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MEDICAL PRACTICE

NEWS AND NOTES

SUPPLEMENT

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CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

The Savage case

SIR,—Having had the opportunity of reading weekly instalments in the *BMJ* of the progress of the Savage case—an unusual opportunity following the decision of the chairman of the inquiry panel that the proceedings would take place in public—I am concerned at the exposure given to certain innocent parties to the inquiry. Other consultants than Mrs Savage herself and junior doctors who worked for her have been openly criticised during the course of the inquiry. I accept that a formal procedure, such as a (61)112 inquiry, was bound to raise sensitive issues involving other doctors, and I also accept that the *BMJ* had a responsibility to cover the case once the decision had been made that it would not be held behind closed doors. What I cannot accept is that it did any good to anyone to hold it in public, including the patients referred to, Mrs Savage herself, and certainly the junior doctors whom I represent.

Whereas the *BMJ* covered the facts of the case accurately, the popular press was naturally more sensational in its reporting, and the distress and anger this generated among the doctors passively involved were utterly unjustified. I am, therefore, writing to urge that the BMA should use its influence to ensure that such inquiries are held in private in future. In seeking this action, I emphasise that nothing should be hidden from the inquiry panel. But I can see nothing to be gained by allowing the public to gaze on the proceedings. Indeed, events on this occasion have shown that a lot—in the way of public confidence in the service offered and in the reputation of doctors not the subject of inquiry—can be lost.

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Chairman

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Cognitive function during hypoglycaemia in type I diabetes mellitus

SIR,—The interesting study reported by Dr S Pramming and his colleagues (8 March, p 647) showing a deterioration in cognitive function during hypoglycaemia in insulin dependent diabetics exposes a further manifestation of this common side effect of insulin therapy. They showed that cerebral dysfunction can occur before the blood glucose falls to a concentration that usually would trigger the counter-regulatory hormonal responses and alert the patient to the incipient danger of hypoglycaemia.[1] This study raises several interesting questions.

Awareness of impending hypoglycaemia is associated with activation of the sympathoadrenal system and the release of catecholamines,[2] which cause the typical autonomic symptoms of acute hypoglycaemia. Altered symptoms or complete loss of awareness of hypoglycaemia often develops in patients with longstanding diabetes, and, although this may be associated with autonomic neuropathy,[3] it also occurs in patients who have normal autonomic neural function. It would be of interest to know whether the patients in the authors' study who either failed to recognise hypoglycaemia with a blood glucose value below 2 mmol/l or were said to show no clinical signs of hypoglycaemia had a history of hypoglycaemic unawareness or had overt autonomic dysfunction. Could a direct correlation be identified between the degree of disability or neuropsychomotor testing and the duration of the diabetes, or was the cerebral dysfunction related to the magnitude of the catecholamine responses to hypoglycaemia, if these were measured?

The authors did not comment on the quality of the glycaemic control of their patients at the time of study, but quote mean HbA_1 values of 8·5% and 8·7% for both groups of subjects. This implies that their overall glycaemic control was extremely good. Insulin dependent diabetics treated with intensive insulin regimens for four to eight months have been shown to have reduced responses of counter-regulatory hormones (adrenaline, cortisol, and growth hormone) to acute hypoglycaemia.[4] This suggests that "tight" glycaemic control may lower the threshold for activation of counter-regulatory mechanisms and introduces the concept that antecedent glycaemia can alter central perception of changes in blood glucose concentration. Is it possible therefore that the prevailing level of glycaemia in the patients of Dr Pramming and others may have influenced the threshold for impairment of cognitive function? Maybe neural activity adapts to chronic neuroglycopenia in the insulin dependent diabetic? It would have been useful to compare the glycaemic threshold at which deterioration of cognitive function starts in the diabetic groups with that in a group of non-diabetic controls.

Their observation that executive functions and decision making are seriously impaired at subnormal blood glucose values (<3 mmol/l) while sparing psychomotor activity of a repetitive nature is important. This has particular relevance to the relation between automatism and hypoglycaemia with the attendant legal implications.[5] In a survey in Edinburgh, several diabetic drivers remarked that they were aware of hypoglycaemic symptoms while driving but were unable to make themselves stop.[6] They described a compulsion to keep going, although they knew that this was both irrational and dangerous. Moderate neuroglycopenia does not therefore interrupt psychomotor activity in this situation but does reduce attention and reaction times, as Dr Pramming and others have shown. The danger of this state is the inability to make a positive decision to stop driving despite recognising the inherent risks.

Dr Pramming and his colleagues have explored a twilight zone of neuroglycopenia between the normal range of blood glucose concentrations and overt hypoglycaemia, which impairs cerebral function but does not necessarily provoke the autonomic warning symptoms. This provides a