Some authors have postulated that the premature ageing that causes degeneration of arteries also causes the histological changes seen in the skin of ELCs [2–5].

Patients and methods. Ocular and systemic conditions were documented in 100 women and 74 men aged 55 to 90, attending general ophthalmic clinics. An 'ELC score' was obtained by grading ELC according to Patel *et al*, a value from 0 to 6 being possible for each patient. Altogether, 90 patients exhibited ELCs of all grades. A score of 3 or more was found in 29 patients; POAG was confirmed in 18 of them ($\chi^2 = p < 0.001$).

Results. No association was found with diabetes mellitus. This small survey suggests that ELC is a potentially useful aid to identifying patients at risk of developing POAG and supports the hypothesis of Patel.

Table 1. ELC in 174 ophthalmic patients

Ocular diagnosis	Patients *n*	Diabetic *n*	Mean age	Mean ELC score	ELC score 3 + *n*
POAG	35	3	74.9	2.8	18
Possible POAG	15	3	74.6	1.5	4
Secondary	7	0	74.6	0.7	0
Diabetic retinopathy	23	23	65.9	0.2	0
Cataract	29	3	71.8	0.8	2
ARM	9	0	71.7	2.1	3
ARM + cataract	3	0	85.0	0.0	0
Miscellaneous	53	4	68.0	0.9	2
Total	174	36	70.5	1.3	29

POAG, primary open angle glaucoma. ARM, age related maculopathy.

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Diagonal earlobe creases

Sir—Patel and associates (July 1992, pages 274–7) reported in a postmortem study an association between the grade of earlobe crease (ELC) and the degree of coronary atherosclerotic disease (CAD). Unfortunately the patients included in the study were all over 65 years of age and thus more prone to develop CAD. A large clinical study of a Chinese population involving 3,155 persons concluded that ELC was a phenomenon of age and had no predictive significance for CAD in the aged population [1].

The fact that the connection between ELC and CAD has not been convincingly proven in such an autopsy study does not necessarily reduce the importance of CAD risk factors in atherogenesis. The association of ELC and CAD or CAD risk factors should be further investigated. Meanwhile ELC should not be used as a clinical diagnostic sign of CAD [2].

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Management of GI haemorrhage

Sir—The article 'Guidelines for good practice in and audit of the management of upper gastrointestinal haemorrhage' (July 1992, pages 281–9) surprisingly omits mention of the diagnostic and therapeutic potential of visceral angiography [1] in selected patients with bleeding unresponsive to conservative measures.

Catheterisation should be performed in two circumstances [2]

1. where endoscopy and nuclear medicine have failed to localise a bleeding site accurately and the surgical team desires more specific information and
2. where haemorrhage originates from a known area but transcatheter infusion [3] or embolisation [4] is desired to control the bleeding. Such therapy is often definitive and may be of particular value in avoiding surgery in high risk patients [5].

Developments in imaging and catheter and guidewire construction have continued to extend the scope of diagnostic and interventional radiology. The role of these techniques in the management of upper gastrointestinal bleeding is now well established and their inclusion in any management protocol would appear appropriate.

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