authors,^{5, 6} and our findings therefore support the use of a conservative vitamin K₁ dosage regimen. With the concentrate the correction was more controlled in preventing overcorrection but in two out of 11 instances they were not sufficiently sustained, and the patients needed further infusions at 24 hours.

Similarly the partial thromboplastin times indicated an overcorrection with vitamin K₁. Values in seven out of nine patients were restored to normal, but only after two of the 11 concentrate infusions did this occur. The findings with the prothrombin time and partial thromboplastin time tests were mirrored in the specific clotting assays of II and X.

There was a strong contrast in factor VII assay results. With vitamin K₂ correction was detectable at two hours and maximal at 24 hours, eight of the nine patients being restored to 100% activity or above. There was, on the other hand, no rise two hours after the infusion of concentrate. At 24 hours there was some rise, due presumably simply to anticoagulant withdrawal, but in almost half the patients the level was still very low (1-7%). The precise clinical significance of the persistent depression of factor VII is not certain, the only analogues being congenital deficiency of factor VII, in which the danger level is considered to be 5-10%.⁷ On first principles, therefore, a concentrate containing factor VII seems to be preferable to a VII-poor preparation. It requires clinical confirmation that the concentrate we used will arrest bleeding at these low levels of factor VII activity.

Contrary to reports of the thrombogenic risk of some concentrates⁸⁻¹¹ there was no evidence to suggest DIC with use of the Prothromplex concentrate.

The hepatitis B risk with factor IX concentrates is established,¹² but the magnitude of the risk of non-B hepatitis with these products is not generally realised. Prince¹³ suggested that 20% of post-transfusion hepatitis was unrelated to hepatitis B virus. Thus, even if the risk of hepatitis B was eliminated there

would still be a considerable risk of hepatitis with these products. Even in this small series the possibility of transmission of non-B hepatitis in one patient by this product cannot be excluded.

Hence, this concentrate provides a more rapid and controlled but less sustained means of correcting oral anticoagulant overdose than vitamin K₁, which, even in a dose of 2.5 mg, always tends to overcorrect. Our results suggest that the risk of hepatitis may be significant despite careful screening for HBsAg in the donors, and therefore its use in oral anticoagulant reversal should be limited to correcting life-threatening haemorrhage.

We thank Dr K Anderle, Immuno Vienna, for generous supplies of Prothromplex; and Dr D M Jones and Dr J Craske, Public Health Laboratory, Withington Hospital, for hepatitis antigen studies. Technical help was provided by Mr S R Armitage, of the coagulation staff, Withington Hospital.

References

- ¹ Lane, J, Rizza, C R, and Snape, T J, *British Journal of Haematology*, 1975, **30**, 435.
- ² Green, G, *et al*, *Lancet*, 1975, **1**, 1311.
- ³ Dioguardi, N, and Mannucci, P M, *Lancet*, 1975, **2**, 188.
- ⁴ *Lancet*, 1975, **2**, 855.
- ⁵ Toohey, M, *British Medical Journal*, 1954, **2**, 1020.
- ⁶ Poller, L, *Theory and Practice of Anticoagulant Treatment*, p 55. Bristol, John Wright, 1962.
- ⁷ Rizza, C R, in, *Human Blood Coagulation Haemostasis and Thrombosis*, ed R Biggs, p 213. Oxford, Blackwell, 1972.
- ⁸ Kingdom, H S, *et al*, *Thrombosis et Diathesis Haemorrhagica*, 1975, **33**, 617.
- ⁹ Cash, J D, *et al*, *Thrombosis et Diathesis Haemorrhagica*, 1975, **33**, 632.
- ¹⁰ Kasper, C K, *Thrombosis et Diathesis Haemorrhagica*, 1975, **33**, 640.
- ¹¹ Preston, F E, *et al*, *Thrombosis et Diathesis Haemorrhagica*, 1975, **34**, 475.
- ¹² Roberts, H R, and Blatt, P M, *Thrombosis et Diathesis Haemorrhagica*, 1975, **33**, 610.
- ¹³ Prince, A M, *Post-transfusion Hepatitis: Aetiology and Prevention*. XIV Congress of the International Society of Blood Transfusion, Helsinki, 1975.

Headache after carotid endarterectomy

JOHN PEARCE

British Medical Journal, 1976, **2**, 85-86

Summary

Forty-eight hours after undergoing a successful right carotid endarectomy a patient complained of headache in and behind the right eye radiating to the temple and forehead. The onset of headache was sudden, and the pain was severe and throbbing. After three weeks of regular four- to eight-hour attacks each day the headaches gradually became less frequent. Two months after operation they had disappeared completely.

Headache as a complication of endarterectomy is rare, but typically it is vascular and subsides spontaneously in one to six months. If a predisposition to migraine were a precipitating factor many more cases would be expected. No possible explanation for headache after carotid prearterectomy can account adequately for its apparent rarity.

Introduction

In 1954 Eastcott *et al*¹ performed the first successful carotid endarterectomy for carotid stenosis. The operation has since been performed widely, although its indications remain somewhat debatable. The operative mortality rate is now about 1%.² Complications include postoperative thrombosis, dissection of the intimal flap, and infarction due to distal thrombosis, embolism, or hypotension. Haemorrhage into the ischaemic hemisphere may occur as a result of luxury perfusion.

Lawson² noted recently that 90% of patients with transient ischaemic attacks remained symptom free after endarterectomy. No patients were made worse by surgery and no additional neurological deficits were produced. Only two published reports^{3, 4} describe a striking symptom encountered during the postoperative period—intense localised vascular headache. No case has been reported from the UK to the best of my knowledge. In view of the manipulation of the common carotid artery, its adventitia, and the carotid sinus, it is perhaps surprising that symptoms directly attributable to the localised vascular trauma have been recorded so rarely.

Case report

A 53-year-old man presented with recurrent episodes of transient left-sided weakness present for two months. These were of sudden

onset affecting the face, arm, and leg, and on three occasions there was transient loss of vision in the right eye, described as a curtain passing across the field of vision from temporal to nasal side only to disappear quickly in the reverse direction during the course of two minutes. The attacks of left-sided weakness lasted from one to 15 minutes and were not accompanied by any disturbance of speech or sensation. Initially attacks occurred weekly, but during the three weeks before admission to hospital they were occurring two to five times a day. Neurological examination on admission to hospital showed no abnormal signs. There was a localised bruit over the right internal carotid artery, but this was not conducted to the cranium or orbits. Blood pressure was 135/85 mm Hg in both arms. Systemic investigations disclosed no factors contributory to his presumed carotid atherosclerosis.

Bilateral carotid arteriography showed a localised stenosis at the origin of the right internal carotid artery, reducing the lumen to an estimated 15% of normal. There were no complications of arteriography. Three days later a right carotid endarterectomy was performed, and an extensive plaque of atheromatous material with some fibrin and platelet-containing thrombus over the intimal surface was removed. Normal blood flow was established and no immediate complications developed.

Forty-eight hours after the operation the patient started to complain of headache. The onset was sudden and the pain was felt in and behind the right eye and radiating to the temple and forehead. The pain was extremely severe, "like someone thumping with a hammer inside my head," and was pulsatile and throbbing in nature. There was no associated blocking of the nose or redness of the eye, but the pain would occur in regular attacks lasting four to eight hours each day; after three weeks they gradually lessened in frequency. They were only partially relieved by simple analgesics and ergotamine suppositories. There was some associated nausea but no vomiting. In the first week he experienced transient left-sided paraesthesiae in the face and arm, and although these were associated with the early phase of the headache attack they seldom lasted longer than two or three minutes and were somewhat variable in their distribution.

Two months after surgery the headaches disappeared completely and he has had no further ischaemic symptoms.

Discussion

Among the many papers on carotid endarterectomy only two briefly mention headache as a complication.^{3,4} Toole *et al*³ referred to severe headache after endarterectomy of a stenosed artery and attributed it to vascular distension. Leviton *et al*⁴ give the only comprehensive description of the syndrome in a 61-year-old musician who developed very similar headaches after a right carotid endarterectomy. They refer to one other patient reported in "Headache Rounds" and emphasise the similarities of these two patients. Headache therefore seems to be a rare complication of carotid endarterectomy but one of considerable clinical interest.

Both the patients of Leviton *et al* had had minor vascular headaches of a migrainous type before surgery, but the patient reported here had no previous experience of migraine. A striking feature was the headache-free latent interval of 36 to 72 hours in the two other reported cases. The headaches started suddenly in each patient, were very severe, and were largely confined to the same side as the endarterectomy. The clinical attributes were typically those of vascular headache and in each case were accompanied by a transient cerebral dysfunction of the ipsilateral hemisphere strongly suggestive of transient ischaemia. In the patients of Leviton *et al* postoperative angiography at the time of severe headache showed a normal carotid arterial tree, so it seems unlikely that there was any recurrent thrombosis or

structural change in the lumen to account for the development of headache. In all the patients described the condition spontaneously subsided within one to six months.

The clinical features of this patient suggest that the headaches are basically vascular, being frontotemporal, limited to one side of the head, and throbbing or pulsatile. No specific trigger factors were identified in the present case, but alcohol and sunshine were incriminated in those of Leviton *et al*. A predisposition to migraine seems to be relevant in the earlier cases but not in the present one. Furthermore, if migraine were the basis of the headache, then the high general prevalence rate of migraine (about 20%) would lead one to expect this type of headache in many of this susceptible group purely on a statistical basis.

The explanation of the latent interval and the mechanism of this vascular headache remain obscure. The preoperative narrowing of the carotid artery followed by sudden disobliteration might certainly lead to a sudden distending force of the full arterial pressure on vessels previously protected by the stenosis. If this were the explanation one would expect the headache to develop immediately rather than after an interval. There is no proof of any protective spasm in the carotid tree in the immediate postoperative period that might theoretically explain the delay. Leviton *et al* discussed a disorder of autoregulation of cerebral blood flow. Autoregulation is the ability to maintain a relatively constant blood flow in the face of changing perfusion pressure.⁵ There is good evidence that autoregulation is disordered during the migraine prodrome.^{6,7} In the context of spontaneous migraine regional cerebral blood flow is considerably reduced and does not respond to increases in arterial PCO₂. In the headache phase the regional cerebral blood flow is increased and has been attributed to distension of normal vascular pathways and to the perfusion of collateral vessels. In the context of endarterectomy a reduction of perfusion pressure by the stenosed segment may be associated with regional vasodilation. The development of normal pressure after endarterectomy might lead to a distending force in a vascular bed dilated by such autoregulatory phenomena, resulting in reactive "luxury perfusion."

A more likely hypothesis is that the regulation of the regional cerebral blood flow is altered by manipulation with the carotid sinus at the bifurcation or by damage to the sympathetic chain in the carotid sheath. These factors would affect, respectively, the afferent and efferent sides of the autonomic innervation of the cranial vessels and in this way might lead to a temporary vascular disturbance, which would produce a vascular headache. This would subside spontaneously in a manner similar to that seen in the changes in the vasculature after sympathectomy. The variation in technical factors involved in the operation would seem insufficient to explain the rarity of this symptom, the mechanism of which remains incompletely understood.

References

- Eastcott, H H G, Pickering, G W, and Rob, C G, *Lancet*, 1954, **2**, 994.
- Lawson, L J, in *Strokes: Natural History, Pathology and Surgical Treatment*, ed E C Hutchinson, and E J Acheson. London, Saunders, 1975.
- Toole, J F, *et al*, *Stroke*, 1973, **4**, 273.
- Leviton, A, Caplan, L, and Salzman, E, *Headache*, 1975, **15**, 207.
- Betz, E, *Physiological Reviews*, 1972, **52**, 595.
- Skinhoj, E, and Paulson, O B, *British Medical Journal*, 1969, **3**, 569.
- Pearce, J, *Modern Topics in Migraine*. London, Heinemann, 1975.