against any tentorial herniation as a cause of the oculomotor palsy.

Wilson et al. recently described three patients with internal carotid artery aneurysms, and basilar artery abnormalities. They observed that the blood supply to the ophthalmic artery is from branches of the internal carotid artery within the cavernous sinus, from multiple branches of the maxillary artery, as well as from the posterior cerebral and basilar arteries posteriorly. This rich blood supply from multiple sources could explain why oculomotor palsy is so unusual, even transiently, after carotid occlusions or carotid ligations for the treatment of aneurysms, since occlusion of several components of the nervous system is probably required to produce significant ischemia. Thus in the oculomotor artery, there are diffuse small vessel abnormalities in the vascular nervous system. Similarly, flow was absent in the ophthalmic and internal carotid arteries in our case and in those of Wilson et al. Whether the resulting oculomotor palsy is transient or permanent may perhaps depend on the adequacy of the collateral blood supply from the posterior circulation, or on the extent of segmental occlusion of the plexus of small vessels along the nerves themselves.

Penicillamine treatment of Wilson's disease and optic neuropathy

We report a case of optic neuropathy associated with penicillamine treatment of Wilson's disease.

A twenty six year old woman presented with a one year history of progressive shaking of her hands and four months of shaking of her head. As a result she had had to give up her job. She had a family history of neurological or liver disease. She had a history of tachycardia and had taken disopyramide 100 mg three times a day for three years. A mitral regurgitation murmur had also been noted in the past.

On examination her pulse was 80/min and regular, and blood pressure was 115/60. There was a mid and late systolic murmur loudest at the left sternal edge. Higher functions were intact, but her manner was disinhibited. Visual acuity was 6/9 on the right and 6/6 on the left. Eye movements showed normal for lateral gaze. There was a bilateral skew deviation of the eyes. She had mild mitral valve prolapse.

Penicillamine was stopped and pyridoxine 50 mg twice a day was started. Nine days later near vision was 6/60. Pupils were equal, round and medially directed. The optic discs showed a small area of pallor and both pupils reacted sluggishly to light. Corrected visual acuity was 6/24 on the right and 6/18 on the left. Near vision was N18 bilaterally. Visual evoked potentials (VEP) showed latencies of 102 ms on the right and 112 ms on the left (Normal <115 ms). Brainstem auditory (BAEP) and somatosensory evoked potentials (SSEP) were bilaterally delayed. The wave form of the BAEPs was small. Electroretinogram and autoimmun profile were normal.

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*J Neurol Neurosurg Psychiatry* 1991 54: 746
doi: 10.1136/jnnp.54.8.746

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