Liver and obesity

Sir,—Following the paper by Braillon et al.,¹ we would like to confirm that also in our experience obesity per se does not play a major role in the development of liver disease. We have, in fact, recently reviewed the frequency of abnormal liver function tests (serum GPT>25 mU/ml and/or γ-globulines >2 g/dl) in 313 obese patients consecutively hospitalised from June 1981 to December 1984 in our medical unit. Fifty five (23 men with Body Mass Index – BMI=W (Kg)/H(m)² – of 40-8±9-8 (DS) and 32 women with BMI of 39-8±9-0) with mean age of 46-6±15-1 years corresponding to 17-5% of the patients were affected by one or both haematological abnormalities while the remaining 258 (85 men with BMI of 38-6±8-9 and 173 women with BMI of 44-3±9-4) with mean age of 48-1±12-3 years did not show any biochemical abnormality indicative of liver pathology. Among the two groups no difference was detected for the frequency of the following: diabetes 29% in the first group vs 36% of the second group, use of hepatotoxic drugs 24% vs 28-6%, alcoholism 13% vs 12%, hypertriglyceridermia 33% vs 45-3%, hypercholesterolemia 9% vs 14%, hyperphagia (with a diet mainly rich in CH and fat) 51% vs 60%, right heart cardiac failure 0% vs 1-2%. On the other hand, B hepatitis virus contact, evaluated by measuring serum B hepatitis antigen and autoantibodies, was much more frequent, 46-3% vs 17-4% (p<0-0001) in the first group. Cholelithiasis was also slightly more frequent (25%) in the first group than in the second group (15%) (p<0-06).

In conclusion these observations in a group of obese individuals, with hypertransaminasemia and/or hypergammaglobulinemia, in South Italy show that liver abnormalities are independent of obesity by itself and are mainly caused by contact with B hepatitis virus (endemic in our region). Alcoholism is the main cause of liver pathology in obese patients living in regions where alcohol consumption is very high (2–5).

F CONTALDO, M MANCINI, L A REED et al

Departments of Medicine and Metabolic Diseases, and Department of Gastroenterology, University of Naples, Naples, Italy.

References


Statistical tests for 2×2 tables

Sir,—The letter from Boyd and Marks¹ in the June issue of Gut is correct in saying that the 2×2 table

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<tr>
<td>TDB</td>
<td>10</td>
<td>2</td>
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<td>Cimetidine</td>
<td>5</td>
<td>8</td>
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gives a χ² value with Yates’ correction of 3-53 leading to p=0-06 rather than p<0-02 as claimed in the Lam et al² paper.

The references they give in their discussion, however, of which tests to use on which occasions, all date from before the main impact of the present computer revolution. As long as there is a suitable computer (and program) available there is now little reason to use anything but the exact test for 2×2 tables. There is a difficulty, however, in agreeing on what is the correct version of this to use for a two-tailed test. It is a pity that even eminent statisticians seem to disagree here. Those who have a taste for such things may be referred to a recent paper by Yates³ and the published discussion with it.

(This paper celebrated the 50th anniversary of Yates’ earlier paper on the subject.)

My own view is that there is nothing to be said in favour of doubling the observed one-tail probability, which is what Boyd and Marks⁴ are doing. Instead I believe the best rule to be to include in the second tail all terms such that the sum of their probabilities does not exceed the probability in the observed tail. In the case under discussion there are 11 possible tables (with the observed marginal totals) corresponding to probabilities of 0-00009, 0-00262, 0-02599, 0-11549, 0-25986, 0-31183, 0-20211, 0-06930, 0-01181, 0-00087 and 0-00002 respectively. The observed tail consists of the first three terms with a sum of 0-0287; the second tail consists of the last three terms, because to take the last four would give a sum exceeding the observed one. This gives a probability of 0-0127, and a total two-tail probability of 0-0287+0-0127=0-0414 which is significant at the conventional 0-05 level.

Most statisticians nowadays would agree that the fact that the probability is round about 1 in 20, in this instance, is what matters, however, rather than
whether it falls just one side or just the other of any arbitrary dividing line.

I D HILL

Clinical Research Centre,
Northwick Park Hospital,
Watford Road,
Harrow, Middlesex.

References


Conservative treatment of gastrointestinal haemorrhage

SIR,—I read with great interest the paper of Rofe et al (Gut 1985; 26: 481–4). Their conclusion that conservative management is good for upper gastrointestinal bleeding finds little support from their data. The number of patients with duodenal ulcer and gastric ulcer is small (86) and the number over 60 years of age (high risk) is not given.

Perhaps the most fundamentally worrying feature of this paper is that of the 18 patients who died of bleeding only six underwent necropsy. The concept of ‘unsalvageable’ patients with hepatic or cardiac failure who died of bleeding from unknown sites is a difficult one to accept. In short, their low ulcer mortality would be more meaningful if the cause of death of more than one third of these patients was known. The claim that a conservative surgical policy was followed is not substantiated; their indications for operation in gastric ulcer seems to match those of our own early or aggressive group.1 How then did they manage duodenal ulcer? The criteria used to decide operation are not given.

If so few of their gastric ulcer patients required surgery despite their defined policy perhaps our defined criteria for duodenal ulcer patients may not have been fulfilled; in short, that these patients did not have severe bleeding. Perhaps the patients with severe bleeding, the real challenges, were amongst those unfortunate patients who died of ‘uninvestigated but unsalvageable’ bleeding from unknown ulcers!

D L MORRIS

Department of Surgery,
Floor E, W Block,
University Hospital, Nottingham.

Reference


Books


The last 20 years has seen a dramatic rise in per capita alcohol consumption together with the inevitable increase in alcoholic liver disease in many parts of the world. This book consists of 14 review chapters written by international authorities on the pathology, epidemiology, and clinical aspects of alcoholic liver disease.

There are clear, detailed, well referenced accounts of alcohol metabolism (Lieber), the pathology (Hall) and the possible role of immune mechanisms in the pathogenesis (MacSween) as well as a consideration of collagen metabolism (Rojkind). There is a series of chapters containing a mass of less readily accessible information on epidemiology of alcoholism and alcoholic liver disease in Europe (including UK), USA, Japan, Australia, and South Africa. In the clinical section of the book there is a detailed critique of the various agents which have been tried in the treatment of alcoholic liver disease. In referring to the unsatisfactory state of current therapy Conn refers to the ‘blind leading the drunk’ and emphasises the paramount importance of abstinence in determining clinical outcome. There was an account of the intensive multidisciplinary treatment programme at the VA Centre in West Haven (Nocks) which relies on an initial period of inpatient treatment. Such programmes are clearly expensive and as they have never been shown to be more effective than more modest efforts, are unlikely to gain more ground in Britain at least during the present period of financial cutbacks. Finally the strategies open to prevent or at least control the problem are discussed by Joy Moser who comments that while the developed world maybe beginning to address the problem, there is little evidence of a will to extend this vigilance to the third world, where the rapidly expanding alcohol market may constitute an additional strain on efforts to promote health and development.'