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.

British hospitals used to have a useful definition of cardiac arrest which specifically excluded expected death. On the noticeboards which illustrated CPR techniques in the 1970s, the heading read 'Cardiac arrest is defined as the sudden cessation of cardiac or respiratory output in *someone who is not expected to die*'. This then raises the crucial question of who is responsible for deciding whether the death is expected, and how that is to be decided.

We are tussling with this issue in Canada and have proposed to amend DNR regulations to include four criteria defining an expected death. The criteria are:

- the patient has a diagnosed condition which is irreversible and fatal;
- no active treatment against the disease process itself is being administered or planned, and all therapy is palliative;
- the patient's death is expected in the near future (up to a few weeks);
- the patient's condition is recognised as one in which CPR is clinically ineffective, inappropriate and/or could only prolong the dying process.

The physician is obliged to hold a sensitive and careful dialogue with the patient and, with terminally ill patients, is under an obligation to explain that the treatment goals are palliative and that no further active treatment against the disease is planned. However, if all of the above criteria are satisfied, then the patient's death is an expected one and a DNR order may be written without the patient's express consent. If however, in the course of the conversation, it transpires that the patient had been expecting CPR, the physician is under an obligation to explain the situation but still does not require the patient's formal consent. If all four criteria are *not* met, then the physician *is* obliged to ask the consent of the patient before a DNR order may be written.

Some form of policy structure or of recommended guidelines in Britain will probably increase patient-doctor trust, despite initial awkwardness and difficulty in holding 'treatment objectives' conversations with the patient, but I would strongly recommend that some definition of expected death be incorporated in any future guidelines. Without it, Britain will face the same difficulties that we are now addressing in Canada.

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References

1 Buckman RB, Senn J. Eligibility of CPR: is every death a cardiac arrest? *Can Med Assoc J* 1989;**140:**1068–9.
2 Blackhall LJ. Must we always use CPR? *N Engl J Med* 1987;**317:**1281–5.
3 American College of Physicians. Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA* 1980;**244:**506.

Completing 'interim discharge letter'

Sir—Dr Fairclough and colleague's letter reported that only 16% of their discharge letters sent to GPs were complete, and highlighted the problem of achieving high quality documentation (April 1992, pages 169–71 and July 1992, page 338). We therefore undertook an audit of the last one hundred general medical 'interim discharge letters' using the fourth copy of the form: 99% had details of the diagnosis and 92% included the outpatient follow-up plans. This is a sustained improvement in quality and content of the discharge letter since its introduction 18 months ago.

We recognise though, that maintaining an improvement requires continued effort and audit cycles; at present only 9% of the forms specified whether the letter had been sent by post or was given to the patient. A significant advantage of the new structured discharge letter is that completion can be readily monitored particularly in specialties where there is a high turnover of junior staff.

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The Brown Kelly-Paterson *not* Plummer-Vinson Syndrome

Sir—I am delighted that my little paper (*Journal*, July 91, p257) should have stimulated a correspondence. Its purpose was to establish the priority of Brown Kelly and Paterson—or Paterson-Brown Kelly if one is from Cardiff—in describing in 1919 the syndrome of dysphagia (due to upper oesophageal spasm or web), glossitis and anaemia, and to point out the entirely mistaken attribution to Plummer and Vinson.

Dr Logan (*Journal*, April 92, p241) rightly draws attention to the late 18th and early 19th century writings of Baillie, Home, Monro (*tertius*) and Syme. However, their reports, perceptive as they are, are confined to dysphagia due to upper oesophageal stricture. There is no mention of glossitis or anaemia, associations which together provide the characteristic combination of features of the syndrome under discussion.

Dr Baron (*Journal*, October 91, p361), while first agreeing completely with my conclusions, then goes on to question the very existence of the syndrome. He presents evidence from two studies [1,2] that there is no association between cervical dysphagia—with or without an oesophageal web—and iron deficiency. The first [1] curiously overlooks its own documentation of lower serum iron levels in persons with webs compared to other dysphagic subjects. It also seems not to have persuaded all its authors, for in two other papers published around the same time [3,4], the association between iron deficiency and cervical dysphagia—whether cause or effect—is clearly enunciat-