prophylaxis was given and on conservative management with antibiotics and dexamethasone the left hemiplegia and the horizontal gaze palsy rapidly resolved. After several weeks an effenter pupill response could be elicited on the right side, corneal sensation was restored, and considerable improvement was seen in the external ophthalmoplegia. Loss of vision in the right eye was permanent and probably resulted from swelling of the optic nerve within the confines of the bony optic canal following contusion. Within a month optic disc pallor was apparent. Permanent damage to cerebellar pathways resulted in ataxia and horizontal nystagmus which did not resolve.

It is evident that the foil entered the orbit through the upper conjunctival fornix producing very little external sign of injury. It traversed the orbit and, taking a course determined by the downward slope of the orbital roof, passed through the superior orbital fissure and on, lateral to the cavernous sinus, before coming to rest in the region of the pons and right cerebellar hemisphere. To date there are few reports of hemiplegia following transorbital penetrating injury.2,11-13 and so far as is known the finding of a gaze palsy in such an injury has not previously been reported. The possibility of intracranial extension must always be considered in cases of orbital injury. It is wise to admit all potential transorbital injuries to hospital for observation and the importance of a careful neuro-ophtalmological assessment at the initial examination cannot be overstated.2

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References

Focal dystonia in association with cerebral infarction

Sir: We wish to report the case of a woman who developed bradykinesia and rigidity on the left hand, arm, and subsequently, a progressive fixed dystonic posture of her left hand in association with an infarct in the region of the right basal ganglia. In 1974, when 43 years old, the patient presented with a six month history of progressive weakness of the left hand, with difficulty in doing up buttons or manipulating a fork. There was no family history of consanguinity or of neurological disease and the patient's previous health had been excellent. On examination there was a mild resting tremor of the left hand, with cogwheel rigidity at the elbow and wrist, and fine finger movements of the left hand were moderately impaired. The blood pressure was 230/140 mm Hg.

An initial diagnosis of Parkinson's disease was made and the hypertension treated, first with methylpocy and later propranolol. No anti-Parkinsonian therapy was given and after a year the tremor remitted, but there was a slight increase in the rigidity and bradykinesia in the left arm. The latter failed to respond to trials of levodopa, bromocriptine or anticholinergic treatment, which was tried over the following two years. In 1977 the patient first developed an abnormal posture of the left hand and this gradually increased during the next three years. On examination at the end of 1981 she was normotensive and her left hand was now permanently maintained in a dystonic posture (fig A). The medial three fingers were flexed at the metacarpo-phalangeal
Association of Klippel-Trenaunay-Weber syndrome with myotonic dystrophy

Sir: Myotonic dystrophy, an inherited disorder, is associated with cataract, frontal baldness, cardiac dysfunction, testicular atrophy, abnormal insulin secretion, Klumpke-Felter syndrome, and Down’s syndrome, in addition to central and peripheral nervous system dysfunction. Cutaneous manifestations, other than balding, are rare. Few cases of pilomatrixoma (a benign calcifying epithelioma) have been reported in myotonic dystrophy. We observed an extensive systematized vascular naevus in a segmental distribution with hypertrophy and affecting one upper limb, the characteristic features of Klippel-Trenaunay-Weber syndrome, in a patient with myotonic dystrophy. An exhaustive review of the literature has failed to disclose similar cases.

A 39-year-old male Muslim, an agriculturist, born of a consanguineous union was seen in December 1980, with complaints of weakness of lower limbs of 2 yr duration. He had most of the cardinal signs of myotonic dystrophy such as myotonia, frontal balding, testicular atrophy, posterior capsular cataract, wasting and weakness of temporalis, masseter, sternocleido-mastoid muscles of the forearm, hands and feet (fig). Electromyography showed well sustained spontaneous, high frequency discharges, myopathic pattern and nerve conduction studies showed normal motor and sensory conduction velocity in the upper and lower limbs. A cutaneous patch on the right side of the chest and medial aspect of the arm was noticed since birth. This was dull red in colour in the distribution of thoracic 1 and 2 segments (fig). Global hypertrophy of the right arm including the shoulder was present. No venous varicosities were seen nor was there any evidence of associated disorders like syphilitic, polydactyly, scoliosis, pulmonary hypertension, mental retardation, epilepsy or paraplegia.

References