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Sir,
**Transient Horner's syndrome associated with
hypertension**

A 46-year-old man presented with a droopy left upper-lid associated with headache, facial, and neck pain. He was otherwise well and a nonsmoker.

Examination revealed 2 mm of left ptosis, anisocoria (see Figure 1) and facial anhidrosis. Acuity was 6/4 in each eye. The blood pressure was markedly elevated (250/140). He had hypertensive retinopathic changes but ocular and systemic examinations were otherwise unremarkable. Although index of suspicion for carotid artery dissection was low, urgent ultrasound Doppler's of the carotid/vertebral circulations and computed tomography scanning with contrast of the head/neck were performed and excluded this possibility.

A weak adrenaline solution (1:1000) was instilled to ascertain if a preganglionic or postganglionic Horner's was present. This resulted in no change in pupil size, confirming a preganglionic lesion or central Horner's syndrome.

Figure 1 Left miosis, ptosis, and facial anhidrosis.

Chest radiography and electrocardiography revealed cardiomegaly and left ventricular hypertrophy. He was referred to the medical team for further investigation/management. Subsequent echocardiography confirmed left ventricular hypertrophy. He was found to have elevated serum lipids and creatinine (at $164 \mu\text{mol/l}$). All other investigations were normal. Hypertension and hypercholesterolaemia were controlled medically and Aspirin commenced.

Although the presence of a Horner's syndrome was irrefutable on clinical grounds (Figure 1), full pharmacological testing with 4% cocaine eyedrops (unavailable in 'out of hours' practice), would have completed the investigations.¹ After 1 month, the central Horner's syndrome had completely resolved. Our patient had sustained a transient ischaemic attack affecting the preganglionic neuron in the ocular sympathetic chain. Given the absence of brainstem signs we suspect the lesion was near the posterolateral hypothalamus, the origin of the ocular sympathetic chain. This area is supplied by small vessels known as the posteromedial penetrating arteries, which arise variably from the Circle of Willis, posterior and middle cerebral arteries. Treatment involves modification of the cardiovascular risk profile—as in this case.

Although hypertension has been described with Horner syndrome, this was in relation to carotid dissection.² This is a life-threatening emergency which may manifest in numerous ways in association with Horner syndrome.³ To our knowledge this is the first reported case of transient central Horner syndrome associated with hypertension.

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Sir,
Quicker painless diabetic laser

Not often does an improvement in delivery of treatment occur because of a misunderstanding in a conversation. Five years ago the authors were at a national meeting and informally discussing pan retinal photocoagulation for proliferative diabetic retinopathy. TR was impressed that WW was able to perform this laser treatment more quickly by shortening the duration of each spot of laser from the conventional 0.1 s to 0.02 s.

TR found that at 0.02 s the automatic repeater on the (Coherent) argon laser was able to produce more than eight burns per second. Naturally the power needed to be raised to compensate for this – rarely more than 500 mW. After these faster sessions of treatment he was delighted to hear his patients ask spontaneously why the treatment was less painful than previous occasions. TR had previously found that the more laser patients had had meant that the treatment sessions became more uncomfortable. Since then TR has not needed any periocular anaesthetic injections for proliferative laser treatment.

Two years later, at another meeting, TR praised WW for his splendid tip of shortening the laser burn to 0.02 s. 'No' said WW, 'I use 0.05 s'. On returning to Glasgow WW tried setting the duration to 0.02 s and was equally pleased with its effectiveness and increased comfort for patients.

Why is treatment less painful at 0.02 s? One can speculate that the zone of heat around the burn does not go as deep and therefore perhaps has less effect on choroidal nerves. Is pan retinal laser at 0.02 s as effective as at 0.1 s? The authors cannot say for sure but it certainly seems to be.

A popular ophthalmic textbook suggests 0.05–0.1 s. A literature search on the duration of laser burns was not fruitful but a reference to a short pulse of 0.02 s causing less pain was found on the internet (www.diabeticretinopathy.org.uk). The authors therefore do not claim anything new but are keen to promote this less painful way of delivering laser treatment. They also ponder on whether the value of coffee breaks at national meetings should not be overlooked when points for continuous professional development are being assigned.

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Sir,
Reply to T Rimmer and W Wyke

We read with interest the correspondence on 'Quicker painless diabetic laser,' whereby a pulse duration of 20 ms with corresponding higher power in argon laser panretinal photocoagulation resulted in less painful treatment sessions.¹ The reduced pain during treatment is thought to be due to lower heat conduction to the choroid and sclera.