frontal tumour. The optic atrophy is commonly felt to result from optic nerve compression and the contralateral papilloedema from increased intracranial pressure.[1,2] Another mechanism suggests that Foster Kennedy syndrome is due to bilateral direct optic nerve compression by a midline basal mass or less commonly by long-standing increased intracranial pressure without direct compression of either nerve.[3]

Since the early cases of Foster Kennedy syndrome, many cases have been reported in the literature caused by other tumours, especially meningiomas such as olfactory groove and sphenoid ridge meningiomas, with gliomas occasionally reported.[4–7] To our knowledge, nasopharyngeal carcinoma is rarely reported in the literature as a cause of Foster Kennedy syndrome.

Other terms have been used in the literature to describe atypical cases of Foster Kennedy syndrome. 'Pseudo Foster Kennedy syndrome' has been used to describe cases caused by non-compressive pathology such as anterior ischaemic optic neuropathy and optic neuritis.[8] 'Pseudo-pseudo Foster Kennedy syndrome' has been used to describe a case caused by two different pathologies such as meningioma and ischaemic optic neuropathy causing optic atrophy in one eye and swollen optic disc in the other.[9]

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Sir,

**Apraclonidine in the Management of Glaucomatocyclitic crisis**

Glaucomatocyclitic crisis (Posner–Schlossman syndrome) is a unilateral inflammation of the uveal tract in which signs of an acute increase in intraocular pressure predominate. As the aetiology is doubtful, numerous treatments have been suggested, the main aim being to reduce the exceptionally high intraocular pressure which, left untreated, will cause permanent optic nerve damage.

Apraclonidine hydrochloride 1%, a clonidine derivative and a peripheral alpha-adrenergic agonist, was developed to lower intraocular pressure while minimising systemic side effects. It has specific receptor-binding and physico chemical properties that limit its access to the central nervous system. In normal human volunteers it produces a significant fall in intraocular pressure.[1] Apraclonidine hydrochloride 1% is being used to reduce the intraocular pressure elevation after anterior segment laser surgery. It is effective in eliminating the large, acute elevation in intraocular pressure after argon laser trabeculoplasty.[2,3] It can also be used as an adjunctive glaucoma therapy.[4]

*Case Report 1*

The patient was a 37-year-old Malay woman referred by her general practitioner with a diagnosis of acute congestive glaucoma of the left eye. She complained of left-sided headache, and mild pain and redness of the left eye with slight blurring of vision for the preceding 3 days. On questioning she said that she saw haloes from the day of onset. This was her first episode.

Examination showed that her vision was 6/6 part, but she said that she felt as though she was seeing through water. Slit lamp examination did not show any significant oedema of the cornea. There were six unpigmented precipitates on the posterior surface of the cornea. There were no obvious precipitates at the angle. Aqueous did not show significant flare or cells. Gonioscopy revealed that the chamber angle was wide open. The pupil reacted less briskly compared with the fellow eye. The iris was similar in character when compared with the fellow eye. Posterior segment was normal. The intraocular pressure in the left eye was 50 mmHg and in the right eye was 14 mmHg.

At 9.50 a.m. the patient was asked to lie down and 1 drop of apraclonidine 1% was instilled in the conjunctival sac. The pressure was monitored every hour until 10.00 p.m. and again at 8.00 a.m. the next morning. Within 1 hour the pressure dropped from 50 mmHg to 18 mmHg. It remained at 18 mmHg for 6 hours and then rose to 22 mmHg during the seventh hour. Another drop of apraclonidine was instilled. Within an hour the pressure went down to 18 mmHg and in another hour to 14 mmHg, and remained the same throughout 3 days of monitoring. Examination on the third day showed the fields were normal. There was no significant fluctuation of pressure on regular follow-up.

*Case Report 2*

The patient was a 25-year-old Chinese woman. She was

seen during the sixth episode of glaucomatocyclitic crisis, which occurred, on average, once a year. She recognised the symptoms and signs without difficulty and came to my morning clinic. She had a left-sided mild headache, slight pain in the left eye with mild congestion, slight blurring of vision and haloes around bright light. In the past she was treated with Diamox 250 mg q.i.d. and dexamethasone eyedrops q.i.d. It took, on average, 3 days for the pressure to settle down to normal.

Examination showed the collected vision was 6/6 in both eyes. Slit lamp examination did not show any significant oedema of the cornea. There was no evidence of keratitic precipitates on the posterior surface of the cornea. There were no obvious precipitates at the angle. Aqueous did not show significant flare or cells. Gonioscopy revealed that the chamber angle was wide open. The pupil reacted less briskly compared with the fellow eye. The iris was similar in character when compared with the fellow eye in spite of repeated attacks.

At 9.10 a.m. the patient was asked to lie down and 1 drop of apraclonidine 1% was instilled in the conjunctival sac. The intraocular pressure, which was initially 54 mmHg, dropped to 38 mmHg in an hour, then to 24, 20, 17 and 14 mmHg over the following 4 hours. It stabilised at 14 mmHg and did not rise again over 3 days of monitoring. Examination on the third day showed the fields were normal.

## *Discussion*

Two patients with typical glaucomatocyclitic crisis was treated with apraclonidine hydrochloride 1%. Both showed a fall in intraocular pressure within an hour of application of the drops. In one patient the attack was aborted with a single drop. In the other patient there was an increase in pressure after 6 hours which was returned to normal after one more application. No other medication was used. Apraclonidine may prove to be useful in the treatment of glaucomatocyclitic crises.

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Sir,

**Oestrogens and Macular Holes: a Postal Questionnaire**

Macular holes are an important cause of central visual loss in the elderly population, and there is a strong female preponderance (70%).[1] Hormonal changes around the time of the menopause, previous hysterectomy, and hormone replacement therapy (HRT) have been cited as possible risk factors[2,3] though not studied in greater detail. Oestrogens have well-documented stimulatory effects on collagen and hyaluronic acid in the skin, and it is possible they may be similarly active within the eye. Factors such as the menopause, hysterectomy, or postmenopausal HRT could modify this effect. We therefore undertook a postal questionnaire of 103 female patients with macular holes, and asked for details of gynaecological and obstetric history. Since 1988, patients with idiopathic full or partial thickness macular holes have been recruited for research studies at Moorfields Eye Hospital. Those with 6 dioptres or more of myopia, and those with a history of ocular trauma or surgery, have been excluded. From this database we were able to identify 103 female patients for the questionnaire, and they all received an introductory letter explaining the purpose of our study, along with the questionnaire itself. (The study was carried out between March and June of 1992.) Ninety questionnaires were returned (response rate 87%).

It was not possible to define the age at onset of macular hole formation since for many patients the diagnosis had been made during the course of a routine eye test, and the time of onset was therefore unknown. Average age was 66 years (range 46–85 years).

The first four questions asked were as follows:

How old were you when your periods started?

How many times have you been pregnant?

How many children have you given birth to?

Have you ever used a contraceptive pill?

Patients experienced the menarche at an average age of 13 years (range 11–16 years), became pregnant 2.5 times, and gave live births on 2.3 occasions. Fourteen of the 90 patients (15%) had taken an oral contraceptive preparation at some time, for an average of 6.7 years. All these figures are similar to those for the general female population.

We then asked:

Have you ever had hot flushes?

Have you passed the menopause?

Have you had a hysterectomy, and if so were the ovaries removed also?

Sixty-three of 88 patients (71%) had experienced hot flushes at some time in the perimenopausal period, and their average age at onset was 47.6 years. Eighty-four of 89 patients (94%) were postmenopausal at the time of diagnosis of their macular hole, the average age at menopause being 50 years. Seventeen of 85 patients (20%) had undergone hysterectomy, and of the 12 patients within this group who answered the associated question on oophorec-