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CORRESPONDENCE

Sir,

**Traumatic superior orbital fissure syndrome**

A case of traumatic superior orbital fissure syndrome is described, which is made more interesting by the fact there were no associated orbital fractures demonstrated on imaging and that despite complete loss of upgaze in the affected eye on voluntary and oculocephalic testing Bells phenomenon was still intact.

*Case report*

A 49-year-old male sustained a severe blunt injury just below the bridge of the nose with industrial machinery.

Visual acuity was 6/6 in both eyes. There was no proptosis. He had a complete left ptosis. Both pupils were equal in size and reactive to light and accommodation. Colour vision was intact. Ocular movements were full in the right eye. The left eye had no movement in any direction (Figure 1). Of particular note, there was no movement of the left eye in voluntary upgaze or oculocephalic testing, however, a moderate Bells reflex was seen. The fundi were normal. The patient had loss of sensation in V.[1]

Since the patient was stable and there was no evidence of optic nerve involvement, the provisional diagnosis of superior orbital fissure syndrome was made and the patient commenced on oral steroids and antibiotics. Pupillary reactions were carried out throughout the night and a CT scan was arranged for the following morning. The CT scan of the orbits (2 mm slices with coronal reconstructions) showed no haematoma or fracture of the superior orbital fissure or surrounding orbital bones. The only fracture found was that of his nasal bones.

After 24 h, the left eye was beginning to open spontaneously. Ocular movements showed a very slight return of elevation and other movements were absent. The steroids were stopped and a diagnosis of neuropraxia of the superior orbital fissure nerves was made. At review in clinic 2 weeks later, the lid was completely open with some return of movement to the left eye. A moderate limitation of elevation and depression and a mild limitation of abduction and adduction were noted. On review 6 weeks later, ocular movements had returned to normal.

**Comment**

Hirschfeld first described superior orbital fissure syndrome in 1858. [1] The syndrome occurs in both complete and incomplete forms, depending on the extent of involvement of structures passing through the superior orbital fissure. Superior orbital fissure syndrome can therefore give rise to a combination of ophthalmoplegia, ptosis, proptosis, anaesthesia in the distribution of V[1] and a fixed dilated pupil. If the optic nerve is involved, it is designated orbital apex syndrome.[1–3]

Documented causes of superior orbital fissure syndrome include tumours, haemorrhage, infection, or trauma.[1] Superior orbital fissure syndrome is rarely associated with trauma; when it is, it is usually in association with fractures involving the facial skeleton.[1,2] The nerves of the

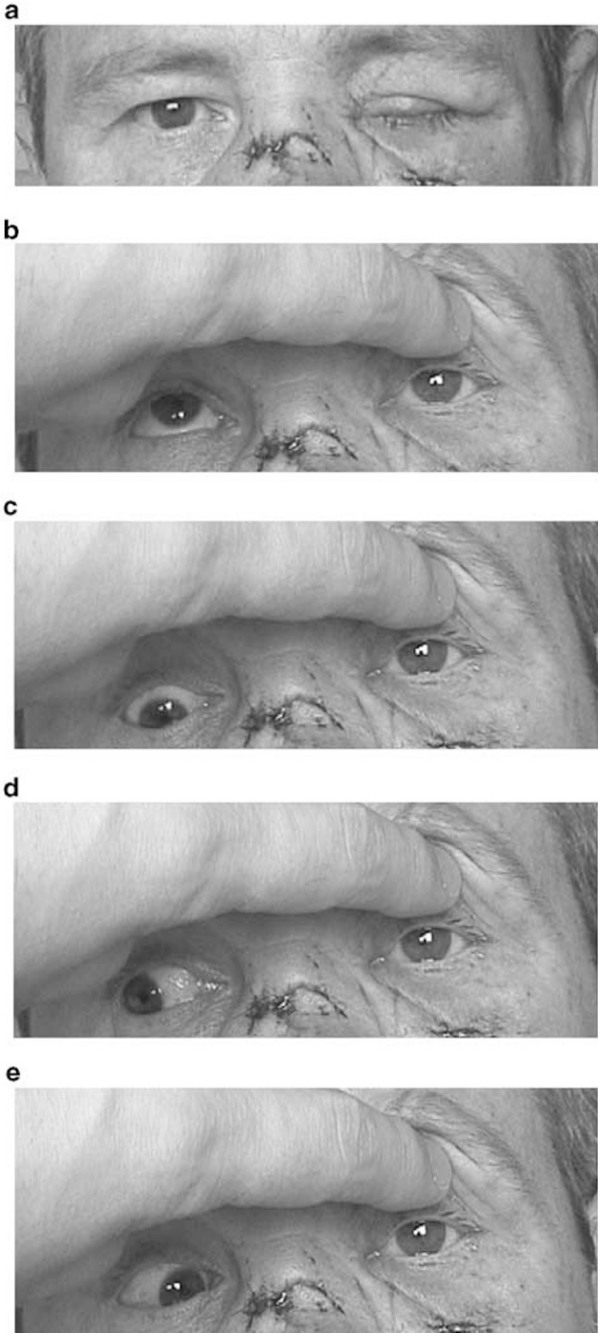


**Figure 1** (a)–(e) Testing of ocular movements demonstrated complete akinesia in the left eye.

superior orbital fissure can be damaged by displaced fractures of the bony boundaries of the fissure or haemorrhage. Since our patient had no associated fractures or haematoma demonstrated on the CT scan, it was felt that the transmitted force from the trauma gave rise to a neuropraxia of the nerves, which then resolved resulting in the rapid recovery of the patient's ocular movements. Neuropraxia is defined as the mildest type of focal nerve injury producing clinical deficits and is followed by complete recovery.[4] The presence of Bells phenomenon in our patient, despite loss of upgaze, suggests that Bells phenomenon is a very strong stimulus for upward ocular movement, much stronger than either voluntary or oculocephalic movements.[5]

In reviewing the limited literature on the treatment of the condition, it appears that the prognosis is usually good especially in those with undisplaced fractures of the fissure. Those cases that are, however, associated with comminuted fractures disrupting nerve function have a poorer outlook. Surgery can be considered when there is evidence of optic nerve compromise either by bony fragments impinging on the nerve or in cases associated with a nonresolving orbital haematoma if after 3–4 months there is evidence of optic atrophy.[1] In the absence of optic nerve dysfunction, the patient could lose vision if surgery resulted in iatrogenic damage to the nerve.[3] It is difficult to know whether the use of steroids in superior orbital fissure syndrome helps, as there are not a sufficient number of cases to give a definitive answer. Theoretically steroids may decrease the inflammatory reaction around the nerves, giving rise to a speedier recovery. Whether or not it affects the final outcome, it is not possible to say.

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Sir,

**Cataract surgery for natural rubber latex allergic patients**

We would like to thank Mr Beare for highlighting the regional variation in the management of natural rubber latex (NRL) allergic patients. This variation is partly due to the lack of evidence-based recommendations for management of NRL allergy. It is hoped that our review article[1] would help ophthalmic units to examine the literature surrounding NRL allergy such that they can formulate their own protocols for operating on latex-allergic patients. As reiterated by Mr Beare, the prevalence of patients with true severe NRL allergy, where contact with NRL allergen causes a life-threatening reaction, for example, bronchospasm and anaphylaxis, is probably extremely low and may never be known due to difficulties in clinical diagnosis, *in vivo* and *in vitro* testing. *In vivo* testing of patients for NRL allergy is considered the definitive confirmatory test, but ethical and safety considerations exist.[2,3] Definitive diagnosis of NRL hypersensitivity revolves around the consideration of the clinical history with confirmatory *in vitro/in vivo* testing.[4]

The management of NRL allergic patients, as with all kinds of risk management, involves the balance of the potential risks of operating on that patient in the normal operating environment *vs* the potential disruption caused by changing to an NRL-free environment. Again, variation exists between different hospital trusts in how the relative risks are perceived. Local policies also appear to be influenced by the subspecialties involved in their creation. In many hospital trusts, it is the anaesthetic department's responsibility to produce guidelines for NRL-free environments.