Clonazepam: effective treatment for restless legs syndrome in uraemia

SIR,—We read with interest the report by Dr D J Read and others (3 October, p 885) concerning the therapeutic efficacy of clonazepam in uraemic patients suffering from the restless legs syndrome.

We have recently completed a survey of restless legs syndrome in 54 surgical outpatients. Assessment involved the administration of a semi-structured questionnaire, designed to elicit the subjective symptoms of the condition, as described by Ekbom.[1] In addition, a brief neurological examination and an assessment of abnormal involuntary movements were carried out. Eight (15%) of these patients fulfilled the criteria for the restless legs syndrome—that is, unpleasant sensations in the legs associated with inability to keep the legs still. This experience was invariably worse when they were sitting or lying down in the evening. A further seven (13%) patients complained of non-specific restlessness; in most cases this appeared to be related to anxiety. The onset of symptoms was not related to surgery in any of the restless patients. None of the patients seen was taking drugs which can cause akathisia and none was known to be suffering from any physical condition, including renal disease, identified as being associated with Ekbom's syndrome.

Thus, applying relatively stringent diagnostic criteria, we found a prevalence rate of 15% for the idiopathic restless legs syndrome. This finding challenges the statement of Dr Read and his colleagues, based on their estimate of a 15-20% occurrence, that uraemia is the most common cause of the restless legs syndrome. The possibility arises that the exacerbation of the syndrome observed with uraemic patients receiving dialysis may be partly accounted for by increased complaints and distress in patients with the idiopathic condition when required to lie relatively still for long periods. The authors note that clonazepam is also beneficial in the treatment of uraemic myoclonus. As myoclonus is a well-described, associated phenomenon in the restless legs syndrome,[2,3] it would have been of interest to know whether any of the patients in their study had also exhibited myoclonus.

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[1] Ekbom KA. *Neurology (Minneap)* 1960;**10**:868-73.
[2] Behrman S. *Br Med J* 1958;i:1454-7.
[3] Hussey HH. *JAMA* 1970;**235**:2224.

**We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—We know of no accurate figures for the prevalence of Ekbom's syndrome in the general population but is it generally regarded as being a rare condition even in busy neurological clinics. We were very surprised that in a small sample of a relatively selected surgical outpatient population the incidence of this syndrome was found to be as high as 15%. This may, of course, be because symptoms were deliberately sought rather than spontaneously volunteered. In this connection it would be helpful to know how brief the "brief neurological examination" was and what active steps (for example, measurement of serum iron and folate levels) were taken to establish that none of these patients was in fact suffering from any condition known to be associated with Ekbom's syndrome.

Since September 1978 all new patients attending our medical outpatient department have filled in a detailed symptom questionnaire which includes questions on both restlessness and pain in the legs. Of the 452 people who filled in questionnaires, fewer than 20 complained of restlessness, which on detailed questioning turned out to be very non-specific and not characteristic of Ekbom's syndrome; these symptoms may have been related to anxiety. These findings are in keeping with the generally held view that Ekbom's syndrome is a rarity. We should also like to point out that in all our renal patients the symptoms of restless legs were complained of spontaneously and were always of sufficient severity to disrupt normal life. Two of our patients found it necessary to sleep apart from their spouses because of the sleeplessness induced in the partner. The syndrome was more troublesome at night and did not occur only when patients resumed dialysis. Predialysis patients constituted five of our fifteen cases. It would therefore be untrue to say that this syndrome appeared only in those "required to lie relatively still for long periods." Lastly, there was no evidence of myoclonus in any of our patients.

On the basis of our experience we are quite unable to agree with Drs Braude and Barnes that their small survey in any way constitutes a challenge to the assertion that uraemia is the commonest cause of Ekbom's syndrome.

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Alcohol in the Third World

SIR,—Dr Richard Smith in his article on alcohol in the Third World (16 January, p 182) gives little information about Africa. From my experience in a general hospital in Zululand, South Africa, I have no doubt that excessive drinking has assumed epidemic proportions in the indigenous population.

An analysis of 1500 consecutive admissions to the hospital during six months in 1979 showed that 17% of patients were admitted as a result of deliberately inflicted injury. This was the most common single reason for admission, commoner than such "traditional" Third World problems as diarrhoea, malnutrition, or pneumonia. The majority of these patients were injured on Friday and Saturday nights and in most cases either they or their assailants exhibited clinical evidence of drunkenness.

Subsequently the Synod of the Anglican Church in Zululand considered the problem to be so serious that it passed a resolution calling on the homeland government to take urgent preventive action. More recently I wrote to six other hospitals in the area in an attempt to place an alcoholic member of staff in an environment away from the temptations of drinking friends, but they all replied that alcoholism in their own locality was too serious to allow them to help.

Comments I have had from doctors working in other countries in southern Africa, together with my own observations, suggest that despite any measures which may have been taken by governments alcohol abuse is already producing widespread damage to whole communities in the subcontinent.

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ABC of Alcohol

SIR,—We would like to add to the list of clinical manifestations of alcoholism given by Dr A Paton and others (12 December, p 1594) the thrombopathic haemorrhagic syndrome that is sometimes seen in heavy drinkers. Haemorrhagic manifestations of the alcoholic are traditionally thought to be secondary to liver cirrhosis alone. Alcohol has been reported, however, as a cause of thrombocytopenia and platelet dysfunction, presumably through a direct toxic effect on the bone marrow.[1]

We have studied the platelet count, Ivy bleeding time, and aggregation in response to adenine diphosphate (ADP), epinephrine, and collagen of 65 heavy drinkers in order to assess the frequency, importance, and clinical manifestations of thrombocytopenia and platelet dysfunction in alcoholism. The Ivy bleeding time and platelet count were within normal limits in most cases while platelet aggregation in response to at least one of the agents tested was impaired in about 50% of cases. Studies performed two weeks after alcohol withdrawal showed that the mean Ivy bleeding time had significantly shortened, the mean platelet count had increased (rebound thrombocytosis has been reported in some cases),[2] and the results of the platelet aggregation tests had become normal, an unusual hyperaggregability having been detected in one patient.

Significant bleeding manifestations were observed in only four patients, who presented on admission prominent ecchymoses and petechiae as well as mucosal bleeding (epistaxis and gingivorrhagias). Two of them had compensated liver cirrhosis. A high Ivy bleeding time, moderate-to-severe thrombocytopenia, and severe platelet aggregation impairment in the cases where the platelet count allowed us to study it were the prominent laboratory features in these cases—the previous administration of antiaggregating and thrombocytopenia-inducing drugs having been excluded. Results of blood clotting tests and serum folic acid were within normal ranges in all of them. Two weeks after alcohol withdrawal, the clinical signs of bleeding had completely resolved and the Ivy bleeding time, the platelet count, and the platelet aggregation tests were normal.

Our study provides evidence that severe bleeding manifestations due to quantitative and qualitative platelet defects may be a rare but well-defined clinical symptom of alcoholism and that qualitative defects without apparent clinical significance are a common finding in heaving-drinking alcoholics. These qualitative defects may account, at least in part, for the reported protection given by alcohol against coronary heart disease.[3]

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[1] Cowan DH. *Semin Hematol* 1980;**17**:137-47.
[2] Haselager EM, Vreeken J. *Lancet* 1977;i:774-5.
[3] Klatsky AL, Friedman GD, Siegelaub AB. *Ann Intern Med* 1981;**95**:139-45.

SIR,—The authors of "ABC of Alcohol: Nature of the problem" (14 November, p 1318) are to be congratulated on publicising the enormity of this problem, which is currently