CORRESPONDENCE

PENTOLINIUM IN POSTOPERATIVE HYPERTENSION

Sir,—I was interested in the report by Jones, Hanler and Knight (1981) on the use of pentolinium to control hypertension after operation. Two of the patients described had undergone surgery which could affect the blood supply of the brain and it is on these that I would like to comment. Carotid artery stenosis is often part of bilateral and disseminated vascular disease. Therefore an adequate cerebral blood flow may require a high perfusion pressure, and any reduction in arterial pressure to "normal" values may lead to cerebral ischaemia and thrombosis of the carotid artery. I would agree with control of gross hypertension, but strongly believe that a short-acting drug should be used so that its effects may rapidly reverse in the event of the development of neurological sequelae.

Considering the problems of aneurysm surgery, vasospasm occurs commonly after operation (Fernside and Adams, 1978) and it has reported that there is a steady decrease in cerebral blood flow after subarachnoid haemorrhage (Meyer, Neil-Dwyer and Lowe, 1981). This may result in cerebral ischaemia which will induce reflex hypertension, the effectiveness of which has been indicated by the improvement in cerebral state by induced hypertension in patients with vasospasm (Brown, Hanlon and Mullan, 1978). I accept that control of severe hypertension would benefit the heart, and reduce the formation of cerebral oedema, but unless it is possible to monitor cerebral oxygenation, in my opinion it is wise to reduce the arterial pressure at all, particularly with a drug which is long acting.

Being a ganglion blocker, one of the side-effects of pentolinium is pupillary dilatation as a result of parasympathetic ganglion blockade (Wade, 1977). One of the principal signs used in the period after operation for monitoring neurosurgical patients is pupil size. Therefore, drug-induced pupillary dilatation in a patient whose cerebral circulation may be jeopardized by the reduction in arterial pressure could make management impossible.

In conclusion, it is potentially dangerous to reduce the arterial pressure in patients with cerebrovascular disease in whom there is no certainty about the arterial pressure required to maintain an adequate blood flow, particularly in those undergoing surgery on intracerebral vessels.

The measurement of central conduction time may allow the onset of cerebral ischaemia to be predicted (Symon et al., 1979). If this technique becomes fully established, only then may it be possible to reduce an increased arterial pressure with a short-acting agent, thereby benefiting the heart whilst ensuring that the brain was not damaged.

F. WALTERS
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REFERENCES

Sir,—Dr Walters raises a number of points, but principally questions the advisability of reducing arterial pressure in patients after carotid artery or aneurysm surgery.

As Towne and Bernhard (1980) have reported, severe postoperative hypertension following carotid endarterectomy is a common and serious problem, associated with an increased mortality rate and increased occurrence of neurological deficit. This hypertension has been ascribed to baroreceptor dysfunction (Bove et al., 1979). Dr Walters agrees that carotid artery stenosis is often part of bilateral and disseminated vascular disease. In a review of anaesthesia for carotid endarterectomy, Keats (1981) draws attention to the fact that approximately half the mortality from the operation is the result of heart disease, particularly myocardial infarction. Earlier work in the development of carotid endarterectomy focused, as Dr Walters does, on protecting the brain from ischaemic damage, but as Riles, Kopelman and Imperato (1979) have emphasized, this may be at the expense of inducing myocardial ischaemia. Keats' review is a critical appraisal of methods of cerebral protection, and gives a balanced account of the benefits of these and the risks they may involve by causing myocardial ischaemia. He concludes that "Anesthetic management should be directed toward avoidance of both hypotension and hypertension, maintenance of sleeping blood pressure, avoidance of tachycardia and bradycardia and utilizing anesthetic agents and adjuvants that would be selected if one assumed all patients undergoing carotid endarterectomy had symptomatic coronary artery disease." This point of view, in contrast to that of Dr Walters, can no longer be considered controversial. Frost (1981), reviewing the care of patients after carotid endarterectomy states "The major cause of serious postoperative morbidity in patients undergoing carotid endarterectomy is myocardial infarction. In addition, hypertension may increase capillary hydrostatic pressure, especially in ischaemic areas of the brain, leading to protein leak, oedema or haemorrhagic infarction. Thus maintenance of preoperative baseline arterial pressure after surgery is critical. In our original communication the patient's arterial pressure after carotid endarterectomy was 210/120 mm Hg. Dr Walters quibbles with the use of pentolinium, stating that he believes strongly that a short-acting drug should be used so that its effects may rapidly reverse in the event of the development of neurological sequelae. Although I realize he is making a general observation, he has also completely missed the point of our report. The patient's arterial pressure was
210/120 mm Hg during the infusion of sodium nitroprusside (SNP) 8 μg kg⁻¹ min⁻¹, used as the first-choice drug to control the postoperative hypertension. The reasons for the resistance to SNP (sympathetic over activity) and the ability of pentolinium to reduce the arterial pressure smoothly (in the patient reported, to 150/85 mm Hg) and interrupt the pathways leading to sympathetic over activity, are the basis of our original communication.

Dr Walters proceeds to consider the problems of aneurysm surgery, and questions its advisability of reducing arterial pressure in patients who may develop the common complication of cerebral arterial spasm. In support of his opinion that arterial pressure should not only be maintained, but perhaps increased, he cites the work of Brown, Hanlon and Mullan (1978). Unlike the situation after carotid artery surgery, the position regarding the control of arterial pressure after aneurysm surgery is more complex. Walter, Neil-Dwyer and Cruickshank (1982) have recently reported the beneficial effects of adrenergic blockade in patients with subarachnoid haemorrhage. Patients received standard management or standard management with alpha- and beta-adrenergic blockade. During the first month, the group treated with adrenergic blockade suffered fewer episodes of clinical deterioration consistent with cerebral arterial spasm and the outcome subsequent to operation was better. In this group, adrenergic blockade was continued after operation; again, among possible mechanisms for the improved outcome, the authors cite the prevention of myocardial infarction. Adrenergic blockade was investigated because of the possible aetiological role played by catecholamines in causing spasm. It is of interest to speculate on a possible role for pentolinium in this respect, as our report has shown how this agent may be used to decrease sympathetic tone.

Mullan, Hanlon and Brown (1978) have also reported on the management of berry aneurysms using induced hypotension with ganglion blockade or SNP. Another report by the same investigators (Brown, Hanlon and Mullan, 1978) cited by Dr Walters details four patients in whom an increase in arterial pressure, in the presence of arterial spasm, was associated with clinical improvement. However, these authors do state that the use of hypertensive treatment is unusual. Nevertheless, it does seem likely that, in the presence of established arterial spasm, it would be unwise to reduce the arterial pressure. As in many circumstances in anaesthesia, each case must be judged on its merits with an understanding of the pathophysiology involved. It is possible that control of sympathetic discharge and arterial pressure after operation will reduce the frequency of arterial spasm. In our original communication the patient who had undergone clipping of a cerebral arterial aneurysm was fully conscious and alert with no evidence of arterial spasm. After full consultation with the neurosurgeons involved it was decided that a reduction of arterial pressure would be of benefit.

Finally, Dr Walters makes two specific points about the use of pentolinium. He usefully emphasizes the fact that pentolinium will cause pupil dilatation and that in certain circumstances this may complicate neurological assessment. However, the patients in our report were alert and awake, making unnecessary the use of pupil size to monitor neurological state, although in other circumstances this may be an important consideration. He is also worried about the duration of action of pentolinium. It has been my experience that the cardiovascular effects of the drug can be simply and rapidly reversed by an increase in intravascular volume, should this be found necessary.

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REFERENCES


POSTOPERATIVE BUPRENORPHINE

Sir,—In a recent paper, Watson and her colleagues studied the duration of analgesia following two different i.v. doses of buprenorphine in the period immediately after operation.

This was done by means of a demand-analgesia technique, in which diamorphine was given “on demand” from the 3rd to 12th hours after buprenorphine administration, and the cumulative dose requirements recorded. The results for each group of patients were expressed by a cumulative mean dose plot and analysed by means of linear regression.

While there is no doubt that the cumulative dose plots for the two patient groups are quite different, it is equally clear that the method of analysis is inappropriate.

Linear regression may be applied to a plot of y upon x under defined conditions. First, each value of y (which may be the mean of several estimates) must be independent from those determined at other x-loci. Second, the error variance must not differ significantly for all values of y.

In this case, neither criterion is satisfied; therefore the analysis is of limited value, since the regression coefficient cannot be compared statistically with that derived from a second set of data.

Since the cumulative doses for two groups of patients at any selected time (say 540 min) may be compared very simply by means of Student’s t test, the authors do their data an injustice by the incomprehending use of a more complex method.

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