The present study suggests that fatty change of a mild and moderate degree is a common, although not constant, accompaniment of alcohol ingestion even in young adults. Viel *et al.* (8), in a coronial autopsy study, showed fatty change in 30% of known alcoholics under 30 years of age. Rubin and Lieber (4), in a small study on human volunteers, were able to demonstrate early fatty change in those who might be regarded as social drinkers. The low prevalence of more advanced ALD in our study is consistent with the observation that ALD and advanced lesions are probably the result of prolonged heavy intake over several decades (1).

The pattern of pathology encountered, mainly reversible fatty change, indicates the first phase of ALD and is consistent with the shorter period of exposure to alcohol (1). However, the appearance of severe fatty liver in two cases and of alcoholic hepatitis in two further cases, all under 35 years, demonstrated that progressive ALD can be accelerated even in young adults.

The high blood alcohol level in a substantial number of victims of road traffic accidents indicates that the population of so-called 'social drinkers' harbour latent alcohol abusers. Finally young victims of road traffic accidents provide a readily available source of detailed studies of the early tissue effects of alcohol.

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