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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References

1 Grierson I. What is open angle glaucoma? *Eye* 1987;**1**:15–28.
2 Frank ST. Aural sign of coronary artery disease (letter). *N Engl J Med* 1973;**283**:327.
3 Shoendfeld Y, Mor R, Weinberger A, Avidor I, Pinkhas J. Diagonal earlobe crease and coronary risk factors. *J Am Geriatr Soc* 1980;**27**:184–7.
4 Kirkham N, Murrels T, Melcher DH, Morrison EA. Diagonal earlobe creases and fatal cardiovascular disease: a necropsy study. *Br Heart J* 1989;**61**:361–6.
5 Pasternac A, Du Caitar C, Vorhe P. Correlation between dermal degenerative changes and coronary atherosclerosis. *Clin Res* 1979;**27**:193A.

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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References

1 Cheng TO. Earlobe creases and coronary artery disease: association or coincidence? *Am J Med* (in press).
2 Cheng TO. Earlobe crease and coronary artery disease. *J Am Geriatr Soc* 1991;**39**:315–6.

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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References

1. Nusbaum M, Baum S. Radiographic demonstration of unknown sites of gastrointestinal bleeding. *Surg Forum* 1963;**14**:374–5.

2. Kerlan RK Jr, Pogany AC, Burke DR, Ring EJ. Arteriographic management of upper gastrointestinal haemorrhage. *Am J Radiol* 1986;**147:**1185–8.
3. Nusbaum M, Baum S, Blakemore WS, *et al.* Clinical experience with selective intraarterial infusion of vasopressin in the control of gastrointestinal bleeding from arterial sources. *Am J Surg* 1972;**123:**162–72.
4. Rosch J, Dotter CT, Brown MJ. Selective arterial embolisation. A new method for control of acute gastrointestinal bleeding. *Radiology* 1972;**102:**303–6.
5. Lang EK. Transcatheter embolisation in management of haemorrhage from duodenal ulcer: long-term results and complications. *Radiology* 1992;**182:**703–7.

Management of upper gastrointestinal haemorrhage

Sir—I enjoyed the feature on medical audits in the July 1992 issue (pages 281–289), setting forward guidelines for good practice in and audit of the management of upper gastrointestinal haemorrhage.

The authors seem a little ambivalent as to whether the expected death rate has fallen from 10% to 4% or less in recent years. This is probably because such a figure means little unless account is taken of the patient's age and the disease which gave rise to the bleed. It seems quite hard for a fit 45 year old to die of a bleeding duodenal ulcer, whereas it may be quite difficult for an 85 year old to survive bleeding oesophageal varices.

Protocols for management of gastrointestinal haemorrhage have also assumed that resorting to emergency surgery in some cases improves the overall outcome. This has never been rigorously examined and there was suggestive evidence in Nottingham many years ago that the lower the surgical rate the better the survival.

In these days of intensive supporting facilities and specific endoscopic treatment for bleeding sites, are we not overdue for a properly randomised prospective controlled trial to see whether emergency or urgent surgery does indeed have a contribution to make?

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GI bleeding and rheumatologists

Sir—Like many rheumatologists one of our major problems and occasional causes of friction with gastroenterologists is the problem of NSAID induced gastrointestinal haemorrhage.

I note on page 283 (July 1992, pages 281–289) the recommendation that 'if the patient has taken non-steroidal anti-inflammatory drugs, this treatment should preferably be stopped. If they are essential...'.

I wish to make two points:

1. It is inappropriate for medication to be stopped without consultation with an appropriate specialist, as medication is usually prescribed for good reason and with the expectation that it will reduce symptoms. There are many alternatives to the use of anti-inflammatories and the use of physical measures to alter a patient's life style can often be effective.
2. A not inconsiderable number of gastrointestinal haemorrhages occur from the use of small doses of aspirin taken for prophylaxis of thromboembolic disease and the effects of stopping medication do not seem to have been considered.

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Who's for CPR?

Sir—Your editorial (July 1992, pages 254–7) 'Who's for CPR?' is an important contribution to the possible formulation of DNR (do not resuscitate) policies, and deals clearly with the merits and disadvantages of formal regulations compared to individual clinical judgement. However, there is an important omission—namely that the indications for CPR (and therefore the circumstances under which it is futile) demand as clear a definition as possible in order to distinguish a cardiac arrest from an expected death. Without this definition, every death is a cardiac arrest unless the (mentally competent) patient has consented to a 'DNR' order.

This may seem like a minor concern, but for terminally ill patients, the DNR conversation can be misleading and upsetting. In Canada, where DNR policies are regulated and orders cannot be written without the patient's consent, there are currently no distinctions between death and cardiac arrest. To assess patients' understanding of resuscitation conversations, we recorded the results of 33 talks with terminally ill patients who knew that they had cancer, were receiving palliative care only and that death was expected (several were on waiting lists for palliative care units). Eleven of the 33 patients answered 'yes' to the question 'In the event of your heart stopping, do you wish us to attempt to restart it?' [1]. Knowing the futility of CPR in terminally ill cancer patients, a 'yes' response puts the physician in the awkward position of either writing a 'code' order or trying to persuade the patient that CPR would be futile and cruel (in which case why is the doctor asking about it?).

There is no doubt that the prospect of cardiac resuscitation is a powerful one, and highly attractive to a patient facing death. However, studies have shown that CPR is futile for patients with terminal illness [2]: in fact the American College of Physicians guidelines specifically exclude patients whose death is expected within fourteen days from those for whom CPR is indicated [3]. As your editorial suggests, no physician is under an obligation to provide any therapy that is futile, and it is therefore important to define precisely those circumstances in which CPR is known to be futile.