
Just as neurologists were slow to appreciate the significance of atheroma in the cervical portion of the carotid artery for the development of vascular disease in the head so, too, the anatomical status of the vertebral artery in the neck has been neglected in the context of stroke. But now that the role of internal carotid artery occlusion in cerebral infarction is better recognized, and given the importance of collaterals between the anterior and posterior circulations, it is important to consider the condition of the cervical portion of the vertebral artery. Drs Hutchinson and Yates report clinical and pathological findings in 48 patients who died from various cerebrovascular lesions and in whom the carotid and vertebral arteries were injected post-mortem with gelatine soaked in radio-opaque dyes to allow radiological depiction and subsequent dissection. Their focus is on the state of the vertebral artery within the cervical canal. As their former Mancunian colleague Baron Stopford of Fallowfield (1888–1961) had found a generation earlier, the authors confirm that one vertebral artery or the other is almost invariably dominant (left 51%, right 41% and 8% equal in size). Within the canal, the medial wall of the vessel abuts the neurocentric joint and gives a posterior vascular supply to the emerging nerve root. Degenerative disease of the cervical spine will displace the artery forwards and laterally; this may produce a gentle curve in the trajectory of the vessel as it passes upwards, or render the vertebral artery distinctly serpiginous.

Atheroma of the vertebral arteries is common, and this has significance for the consequences of any concomitant carotid disease that may be present and for infarction of the brain stem and cerebellum. Nineteen of 48 patients have atheroma varying from localized plaque without stenosis to diffuse degeneration of the vessel. In 10, the vertebral artery shows >50% stenosis and, in 3, thrombus superimposed on plaque has totally occluded the vessel. Degeneration of the vessel is invariably due to atheroma, often with intramural haemorrhage, and not an unrelated vascular pathology. Atheroma does not preferentially localize to the part that curves around the axis and atlas, to the larger or smaller of the pair, or to those segments displaced by cervical spondylosis. Rather, it is most usually contiguous with disease of the subclavian extending a variable distance from the mouth of the vessel at its origin. Vertebral artery atheroma may occur in relative isolation or in the context of more generalized intracranial vascular disease.

The carotid artery is diseased, showing >50% stenosis, in 15 of 48 patients; of these, nine also have significant unilateral or bilateral vertebral disease, which is generally severe and with occlusion in two instances. In one patient with carotid occlusion, symptoms only became manifest when the thrombus extended into the middle cerebral artery indicating that collateral circulation through the intact carotid or vertebral arteries had adequately maintained blood supply to the hemispheres until that point. A second patient with an occluded carotid has an associated cerebellar infarction suggesting that the vertebral supply may not have been adequate (Fig. 1). The third patient, R.G. (aged 60 years), experienced transient numbness of either the left or right arm lasting a few minutes and with recovery, later associated with unexplained episodes of altered consciousness, for 18 months before he developed a sudden dense hemiplegia with sensory loss. Examination confirms these disabilities and shows a dense homonymous hemianopia, slow cerebration and reduced impulse of the carotid pulse on the right. Following a single convulsion, coma intervenes and death ensues soon after. Autopsy shows generalized severe atheroma, old infarctions in the right insula, and more recent infarctions of the parietal region on that side and of the left globus pallidus and internal capsule. The left internal carotid artery is occluded and the right contains recent thrombus that has advanced into the middle cerebral artery; the left vertebral is stenosed at the C4/5 level and the right at C5 as it passes through the axis. But the rest of the intracranial vasculature is remarkably free of atheroma. Struggling to reconcile the apparent distribution of RG’s vascular lesions with his prior symptoms—localized to structures at the junction between the territories of the carotid and vertebral circulations—the authors defer to Sir Charles Symonds (1890–1978) who proposed that episodic loss of function occurs not as a result of fresh ischaemia but through failure of compensatory mechanisms—neither blood flow, local pCO2 concentration nor changes in blood pressure but, rather (as they suggest) vascular spasm triggered by intramural haemorrhage in
but the vessel remains patent. There is posed thrombus; extensive disease is also present on the right atlas due to an atheromatous plaque, and occluded by superim- vertebral artery is markedly narrowed as it winds around the lesions of older age in both cerebellar hemispheres. The left recent right internal capsule and basal ganglia infarction with atheroma of the extracranial vessels (Fig. 2). The brain has a Autopsy confirms a recent myocardial infarction, with generalized atheroma from which he dies soon after admission to hospital. loss of consciousness lasting a few minutes attributed to complete iberia infarctions, usually bilat- } 

In four patients, cerebral and cerebellar infarctions, usually bilateral, coexist. J.P. (aged 77 years) has had nine episodes of sudden loss of consciousness lasting a few minutes attributed to complete heart block from which he dies soon after admission to hospital. Autopsy confirms a recent myocardial infarction, with generalized atheroma of the extracranial vessels (Fig. 2). The brain has a recent right internal capsule and basal ganglia infarction with lesions of older age in both cerebellar hemispheres. The left vertebral artery is markedly narrowed as it winds around the atlas due to an atheromatous plaque, and occluded by superimposed thrombus; extensive disease is also present on the right but the vessel remains patent. There is >50% narrowing of both carotids by atheromatous plaque with haemorrhage. S.W. (aged 56 years) is accidentally exposed to carbon monoxide after suffering a cerebral thrombosis that results in aphasia and facial weakness; 2 days later, she has a further episode with dense hemiplegia that proves fatal (Fig. 3). At autopsy, there is evidence for recent infarction in the territory of the left middle cerebral artery which is thrombosed; in addition, both cerebellar hemispheres are infarcted in the territory of the superior cerebellar arteries. The dominant left vertebral artery (four times larger than that on the right) is occluded by atheroma 2 cm from its subclavian origin but is otherwise reasonably healthy. The left internal carotid artery is occluded at its origin and the right is normal. E.C. (aged 55 years) underwent mitral valvotomy for severe stenosis but develops a dense right hemiplegia and occluded left iliac artery per-operatively from which she soon dies. At post-mortem there is a recent infarction in the left hemisphere close to the pre- and post-central gyri, and another recent lesion in the territory of the left superior cerebellar artery. Both common and internal carotids show extensive atheromatous narrowing; the right vertebral artery is reduced to a slit in the cervical portion and the left occluded by thrombus superimposed on atheroma in two places. L.A. (aged 73 years) develops sudden incoordination of all four limbs and ataxia; later she has headache and slurred speech and soon after admission, when the signs match her symptoms, she lapses into coma and dies. In addition to an old right hemisphere lesion, she has a recent infarction in the right parietal lobe, and in both cerebellar hemispheres in the watershed zone between the territories of the inferior and superior cerebellar arteries. Although the intracranial vessels are relatively normal, there is extensive narrowing of the extracranial portions of the common and internal carotids, and reduction to <25% of the normal lumen in the right dominant vertebral at the level of the axis but with less marked involvement on the left.

Drs Hutchinson and Yates wish to emphasize that the recent recognition of extracranial carotid disease rather than local vasospasm as a common cause of cerebral infarction is now matched by evidence that infarction of the cerebellum is also associated with atheromatous disease of the vertebral arteries outside the skull. Usually sparing the brainstem, the ensuing infarctions differ from those associated with localized occlusion of the individual cerebellar arteries in that they are often bilateral, symmetrical, in the territory of the superior cerebellar artery, and generally masked by cerebral hemisphere symptoms arising from associated disease of the carotid vasculature. That the stenoses of
collateral channels play an important part in the development of symptoms related to occlusion of the carotid arteries seems to be clear. But the authors struggle for an explanation of why infarction occurs at sites remote from extensive localized atheromatous disease, given the relative absence of intracranial vascular disease. Eventually they settle for an explanation on Professor Biemond’s demonstration that rotation and extension of the head to one side may obstruct the contralateral vertebral artery. It follows that atheromatous disease in both vertebral arteries is particularly likely to accentuate such flow-related symptoms, and—returning to their evidence for displacement adjacent to the neurocentral joint—Drs Hutchinson and Yates raise the spectre that cervical spondylosis may also be a contributory factor.

Later, Dr Castaigne and colleagues systematically collect post-mortem specimens of the extra- and intracranial vessels over a period of 8 years and report 44 with embolic, thrombotic (in situ or propagated from a more proximal site) or undetermined occlusive and stenotic lesions in the vertebro-basilar territory—leaving aside a further 13 cases in whom infarctions occurred but without demonstrable abnormality of the main vertebro-basilar arteries or their branches. Most lesions result from local thrombosis at the site of a tight stenosis; a few (eight in total) are due to embolism from a source of atheroma in the proximal vertebral artery or the heart and usually reaching one or other posterior cerebral arteries. Occlusion of one or both vertebral arteries is found in 50% of the patients, very occasionally due to retrograde propagation from the basilar and often with vessels in the posterior circulation affected at more than one site, the left posterior inferior cerebellar artery being especially vulnerable. When asymmetrical, either the larger or smaller vertebral may be affected.

The French authors find that the majority of occlusions occur primarily in the intracranial part of the vertebral artery with only 25% being proximal lesions, although distal occlusion may follow embolism from a proximal site, and the clot often propagates a considerable distance. To their surprise, anterograde extension is not apparently attributable to disease in the circle of Willis or the other vertebral artery, co-morbidities that might be expected to reduce collateral blood flow and so encourage stagnation beyond the primary thrombus, although this mechanism does appear more relevant when the main basilar artery is occluded.

Vertebral artery occlusion is likely to result in infarction of the medulla, in particular the retro-olivary territory or inferior olive, and the cerebellum within the territory of the posterior inferior cerebellar artery especially when there is occlusion at its origin or unilateral vertebral occlusion occurs in association with partial damage to the contralateral vertebral vessel. Conversely, 50% of patients with a single vertebral occlusion and no propagation or associated lesions escape infarction. Whereas the brain stem and cerebellar infarctions often result from primary or extended thrombus, those in the posterior cerebral artery (unilateral and bilateral) are almost invariably embolic, or the result of propagation from an occluded basilar artery unprotected by collateral supply from the carotid circulation due to a vestigial or diseased posterior communicating artery on that side. Dr Castaigne and colleagues also correlate their findings in the vertebro-basilar system with carotid disease. Taken together, 21 of 35 (60%) patients with vertebro-basilar occlusions due to atherosclerosis have no significant disease of the carotid vasculature. Of those who do, tight stenosis and complete occlusion are equally frequent.

What do the authors make of their meticulous listing of pathological features in these 44 Parisian patients studied at autopsy? Most have atherosclerosis at the site of the occlusion and this correlation between local disease and clinical symptoms is more apparent than in the authors’ series on carotid disease published elsewhere, reflecting the relative importance of embolic and thrombotic occlusion of these vessels. Specifically, cardiac embolism is more likely to traverse the anterior than the posterior circulation. Local thrombotic occlusions in the vertebro-basilar territory are almost invariably associated with tight atherosclerotic stenosis, and this observation also differs from their findings in the carotid vessels. That said, thrombotic occlusion of the vertebro-basilar territory does commonly lead to embolic infarction in the
posterior cerebral artery and, in this respect, the findings match events occurring in the carotid tree. Unlike other authors, including C. M. Fisher and Drs Hutchinson and Yates (see above), the French team suggests ‘that the pathological role of proximal vertebral artery stenosis should not be over-estimated’ and ‘this may be of some help when surgery upon a proximal vertebral artery is contemplated’. They are not impressed by the interpretation of Hutchinson and Yates (writing elsewhere) that the relative lack of anterograde extension in the vertebral versus basilar arteries relates to the many branches receiving a rich retrograde collateral flow that limits clot extension. But this argument breaks down when they fail to correlate patency or not of the circle of Willis with anterograde propagation beyond a basilar occlusion.

Dr Castaigne and colleagues have written a paper that is not easy to read, involving unstable numerators and denominators and much restating of numbers in the results, discussion and summary that may exhaust even the most diligent reader trying to keep track on the fate of these 44 Parisian patients. Readers will search the repetitive catalogue of what the authors observed for something also of what they thought. But these early papers, exploring the effects of atherosclerosis in the four main vessels of the head, based on pathological examination in hospital series, is now updated and complemented by the population-based series from Oxford that also compares vertebrobasilar and carotid disease, and the clinical consequences of stenoses affecting these vessels (page 982).

Alastair Compston
Cambridge