CORRESPONDENCE

PENTOLINIUM IN POSTOPERATIVE HYPERTENSION

Sir,—I was interested in the report by Jones, Hantler 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 unwise 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
Bristol

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 Imparato (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 “Anaesthetic 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 mmHg. 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 be rapidly reversed 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...