**Letters**

Vertebrobasilar insufficiency in a patient with anomaly of the thyrocervical trunk: an unusual steal syndrome?

Sir: The subclavian steal syndrome is a well recognised cause of cerebral ischaemia. Nevertheless, more rarely, vessels arising from the subclavian artery may be responsible for the same symptoms as suggested by the following case.

In April 1979, a 54-year-old farmer complained of progressive post-prandial somnolence, lasting from one to three hours, during which chewing or elaborate activities, or sometimes a staggering walk, were noted. Recent occipital headache occurred in the morning, after a diminishing period of sleep. The patient had a previous history of peripheral thyroid-gland insufficiency of unknown aetiology, without goitre, revealed 4 years before by cold sensitivity and a deeper voice. He did well with 50 µg of liothyrone per day, which had normalised the TSH level. The neurological symptoms progressively worsened in severity and frequency. In July, they prevent the patient from working. On admission, neurological and ocular examination was negative. Memory, attention, and language were normal. There was no goitre or, vascular murmur, or signs of hypothyroidism. Auscultation of the heart was negative. No orthostatic hypotension was noted. Bitemporal theta waves were recorded on the electrocardiogram within a normal basal rhythm. Doppler studies showed normal vertebral flow, without decrease or inversion of the blood flow. CT scan showed moderate diffuse cerebral atrophy without low-density area or focal contrast enhancement. The clinical data suggested transient cerebral ischaemia mainly in the vertebro-basilar circulation. Angiography was performed by right brachial puncture with contro-lateral cervical compression. Both internal cervical and intracranial carotid arteries appeared normal, as were the right vertebral and basilar arteries and their branches. There was no atheromatous stenosis. On the cervico-thoracic views (fig) the vessels arising from the right subclavian artery, and especially the inferior thyroid artery, appeared enlarged without any aneurysm or angioma. Both the severe clinical picture and the angiographic studies suggested a right thyrocervical trunk steal syndrome, at the expense of the vertebral circulation. In September 1979, 6 months after the onset of the clinical signs, operation showed that both thyroid lobes were normal. There were no angiomatic vessels but the right inferior thyroid artery was unusually big, though flexible. The right thyrocervical trunk, arising from the subclavian artery a little lateral to and in front of the vertebral artery, was ligated. Following operation night sleep became normal within two days. Headaches, transient cerebellar ataxia and somnolence almost disappeared within a week. The patient resumed work two months later and remained free from cerebral ischaemic symptoms for 4 years, except for a brief episode of automatism under transient vasodilator treatment. Hormonal therapy was progressively reduced to 12.5 µg of liothyrone per day, the TSH level staying at 5 µu/ml.

Relapsing vertebrobasilar ischaemia was suggested by the transient symptoms, and led to angiographic studies, which showed no classical aetiology such as atheromatous stenosis, or congenital anomaly of the brachiocephalic arteries. Only an enlargement of the arteries originating from near the ostium of the right vertebral artery was shown. This “mega-arterial” finding could not be related to a goitre nor to an angiomatous malformation, but seemed isolated. The clinical picture might have been due to a precarious equilibrium of blood flow between the right vertebral artery and this neighbouring large vessel, leading to a steal syndrome especially after eating. Spectacular improvement of the symptomatology after ligation of the thyrocervical trunk corroborates this hypothesis. To our knowledge, there is no previous report of a thyrocervical trunk steal syndrome in the literature. Lesoin et al. have related a possible steal syndrome of the right superior thyroid artery in a patient suffering from frequent ischaemic transient hemiparesis. In this case, the steal syndrome diverted the precarious blood flow of the carotid arteries, both originating from a common trunk shared with the right subclavian artery and shifted by a goitre which extended to the upper thorax. The goitre was removed and complete recovery followed. In our case, the improvement of hypothyroidism after surgery, seems fortuitous and cannot be related to the arterial ligation; probably it occurred in the natural course of a chronic thyroiditis. This observation illustrates the necessity of a complete heart-to-brain angiographic study for patients presenting with unexplained cerebral transient ischaemic symptoms.

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Fig  Right subclavian arteriography by humeral puncture. Anteroposterior cervico-thoracic views. (a) early arterial stage (b) late arterial stage.
Letters

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References

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Familial writer’s cramp

Sir: The aetiology of writer’s cramp is still controversial.1–4 In spite of the facts that favour an organic origin, genetic influence on this anomaly has not been demonstrated. We report a patient with writer’s cramp, whose father had presented the same disorder 35 years previously.

The propositus was a 41-year-old male right-handed lawyer. He had a previous history of hyperuricaemia and gout treated with allopurinol, 100 mg/day. He had not received any other specific treatment before, was not exposed to contact with toxic substances, did not consume illegal drugs and had not suffered any psychiatric or neurological illness. There was no consanguinity between his parents who did not belong to a particular ethnic group. The patient had had postural tremor since youth. At the age of 33 years he suffered episodic tremor in the right elbow which remitted with local therapy. Since then, he had experienced difficulty in writing with the right hand, that adopted an abnormal posture such that it attracted the attention of other persons. The disturbance was accompanied by a feeling of stiffness and pain in the forearm and wrist. Any other manual activity was carried out normally. The difficulty in writing had remained stable since the onset of the symptoms but its intensity varied at times and is influenced by the emotional state of the patient. On examination, moderate obesity and very light postural tremor in upper limbs were noted; in addition, there was a minimal tremor in the finger-nose-finger test. His writing was legible, but he wrote with moderate difficulty, with the wrist tending to extension from the start, while the fingers bent together clutching the pen. The pressure on the paper was slightly increased. There was no micrographia. These signs lasted as long as writing continued. The remainder of the examination was normal. Routine haematological and biochemical studies, uric acid, LE phenomenon, rheumatoid factor, radiographs of the chest and skull, ECG, brain high-resolution CT with contrast, ceruloplasmin and cornea were normal.

The father of the propositus is a 70-year-old retired lawyer, right-handed. Since the age of 35 he had experienced a non-progressive severe difficulty in writing, consisting of abnormal posture of the hand, inability to maintain the pen in the correct position, involuntary extension of the index finger (“it escapes”), and tremor. All these symptoms were present only during writing or detailed drawing. The resulting script was almost illegible, and the difficulty in writing so marked that he has used a typewriter from the onset of trouble. He has practised artistic painting without problems except for delicate drawings, when aid with the left hand becomes necessary. For 2–3 years before consultation, he had had cephalic tremor and postural tremor in upper limbs. He had no other symptoms. Examination revealed slight head tremor, mild postural tremor in upper limbs and very slight tremor on finger-nose testing. A moderate increase in tone in wrists, a little more marked on the right, with contralateral activity was noted. Writing and drawing were very difficult: from the start, the right wrist was placed in extension and ulnar deviation, with its ventral aspect on the table and fingers forced in flexion. When writing, the posture worsened, a remarkable tremor was superimposed and irregular jerks separated the index finger; the pen became sustained between thumb, middle and ring fingers and, eventually, escaped. When the attempt to write ceased all these signs disappeared. The remainder of the examination was normal, as was routine analytical and radiological tests. The patient refused specific studies.

These patients were considered to suffer from simple writer’s cramp.5 According to Marsden,6 even simple writer’s cramp is a “minor” form of dystonia. Occasionally, some patients with writer’s cramp provide data suggestive of familial involvement,7 but there is no clear evidence of genetic factors in most patients with writer’s cramp.8 Writer’s cramp was considered to be a variant of focal dystonia.9 This observation favours an organic origin of the disorder.

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References

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Asterixis due to pontine haemorrhage

Sir: Asterixis is a common sign of metabolic encephalopathy and arises from various causes.1 This involuntary movement has also been observed in a focal brain lesion.2–7 In this letter, we describe a patient with asterixis due to pontine haemorrhage.

A 65-year-old woman was admitted because of left motor weakness and dip-
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