ing of glaucoma: 6% did not know they had the condition and 46% scored zero for the question "What is glaucoma." Only 5% gave a completely correct answer to this question. The mean score declined in a steady fashion with age (Fig. 1). The score was unrelated to duration of disease but was higher in those with affected relatives ($p<0.01$).

Understanding of the treatment was better: 57% knew that glaucoma could be effectively treated, 76% understood what would happen without treatment and 49% knew that treatment should be lifelong, however, 41% erroneously believed that their sight would be improved by treatment. Thirty per cent gave correct responses to all of these. It was alarming to find that only 12 of 39 patients who had undergone surgery had any understanding of trabeculectomy and 44% thought this carried no risk!

The familial nature of glaucoma was appreciated by 42%, and as expected this awareness was more frequent in those with affected relatives, ($p<0.001$).

The most revealing question concerned desire to know more about glaucoma and 67% said they did not want to learn more. Those who had scored least to the 'key' question 'What is glaucoma' were less likely to want more information; those who already knew the most wanted to learn more ($p<0.001$). Such apathy was not more frequent in older patients.

In summary, there is an astonishing lack of knowledge in glaucoma patients. Few comparisons are available but patients in Leicester[1] and Southampton[2] gave broadly comparable results. Many seem to be adopting a 'head in the sand' attitude which presents considerable difficulty if the basic hope that improved understanding leads to better compliance is true, however it is notable that in one large study a well organised education programme failed to increase medication compliance in benign hypertension.[3] Nevertheless the average glaucoma patient has a woeful ignorance of the disease and efforts should certainly be made to improve this, although this study suggests that such well meaning attempts may not be universally welcomed.

A. J. Elliott, FRCS, MRCP.
Senior Registrar in Ophthalmology,
Addenbrookes Hospital,
Cambridge.

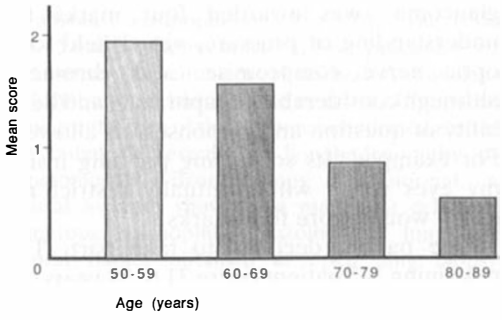

Fig. 1. *Mean score for question (a) according to age of patient.*

References

[1] Rosenthal AR, Zimmerman JF, Tanner J: Educating the glaucoma patient. *Br J Ophthalmol* 1983, **67:** 814–7.

[2] MacKean JM and Elkington AR: Alerting close relatives of patients with glaucoma. *Br Med J* 1984, **289:** 800–01.

[3] Sackett DL, Haynes RB, Gibson ES, *et al*: Randomised clinical trial of strategies for improving medication compliance in primary hypertension. *Lancet* 1975, **i:** 1205–07.

Sir,

Skin signs during giant cell arteritis, (GCA), varying from redness of the temples to frank gangrene of the scalp, occur infrequently.[1] Most reported cases of the skin manifestations of GCA have been in dermatological journals.

Having recently examined five patients where these skin signs were of diagnostic importance, two are briefly described to bring this sign to the attention of ophthalmologists.

Case 1

A male, aged 64, presented with sudden loss of right vision. Two weeks earlier he had developed a painful, rash on the temples.

Visual acuity was light perception right and 6/9 Snellen left. There was right anterior ischaemic

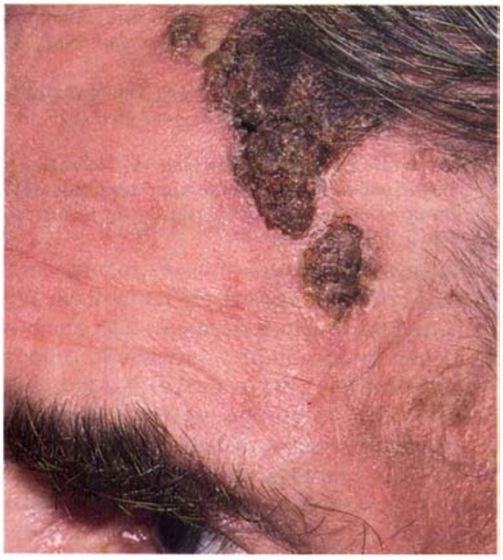

Fig. 1. *Patient 1. Crusts and ulceration on temple.*

optic neuropathy (AION). Bilateral carotid, subclavian and femoral bruits were present. Bilateral symmetrical crusting with underlying ulceration was present over the temples (Fig. 1). The temporal arteries were not pulsatile.

The plasma viscosity was 1.77 cp (1.5–1.72 cp) and Westergren ESR 19 mm/hr. Right temporal artery biopsy revealed active GCA with luminal thrombosis. Despite 60 mg prednisolone daily, left AION occurred. Vision did not recover.

Case 2

A female, aged 75, presented with left amaurosis fugax. She reported recent headaches, scalp pain and a rash over both temples. Brushing the hair was painful. She had a history of transient ischaemic attacks, hypertension, arteriopathy, and cardiac arrhythmia. Multiple medications included anticoagulants.

Visual acuity was 6/6 right, 6/36 left Snellen. There was a marked left relative afferent pupil defect (RAPD). There were several retinal infarcts in the left eye. Cardiac and carotid bruits were present. Areas of tender scalp erythema were present symmetrically over both temples (Fig. 2). There were scattered crusts overlying small superficial ulcers manifested on parting the hair. Both temporal arteries were tender and the left was occluded.

The PV was 1.75 cp and the ESR 19 mm/hr. GCA was diagnosed and systemic steroids prescribed. Temporal artery biopsy confirmed active GCA with luminal thrombosis. The retinal infarcts and left RAPD resolved and vision recovered to 6/9.

Comment

The skin lesions in Case 1 were initially thought to be herpes zoster. Similar events occurred in other reported cases.[2] Diffuse symmetrical erythema with or without ulceration occurring bilaterally, centred over the temporal arteries in GCA, should be differentiated from the painful eruption of vesicles localised to the sensory nerve root occurring in zoster.

Normal ESR levels occasionally occur in GCA. In nine cases of GCA with scalp involvement where ESR results were available; four were of less than 40 mm/hr.[2] In four out of the present five cases of GCA with scalp involvement the ESRs were $\leqslant$40 mm/h, which highlights the importance of this sign.

Scalp lesions healed as rapidly on the biopsied as on the opposite side. The possibility that biopsy might aggravate ischaemia was not encountered, despite use of a vasoconstrictor in the local anaesthetic.[3] In Case 2, the ocular and scalp perfusions improved clinically with treatment.

These acute painful, scalp lesions appearing on the elderly temples, especially bilaterally and symmetrically with visual disturbance, should arouse suspicion of GCA. They can be subtle and require parting the hair for

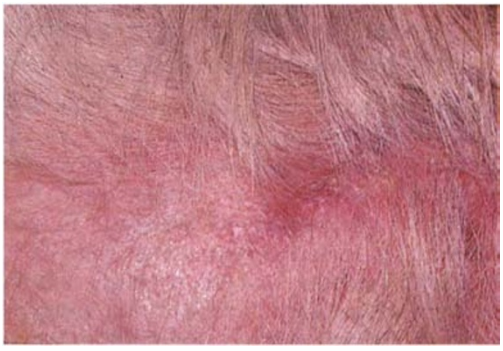

Fig. 2. *Patient 2. Left temple; an area of intense, tender, scalp erythema with early ulceration.*

exposure. Prompt identification may prevent visual loss.

S. P. Kelly, FRCS, FCOphth.
Manchester Royal Eye Hospital,
Oxford Road,
Manchester.

References

[1] Baum EW, Sams WM, Payne RR: Giant cell arteritis: a systemic disease with rare cutanious manifestations. *J Am Acad Dermatol* 1982, **6:** 1081–8.

[2] Soderstrom CW and Seehafer JR: Bilateral scalp necrosis in giant cell arteritis. *Am J Med* 1976, **61:** 541–6.

[3] Siemssen SJ, Larsen OD, McNair A: Necrotising tongue and skin lesions in temporal arteritis; follow up of a case with possible iatrogenic factor. *Br Med J* 1985, **290:** 819–20.