This method uses an infrared plethysmograph observing any changes in the unstressed volume of the finger arteries, mounted in an inflatable cuff providing at each instant a counter pressure sufficient to just counterbalance intraarterial pressure. Since this requires dynamic control of cuff pressure it is performed by a wideband servo system which uses the plethysmogram as one input. The other input is the proper unstressed finger arterial volume level which is determined automatically by an expert system programmed on a microcomputer built into FIN.A.PRES.

With respect to the transcranial Doppler ultrasonic recording of cerebral blood flow this seems to us an attractive method for studying changes in cerebral blood flow, whereby we leave open the question of which circulation is more peripheral, the one in the finger or the one in the cerebrum. However, the discordance between central blood flow and systemic blood pressure, in particular in patients with autonomic failure has been well noted and has been attributed to a change in autoregulation of the cerebral circulation. In the light of these facts, the statement of Drs Reinecke and Langohr that “measurements of cerebral artery flow velocity allow a better visualisation of autonomic reflexes in a more central part of the circulation...” cannot be held unmodified. Their fine technique may, however, prove a promising clinical tool in studying the instantaneous and varying discordance between flow and pressure in different parts of the circulation in combination with the equally non-invasive registration of continuous blood pressure in the finger.

Recurrence, hyponatraemia and neuroleptic malignant syndrome

Sir: We found the case-report by Gibb et al describing recurrent neuroleptic malignant syndrome in association with hyponatraemia very interesting. However, we do not agree with the authors on several points. The authors maintain that recurrence of neuroleptic malignant syndrome has not been reported despite reintroduction of neuroleptic therapy on frequent occasions after single episodes. A careful review of published literature gives a totally different picture. Recurrence was noticed in six out of eight cases when rechallenge was done with the same drug. Rechallenge with drugs of the same milligram potency as the offender (6 cases) resulted five times in recurrent neuroleptic malignant syndrome with two fatalities. On the other hand, rechallenge with lower potency antipsychotics, particularly thioridazine, was safe in nine out of 10 cases. If the rechallenge is attempted with the same drug but at a slower loading rate, there may not be any recurrence. Relapse has also been observed in cases where the treatment was performed prematurely. Besides this we wonder whether the diagnosis of neuroleptic malignant syndrome was made prospectively or retrospectively. There are several indicators which suggest that the diagnosis was retrospective. The clinical details provided in the first presumed episode of neuroleptic malignant syndrome are insufficient to make a diagnosis of neuroleptic malignant syndrome. No alteration in consciousness was noted in the first “episode”. Altered consciousness is essential for diagnosing neuroleptic malignant syndrome. The authors made no comments regarding presence or absence of autonomic dysfunction in the first “episode”. We have no information about serum CK levels in the first episode. No muscle biopsy findings are available for either of the episodes. Moreover, in both the “episodes” the patient was not given any specific treatment for neuroleptic malignant syndrome. One of the problems which has been faced in analysis of cases of neuroleptic malignant syndrome is failure on the part of the authors to provide a complete clinical description. It has been proposed that the published reports of neuroleptic malignant syndrome should not be accepted uncritically.

We feel that the authors should have provided more clinical details before making a diagnosis of “recurrent” neuroleptic malignant syndrome if such a diagnosis was made prospectively.

ADITYANJEE

References


Gibb et al reply:

Dr Adityanjee has challenged our statement that recurrent neuroleptic malignant syndrome (NMS) has not been described previously, by citing three papers that apparently conflict with this. One paper discusses the possibility of recurrent NMS without referring to such a case, while the other two do not mention in their reviews of the literature the complicated cases in which entirely separate episodes of NMS are described. The cases referred to are as follows. One case was an acute dystonic reaction, drugs were stopped and no rechallenge done. One case was an NMS-like reaction following withdrawal of tetrabenzine, but there was no recurrence when haloperidol was started on the 42nd day. In five cases NMS was provoked before full recovery, when drugs were restarted before the tenth day. Furthermore two of these cases may not have had recrudescence NMS for there was little or no fever or record of rhab-