among the human infants, though birth weight was reduced. They concluded from these results that serious developmental defects did not occur unless carboxyhaemoglobin levels were well above those normally found among women who smoke.

The other question that has remained unanswered until recently is whether the offspring of mothers who smoke in pregnancy show any evidence of intellectual handicap. There appears to be some conflict of opinion on this point. R. R. Beard and N. Grandstaff[17] showed that even with a low concentration of carboxyhaemoglobin the hearing was affected, while Cole and colleagues[11] cite an unpublished observation from the Perinatal Survey that at 7 years of age the children of mothers who smoked showed some intellectual impairment. However, Janet B. Hardy and D. D. Mellits,[18] in a careful study from the U.S.A., could find no evidence of physical or mental handicap at 7 years of age, though they did observe a progressive increase in neonatal death among infants of mothers who smoked more than 20 cigarettes a day.

If these hazards to the fetus are to be avoided, real efforts should be made to educate women on the dangers of smoking, so that at least if they cannot be dissuaded from smoking at other times they will be prepared to give it up as soon as they become pregnant.

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Road Accidents Epidemic

The increase in road accidents in Europe in the past decade has been such that in most technically developed countries they now account for a third to a half of all male deaths in the age group 15-24. The number of drivers under the age of 25 injured in road accidents in the United Kingdom between 1960 and 1970 increased more than three-fold.[1] If this situation had been caused by poliomyelitis or some other disease traditionally associated in the popular imagination with public health the term "epidemic" would be freely used and the alarm bells would be ringing. But they are not.

A report[2] recently issued by the European Conference of Ministers of Transport makes it possible to compare, within limits, the incidence of deaths and injury on the roads in different European countries. It is based on a special survey and it supplements the very inadequate information to be obtained from official returns of road accidents. The wide variety of criteria whereby the different countries record such information renders comparison difficult, but trends occurring over a decade can yield useful information. It is interesting to note that the U.K. comes out well in comparison with other European countries, with the lowest overall increase in road accident fatalities (9%) from 1961 to 1970. It also has the best record for pedestrians, with only an 8% increase. The increase in fatalities among car users in the U.K. was a staggering 86%, yet even that is lower than in any European country for which a valid comparison is possible. A substantial part of this increase must be due to collisions between cars and heavy lorries. W. Gissane and J. Bull[3] have recently analysed the great and growing danger to car occupants in these circumstances.

For child pedestrians, however, we have the highest incidence of casualties in Europe in proportion to the population at risk up to the age of 5 as well as between the ages of 10 and 14. Only Germany has a worse record for the age group 6-9 years, and that only marginally. It is a cause for concern that we should have proportionately four times as many child pedestrian casualties under the age of 6 as does France, and more than three times as many in the age group 10-14. In comparison with the other European countries our problems are shown to be far more concentrated in built up areas, where 56% of our road deaths and 73% of our casualties occur. The risk is particularly high in the older cities where density of population and volume of traffic are high, and income and social class are low.[4] It may also be significant that the U.K. is the only technically developed country in Europe where pedestrians are allowed to cross the road whenever, wherever, and however they like.

The report is cautious, with good reason, about the causes of road accidents. It lists as the main "presumed causes" failure to give way, unsafe speed, pedestrians at fault, drunken driving, and unlawful overtaking. The experts who drew up the report unanimously agreed that unsafe speed is the principal "behavioural" cause, and the relatively low figure returned for drunken driving by most countries is dismissed as being "not in line with the facts," a criticism which is almost certainly true of the major wine-producing countries, most of which have not yet introduced an effective statutory blood alcohol level.

The failure to control the rising mortality and morbidity from road accidents should be regarded by public health authorities as a major epidemiological challenge. The case control surveys on which the alcohol provisions in the Road Safety Act 1967 were based provide a good example of the effectiveness of modern epidemiological techniques in road accident prevention, and it is unfortunate that the role of public health authorities has been limited to dealing with the consequences of road accidents instead of their prevention. The demarcation agreement which applies in most European countries, whereby transport authorities deal with prevention, may be convenient for administrative purposes, but it should be re-examined in the light of the facts now before us. Both the annual report of our Department of Health and Social Services and that of its Chief Medical Officer on "The State of the Public Health" ignore the prevention of road accidents, and the Transport and Road Research Laboratory assigns a relatively low priority to studies on the behaviour of road users while spending large sums of money on developing sophisticated techniques of road transport

which, in the distant future, could eliminate the need for the driver to exercise any judgement. The conventional motor car will be with us at least until the end of the century and the vast numbers of persons who will be killed or permanently incapacitated can be confidently predicted—unless a new approach is made to the problem.

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3 Gissane, W., and Bull, J., *British Medical Journal*, 1973, **1**, 67.
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Cannabis Debate Continued

Owing to its widespread illicit use an objective assessment of the type and degree of risk inherent in taking cannabis is of great importance. A report by H. Kolansky and W. T. Moore[1] provides new material for this debate. They present 13 detailed case histories of adults aged between 20 and 41 whom they had seen in the course of general psychiatric practice. These patients had all been smoking cannabis fairly heavily over a longish period. The clinical picture was of a rather stereotyped pattern of symptoms, which included headache, reversal of sleep rhythm, difficulties in recent memory, loss of interest in life goals, and what the authors describe as "mental and physical sluggishness." That the symptoms began after the patients started the heavy cannabis use and then tended to fade out within 3 to 24 months of its cessation is put forward as prima facie evidence of a causal relationship. These same authors had previously come to a similar conclusion on the basis of 38 case studies of adolescent cannabis takers.[2]

The cannabis debate has at times been a little too hotly partisan, with the contestants twisting or selecting the evidence to support extreme views. It is much to be hoped, therefore, that Kolansky and Moore's interesting clinical observations will receive thorough consideration. Careful clinical documentation of a short series of cases is not the end point of a resarch process, but it is often the valuable first step to a definitive investigation, though the interpretation of the findings deserves some thought. It should particularly be noted that Kolansky and Moore's patients were only from that segment of cannabis users who find themselves in trouble. The authors recorded clinical impressions rather than the results of psychometric tests. And some of the social decompensation which is prominent in the case schedules may reflect the patient's purposeful rejection of social convention, with cannabis as the symptom rather than the cause of the rejection. Kolansky and Moore's suggestion that some of their patients suffered actual brain damage rather than physiological disturbance would find support in views expressed by A. M. G. Campbell and his colleagues.[3] They reported on a series of 10 cannabis smokers whose air encephalograms were interpreted as showing ventricular atrophy. A lively correspondence was sparked off, in which Professor James Bull[4] challenged the radiological evidence, Dr. D. J. Fink[5] questioned the basis of the research design, and the authors made a reasoned reply.[6] And, as with the report by Kolansky and Moore, an open verdict would again seem to be the only fair reading.

These two recent studies have to be viewed in the context of many reports and rumours that cannabis is a hazard to health. This literature has been usefully reviewed in a World Health Organization publication.[7] The evidence on which some suppositions have been based has on close scrutiny proved to be rather insubstantial, yet there are hints that do deserve to be taken seriously. Possible effects of cannabis on time perception and on car driving have been briefly discussed in these columns.[8] A recent report[9] suggests that cannabis may cause hormonal disturbances leading to gynaecomastia. This is perhaps a relatively trivial complication, but it nonetheless suggests we are dealing with a powerful and little understood chemical.

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3 Campbell, A. M. G., Evans, M., Thomson, J. L. G., and Williams, M. J., *Lancet*, 1971, **2**, 1219.
4 Bull, J, *Lancet*, 1971, **2**, 1420.
5 Fink, D. J., *Lancet*, 1972, **1**, 143.
6 Campbell, A. M. G., Thomson, J. L. G., Evans, M., and Williams, M. J., *Lancet*, 1972, **1**, 202.
7 World Health Organization, *The Use of Cannabis*, 1971, Technical Report Series No. 478.
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Treatment with Calcitonin

Though there is still uncertainty about the physiological role of calcitonin,[1] it has several important diagnostic and therapeutic uses. The finding of increased circulating levels of calcitonin is helpful in confirming a diagnosis of medullary carcinoma of the thyroid—a tumour of the parafollicular or C-cells, which normally produce this hormone.[2] It is sometimes necessary to stimulate calcitonin secretion by an infusion of calcium to produce detectable levels, but the sensitivity of the radioimmunoassay has been improved recently. Raised levels of calcitonin are also of value in predicting recurrence of the tumour.

Calcitonin is helpful in the treatment of Paget's disease of bone[3] and of hypercalcaemia.[4] Features of Paget's disease are bone deformity and pain, rapid bone turnover, an increase in serum alkaline phosphatase levels, and excessive excretion of hydroxyproline in the urine. Administration of porcine,[5] salmon,[6] or human calcitonin[4] has abolished the pain and reduced the skin temperature over the affected bones. This treatment reduces the number of osteoclasts. Biochemical indices of bone turnover also improve. The serum alkaline phosphatase and urinary hydroxyproline levels fall, and bone takes up less calcium. Calcitonin must be administered parenterally, but the optimal dose schedule for each preparation has yet to be established. For human calcitonin it probably lies between 0·5 mg daily as a single injection (equivalent to 50 Medical Research Council units) and the same dose once a week.[7] The commercially available porcine calcitonin should be given initially in a dosage of 0·5—2·0 M.R.C. units per kg body weight per day divided in two doses. Salmon calcitonin, which has a longer biological half-life, is effective in a single daily dose of about 50 M.R.C. units.[6] Long-term therapy with calcitonin poses the problem of antibody formation. So far none of the patients who have received 1 mg of human calcitonin daily for 4-18 months developed antibodies,[7] whereas several of