Treatment of bruxism with botulinum toxin injections

Local injections of botulinum toxin have been used with success for treatment of strabismus and blepharospasm. This approach has been extended to different types of focal and segmental dystonia (torticollis, oromandibular dystonia, laryngeal dystonia, writer's and musician's cramp) and hemifacial spasm. We now report the successful treatment of bruxism with botulinum toxin injections.

A 32 year old woman was admitted to a coma after a car accident. The right pupil was larger (3 mm) than the left (2 mm); there were roving eye movements with incomplete abduction of the right eye. Spontaneous extensor spasms of the right arm and leg were observed; the left plantar response was extensor.

A CT scan showed an inhomogeneous hyperdensity in the left parietotemporal area, consistent with a brain contusion. The patient was intubated and ventilated but recovered slowly. After four months, some communication with the patient was possible and she obeyed verbal commands. However, at this stage the patient had developed bruxism consisting of jaw clenching and chewing movements. The teeth grinding was very loud and almost continuous, causing damage to the teeth and annoyance to nearby patients.

Six months after the accident, 25 units, or 10 ng毒in-α-hemagglutinin complex of botulinum A toxin were injected into both the temporal and masseter muscles; no attempt was made to infiltrate the pterygoid muscles. After five days, we observed a marked reduction in the bruxism. There was no excessive weakness in the masticatory muscles so that feeding became possible. The favourable effect of the injections lasted for eight weeks. Two weeks later, a second injection of a total of 100 units (40 ng) of botulinum toxin was given. The results were similar and the effect persisted twelve weeks later.

Bruxism consists of rhythmic teeth grinding during sleep. It occurs in the general population, with a prevalence of five to 21%. However, an association of bruxism with organic brain damage and altered states of consciousness has been documented. The onset of bruxism is frequently linked to the return of sleep-wake cycles in comatose patients; damage to the teeth and related bony structures is one of the major complications of chronic bruxism. Treatment with a special dental prosthesis may be needed to prevent injury to the teeth or dental loss but could not be fitted in our patient due to lack of cooperation. Bruxism can now be added to the list of indications for botulinum toxin treatment.

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Internal carotid artery dissection and ipsilateral hypoglossal nerve palsy

Dissections of carotid and vertebral arteries are increasingly recognised in recent years as a cause of stroke and account for at least 5% of ischaemic stroke in young adults. High quality angiography has contributed to their improved recognition. The clinical picture of carotid dissection typically associates hemispheric symptoms with ipsilateral neck pain or headache, and oculosympathetic paresis (Homer's syndrome). Minor forms are possible. We report a very unusual case in which internal carotid artery (ICA) dissection presented only with Homer's syndrome and hypoglossal nerve palsy.

A 48 year old normotensive man received a left cervical blow while fighting with fellow workers. He immediately experienced pain at the site of the blow that radiated to the head on the same side. Six days after he had difficulty in chewing but not with swallowing. There were no other complaints except for the pain.

Examination revealed a left Horner's syndrome. The tongue appeared to divert to the right at rest and to the left when protruded (fig 1a), revealing a left hypoglossal nerve palsy. Phonation and deglutition were unimpaired. The rest of the neurological examination was normal. There were no cervical bruits. The diagnosis of left ICA dissection in the neck was confirmed by the presence of a “string-sign” on left carotid angiography (fig 1b). Dissection was located approximately six cm above the carotid bifurcation, at the point at which the artery penetrates the bone.

CT scan of the brain showed no ischaemic lesion. An electromyogram of the tongue was performed showing a weak response on the left side. H-E stained sections of the tongue muscle showed capillary proliferation in the submucosa.

Fig 1a  The tongue appears to divert to the right at rest and left when protruded.
Fig 1b  Left, carotid angiography showing a string-sign" 6 cm above the carotid bifurcation.
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% hydroxyamphetamine 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. 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 neurovomeningeal trunk and finally the hypoglossal branch in the hypoglossal canal. This explanation seems very unlikely, 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. In our report, the dissection caused a hypoglossal nerve dysfunction probably by a focal enlargement of the ICA by the haematomata leading to a local compression. Such an enlargement has been shown by magnetic resonance imaging (MRI) of ICA dissection 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 4 and in two other instances a pre-existent haematoma increased in size during CT examination. 4 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 x 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.
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*J Neurol Neurosurg Psychiatry* 1990 53: 530-531
doi: 10.1136/jnnp.53.6.530-a