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.


1 Department of the Environment, *Road Accidents 1970*, Table 6. London, H.M.S.O., 1972.
2 European Conference of Ministers of Transport, *Fourth Report on Trends in Road Accidents* (CM(72)10), 2 rue André Pascal, 75 Paris 16e. 1972.
3 Gissane, W., and Bull, J., *British Medical Journal*, 1973, **1**, 67.
4 Clayton, A. B., *Health Bulletin*, 1972, **30**, 277.


# 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.


1 Kolansky, H., and Moore, W. T., *Journal of the American Medical Association*, 1972, **222**, 1.
2 Kolansky, H., and Moore, W. T., *Journal of the American Medical Association*, 1971, **216**, 486.
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.
8 *British Medical Journal*, 1971, **2**, 293.
9 Harmon, J., and Aliapoulios, M. A., *New England Journal of Medicine*, 1972, **287**, 936.


# 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