performed 16 days after the onset of the nerve palsy, at a time when amyotrophy and fasciculations were present in the left lingual muscle. It was abnormal showing spontaneous fibrillation and denervation potentials limited to this area. Pupillary reaction was tested successively with 4%, cocaine hydrochloride and 1% hydroxyzamethamine hydrobromide eye drops. The left miotic pupil proved unresponsive confirming a third cranial nerve injury.

This tongue palsy ipsilateral to the carotid dissection was clearly of peripheral origin according to the clinical and electromyographic findings and related to a hypoglossal nerve lesion. There may be two explanations for this. An ischaemic truncular origin is suggested by the embolic potential of the dissection. Paralysis of the four lower cranial nerves has been described after involvement of the external carotid artery.14 However, to imply such an explanation in this case, it would be necessary to suspect an embolic process from the external carotid artery (ICA), reaching the ascending pharyngeal artery, then the neurenoineal trunk and finally the hypoglossal branch in the hypoglossal canal. This explanation seems vincible, however, because the dissection did not involve the ECA and it was located downstream from the carotid bifurcation. Hypoglossal nerve palsy associated with ICA dissection suggests a local mechanism in the genesis of this syndrome.

To our knowledge, there are only two previous documented cases of hypoglossal nerve palsy associated with ICA dissection, and only one in which nerve palsy is isolated.5 In our report, the dissection caused a hypoglossal nerve dysfunction probably by a focal enlargement of the ICA by the haematoma leading to a local compression. Such an enlargement has been shown by magnetic resonance imaging (MRI) of ICA dissection6 confirming the potential value of MRI in this condition. Unfortunately, MRI was not performed in our case. We cannot explain the reason why the other lower cranial nerves were not compromised by the local compression.

This report makes two points with practical implications: 1) A stroke-related peripheral palsy of the hypoglossal nerve with or without Horner’s syndrome, may be caused by a lesion outside the posterior circulation 2) ICA dissection should be added to the list of aetiological diagnosis of isolated hypoglossal nerve palsy, and looked for especially when cervical pain is associated.

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Development of multifocal haemorrhage in a cerebral infarct during computed tomography

A 71 year old woman with endocarditis and a brain infarct, who was treated with oral anticoagulant therapy, developed an intracerebral haemorrhage during non-contrast CT scanning. The haemorrhage originated in three different parts of the infarct, at the same time.

It is rare to see an intracerebral haemorrhage (ICH) develop during computed tomography (CT). Two examples have been reported after the administration of radiographic contrast material during CT.12 and in two other instances a pre-existent haematoma increased in size during CT examination.13 To our knowledge CT documented sudden haemorrhagic transformation of an infarct without previous injection has not been witnessed before.

On 13 September 1985, a 71 year old woman was admitted to the department of neurology because of a sudden and transient attack of dysphasia with left-sided headache, lasting 30 minutes, two days earlier. In childhood she had suffered from rheumatic fever. Since 1982 she had been treated with oral anticoagulants (coumadin) because of atrial flutter and mitral insufficiency.

Examination showed a rectal temperature of 38-5°C, an irregular pulse rate of 80 per minute, and a blood pressure of 140/90 mm Hg. Over the heart there was an apical high-pitched pansystolic murmur transmitted to the axilla. There were no systemic signs of endocarditis. The level of consciousness was normal. There was a mild non-fluent dysphasia, but no other neurological deficit. Laboratory investigation showed anaemia (haemoglobin 5-2 mmol/l), normal range in female 7-5-10-0 mmol/l); the haematocrit was 0-25. The number of white cells was 11-0 × 10⁹/l with 94%, polymorphonuclear cells, the ESR was 103 mm/hour. The level of anticoagulation was appropriate, corresponding with an international normalised ratio (INR) of 2-5. Blood cultures yielded the growth of a species of streptococcus and E coli. Electrocardiography showed sinus rhythm. Non-contrast CT scanning of the brain on the day of admission was normal. After intravenous injection of contrast, slight enhancement was seen in the insular cortex on the left side, compatible with infarction. A diagnosis of subacute bacterial endocarditis was made and the patient was treated with 24 million units of penicillin daily.

Figure  Scan A, shows a slightly haemorrhagic component in the left temporal region. Scan B, made one minute later, shows this more clearly. Scans C and D, taken 15 minutes later at corresponding levels, demonstrate a marked and multifocal increase of haemorrhagic infarction, with some fluid levels.
Four days later CT scanning was repeated, without contrast, to assess the extent of infarction. An ill-defined hypodense region was visible in the basal part of the left temporal lobe. The adjacent slice showed a slightly haemorrhagic component, with some compression of the sulci and ambient cistern (fig 1a). This was even clearer on the next slice (fig 1b). Intracranial pressure was 150/90 mmHg and the patient's level of consciousness suddenly deteriorated (Glascow Coma Scale Score E1 M3 V1, with small, unreactive pupils).

CT scan fifteen minutes later showed fresh haemorrhage, heterogeneously distributed over at least three foci, with a marked shift to the right (fig 1, C and D). At this time the level of anticoagulation remained appropriate (2-4 INR). The anticoagulant was reversed with vitamin K, the coagulation factors II, VII, IX, X, and fresh frozen plasma. Nevertheless, the patient died after several hours. Permission for necropsy was not obtained.

In this patient with bacterial endocarditis the brain infarct may have been caused by an infected embolus from the heart. It is uncertain whether arteritis was a factor in the cerebral infarction. Treating the patient with anti-embolus and antibiotic therapy may show multifocality and can be recognised on CT-scanning, even in anemic patients.

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**Interruption opthalmoplegia in giant-cell arteritis**

**Patients with giant cell arteritis (GCA) occasionally complain of double vision.**

**Arteritic involvement of the ophthalmic artery within the orbit is usually the cause.**

*We describe two patients with GCA who were suspected to have bilateral involvement of the ophthalmic artery (INO). Involvement of the medial longitudinal fasciculus (MLF) in GCA has not been previously reported.*

**The first patient was an 81 year old woman who presented with a three month history of anorexia and 14 kg weight loss. She was confused. One month before admission she had developed diplopia on looking to the left. She also complained of head pains over her vertex "as if gnomes were chipping away at my brain", and she had some jaw stiffness. Examination revealed a visual acuity in her right eye of 6/7.5, N6 and in her left eye of 6/12, N6. Colour vision was normal in both eyes and visual fields were full. Fundoscopy revealed moderate lens opacities and myopic discs. Papillary responses were normal. There was a right INO. She was able to converge normally. The temporal arteries were variably tender though pulsatile normally. Her erythrocyte sedimentation rate (ESR) was 84 mm/hour and haemoglobin (Hb) 9 gm/100 ml. Temporal artery biopsy showed transtorial inflammation with disruption of both the internal and external elastic laminae. Treatment with steroids led to a resolution of her symptoms and normalisation of head pains within two days. When she left hospital five days later, her eye movements had improved considerably though the internuclear ophthalmoplegia persisted.*

**The second patient was an 85 year old man who had a four month history of left peribial head pains, drooping of the left upper lid, 7 kg weight loss and some scalp tenderness. Two weeks before admission he developed diplopia when looking to his right. His double vision developed whilst playing bridge and he accused his friend of having too many cards and cheating. Examination of his eyes revealed visual acuities of 6/6 right and 6/6, N5 on the left. Colour vision was normal. A small right choroidal haemorrhage was seen in the right eye. The left fundus was normal. His visual fields were normal. A left Horner's syndrome was present. There was a left INO with associated vertical nystagmus. He was able to converge. The temporal arteries were clinically normal. His ESR was 100 mm/hour, Hb 10 gm/100 ml and the alkaline phosphatase was raised. Temporal artery biopsy revealed near complete occlusion with intimal proliferation and destruction of media and elastica. A prominent chronic inflammatory infiltrate around vessel cells was present throughout the vessel wall. His head pains and scalp tenderness responded overnight to steroid treatment and at the time of discharge the Horner's syndrome had resolved and the internuclear ophthalmoplegia was improving.**

*Ten to 15% of patients with GCA may complain of double vision.**

*Eye movement abnormalities in this condition are due either to arterial involvement of the vasa nervorum of the oculomotor nerves, or to ischaemic necrosis of the extracocular muscles themselves.*

*The examination of the ophthalmic artery within the orbit is the cause in both cases.*

*However, in our patients INO was the cause of diplopia. The lesion is in the MLF which runs from the pons to the superior colliculus, and the blood supply to this area is from perforating branches of the basilar artery. As the extradural portions of the vertebral arteries are known to be involved in the arteritic process in almost all cases of GCA, we presume that the perforating branches to the MLF became involved on the basis of embolisation. It is perhaps surprising that INO has not been reported before in GCA although presumably this is due to the difficult anastomotic supply to the pons and midbrain.**

*The Horner's syndrome in patient two proved pharmacologically to be post-ganglionic and was probably due to a lesion in the "carotid" sympathetic fibre. Many patients with GCA are reported with ptoisis and miotic pupils although verification of sympathetic denervation is rare.**

*These cases highlight the diversity of clinical presentations of GCA and support the maxim that all elderly patients presenting with neurological disease of possible vascular origin deserve an ESR and a biopsy of temporal artery biopsy and perhaps a therapeutic trial of steroids.**

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**PATTY TRENDS**

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