cytological change in the bone marrow. Multiple extramedullary plasmacytoma is a rare tumour with an incidence of about 1% of all plasma cell tumours. Cranial and intracranial plasma cell tumours are also extremely rare. Cushing found that among 2000 intracranial tumours, only four were intracranial myelomas but he did not record the details.

Clarke reviewed 24 cases of cranial and intracranial plasma cell tumours from the literature and added four of his own. He distinguished three separate syndromes; multiple cranial nerve palsies, intracranial tumour formation, and a constellation of signs due to invasion of the orbit by plasma cell tumour. Intracranial abnormalities are thought to be due to plasmacytomas arising in the base of the skull. Alternatively, it has been suggested that increased intracranial pressure could be due to abnormal globulin production inducing a hyperviscosity syndrome with protein deposition in the central nervous system. In our patient, CT scan showed that there was no significant mass lesion and plasma viscosity was normal. Dural involvement around the left mastoid may have lead to sinus thrombosis and consequent raised intracranial pressure.

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Abducens palsy after rapid shrinkage of a prolactinoma

Sir.—The sixth cranial or abducens nerve, by virtue of its anatomical course, is particularly susceptible to damage in pathological conditions affecting the cavernous sinus. An expanding pituitary tumour may produce a sixth nerve palsy by lateral displacement and stretching of the cavernous portion of the nerve, often with accompanying third and fourth nerve damage. We report the occurrence of a transient sixth nerve palsy in association with the rapid reduction in size of a pituitary prolactinoma induced by bromocriptine, a relationship not previously documented.

A previously well 31-year-old man presented with a two-month history of blurring of vision of the left eye, and diminished libido and potency. He had no associated headache, diplopia or other symptoms. Examination of the left eye revealed a visual acuity of 6/12, an upper temporal quadrantic field loss and pallor of the optic disc. The right eye was normal, as was the remainder of neurological and the general physical examination.

Skull radiographs showed massive expansion of the pituitary fossa, with destruction of the dorsum sellae and almost complete replacement of the adjacent sphenoidal sinus. A large, partly cystic pituitary tumour with suprasellar, cavernous sinus and sphenoidal sinus extension was delineated by axial and coronal CT scanning (fig. a). Visual evoked potentials showed bilateral prolongation of latencies with attenuation of the major positive peaks, these abnormalities being more severe on the left. The serum prolactin, as measured by radioimmunoassay, was 11,300 ng/ml (normal less than 25). Other parameters of endocrine function were normal, including serum TSH, thyroxine, FSH, LH, growth hormone, cortisol and testosterone levels. Bilateral carotid and left vertebral angiograms confirmed significant suprasellar extension, with elevation of the A1 segment of the anterior cerebral artery, and lateral displacement of the cavernous portions of the internal carotid arteries. No evidence of intracranial aneurysm formation or of arterial encasement was present.

Oral bromocriptine was commenced at a dosage of 2.5 mg nocte, gradually increasing to 15 mg per day. The patient’s condition remained unchanged until 4 weeks after the institution of treatment, at which time he developed increasing horizontal diplopia over 4 days. There was no associated headache or changes in visual acuity. Exam-
Letters

In recent years, there has been a growing awareness of the importance of maintaining a healthy lifestyle and regular exercise. These findings are supported by a number of studies that have shown a direct correlation between physical activity and improved health outcomes.

Fig (a) Pretreatment axial (a) and coronal (b) post-contrast CT scans showing a large, partly cystic sellar tumour with suprasellar and lateral extension. (b) Post-contrast CT scan performed after 4 weeks of treatment with bromocriptine showing a dramatic reduction in tumour size, including extrasellar component.
This is particularly true with rapid tumour enlargement, as may occur with haemorrhage into the tumour. Other possible causes of a sixth nerve palsy related to pituitary adenoma are hydrocephalus due to obstruction of the foramen of Monro by tumour, and a coexistent cerebral aneurysm.8,9

Bromocriptine, a dopamine agonist, is a potent inhibitor of the synthesis and release of prolactin. It may dramatically reduce the size of large prolactinomas and there have been several reports of improvement of visual fields and extracocular movements after its administration.10,11 The tumour shrinkage is most likely related to reduction in cell size. There is no evidence of widespread tumour necrosis, vascular injury, platelet aggregation or thrombosis after treatment with bromocriptine.10,11

Our patient developed a sixth nerve palsy in association with a rapid reduction in prolactinoma size, including its extrasellar component. There was no evidence of localised tumour expansion or haemorrhage, hydrocephalus, cerebral aneurysm, recent viral infection or other identifiable causes for the nerve palsy. Although simultaneous independent disease is a possibility, we consider that sixth nerve damage was more likely to have been related to rapid decompression or shift in position of the nerve caused by tumour shrinkage. Hence, the appearance of a sixth nerve palsy during treatment of a large prolactinoma with bromocriptine may signify either rapid tumour expansion or, more rarely, a sudden decrease in tumour size.

No enophthalmos in Horner's syndrome

Sir: Enophthalmos is a controversial feature in Horner's syndrome. Some regard it as mere illusion created by narrowing of the palpebral fissure.1 We investigated the presence of this sign in a prospective series of patients with oculosympathetic dysfunction.

Thirteen patients with unilateral oculosympathetic dysfunction were examined with a "Krahn" exophthalmometer. This instrument makes it possible to measure the exact distance between the anterior surface of the cornea and the lateral margin of the orbit. The patients were examined while sitting. The readings were "blind", owing to an error of calibration, which caused an artificial difference between the right and left eye, and of which the examiner was unaware. We excluded all intracranial or intracanalicular diseases that could affect the position of the eyeball other than by oculosympathetic denervation. Horner's syndrome had been present in these patients from a few months up to more than ten years. The diagnosis of unilateral oculosympathetic dysfunction was based on previously published criteria involving serial photographic of the pupil.2

References


Accepted 15 June 1986

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Sir: Enophthalmos is a controversial feature in Horner’s syndrome. Some regard it as mere illusion created by narrowing of the palpebral fissure.1 We investigated the presence of this sign in a prospective series of patients with oculosympathetic dysfunction.

Thirteen patients with unilateral oculosympathetic dysfunction were examined with a "Krahn" exophthalmometer. This instrument makes it possible to measure the exact distance between the anterior surface of the cornea and the lateral margin of the orbit. The patients were examined while sitting. The readings were “blind”, owing to an error of calibration, which caused an artificial difference between the right and left eye, and of which the examiner was unaware. We excluded all intracranial or intracanalicular diseases that could affect the position of the eyeball other than by oculosympathetic denervation. Horner’s syndrome had been present in these patients from a few months up to more than ten years. The diagnosis of unilateral oculosympathetic dysfunction was based on previously published criteria involving serial photographic of the pupil.2

The exophthalmometric readings in the 13 patients with oculosympathetic dysfunction gave an average of 16.2 mm on the side of oculosympathetic dysfunction and 15.8 mm at the normal side (after correction for the error of calibration). In five cases there was no difference at all, three cases showed enophthalmos (0.5 mm or 1.0 mm) and five showed exophthalmos (1.0 mm or 2.0 mm). Four patients had Horner’s syndrome for more than ten years, one for at least five years, the rest for two years or less.

Enophthalmos should no longer be regarded as a part of Horner’s syndrome. In his original description Horner mentioned only in passing that the position of the eyeball seemed slightly inward (“sehr unbedeutend zuruckgeschusen”).2 The casuistry of this remark contrasts with the completeness of his description of miosis and ptosis and with his measurements of the temperature of the face. Later writers, however, have included enophthalmos among the main features of the syndrome, and Horner’s chance remark has been perpetuated into the textbooks of the present day.4–6 Our measurements fail to show even the slightest relationship between enophthalmos and oculosympathetic dysfunction, not even in patients with miosis and ptosis of more than 10 years’ standing. Similar findings have recently been reported by Lepore7 and Nielsen.8

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Abducent palsy after rapid shrinkage of a prolactinoma.

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