($n=3$) represent a true reflection of success/failure in comparison with other groups. Indeed, there is no statistically significant difference between arterial and traumatic ulcers ($P=0.183$, Fisher's exact test) despite a 50% difference in success—undoubtedly a Type II effect.

It is particularly interesting to note that only two patients had been investigated for arterial disease and 'four had had varicose vein surgery'. We presume that none of the other patients had undergone any form of vascular or venous assessment. Hence we cannot be sure that arterial and/or venous insufficiency were not contributing factors in patients in the other groups.

We fully support the authors' call for formal assessment of the peripheral vascular status in all patients with chronic ulcers before consideration of skin grafting. After clinical assessment and the measurement of ankle brachial indices, non-invasive imaging with colour-flow duplex will allow a rapid and reliable interrogation of both arterial and venous insufficiency, and is the investigation of choice for this group of patients.

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Acute scybalous colonic obstruction and perforation

I read with interest the leading article by Shandall and Stephenson (*Annals*, July 1995, vol 77, p241) about stercoral colonic perforation, because it highlights a subject which is associated with very high mortality.

I agree with the authors that this condition is not as uncommon as previously thought, perhaps because of the increase in the elderly population.

The majority of these patients are operated on as emergencies out of hours by a trainee surgeon, perhaps not sufficiently experienced to recognise the condition and therefore wrongly labels the pathology as perforated diverticular disease.

The ideal surgical management is a slick, experienced surgeon performing a Hartmann's procedure with an abundance of peroperative peritoneal lavage. I use 8–10 litres of warm aqueous chlorhexidine solution. There is then a small chance of patient survival.

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Inpatient and post-discharge wound infections in general surgery

We read with interest the paper by Messrs Keeling and Morgan on inpatient and post-discharge wound infection in general surgery (*Annals*, July 1995, vol 77, p245). The authors imply that delayed wound infection goes undetected in hospital audit owing to recent trends in surgical practice towards shorter inpatient stay and fewer routine clinic reviews. However, this is not supported by the data presented.

The paper gives no breakdown of patient category by length of stay, and no information is given as to how many patients were day cases, discharged early or discharged after conventional hospital stay.

The authors go on to claim that patients' self-assessment of wound infection is accurate. However, they only reviewed 36 of 155 patients in clinic, which led them to reclassify five of 25 patients from intermediate/major infection to no infection (a 20% error).

We would agree that routine follow-up is unnecessary simply to document wound infection, most of which can be and is effectively treated by the patient's GP. Serious cases which need review by the surgical team will still be referred to hospital, and in addition GPs could be requested to notify the relevant surgical team if they treat postoperative wound infection in the community. A 'walk-in' wound surveillance clinic in hospital would cause considerable extra expense with no further benefit.

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Evaluation of vascular and metabolic deficiency in patients with large leg ulcers

The paper by P Balaji and John Mosley (*Annals*, July 1995, vol 77, p270) is to be welcomed and demonstrates the high quality of work with a practical outcome that can be done in district general hospitals.

I think the authors are quite correct to focus on the large leg ulcers which are the greatest problem; their results overall seem excellent.

I note with interest that in 50 of these patients there were eight in which there was no evidence of arterial or venous disease. I have a similar experience with 73 such patients in a group of 508 ulcers analysed recently (1993). I have called these non-specific ulcers, but I think most people would classify them as venous on the grounds that there is no arterial disease. However, at the same time there is no evidence of venous disease and I find it difficult to understand how a 'venous' ulcer can be produced, especially the large circumferential and often bilateral ones seen in the group without any stigmata of venous disease at all.

The authors have found significant nutritional abnormality in these patients. I did a similar survey 10 years ago in which I compared 24 patients from my leg ulcer clinic with 24 age and sex matched controls from a geriatric clinic who had no evidence of venous disease. Although significant abnormalities were found in both groups, there was no overall difference between them in respect of haemoglobin, iron, zinc and copper; in other words the distribution of abnormalities seemed to be quite random. On the grounds that there were patients with deficiencies who had no ulcers and patients with ulcers who had no deficiencies, I discounted this approach, but this paper has stimulated me to think about it again and it may be that a more closely controlled trial is indicated. Unfortunately, the authors have proved the deficiencies and have treated them but this is not evidence that these deficiencies are causal in the nature of the ulcer; this would require pretreatment measurements, a long course of treatment and post-treatment measurements, together with an assessment of the healing rate and no other interventions apart from standard bandaging.

I am somewhat staggered that these authors were able to institute a bypass graft in 15 out of the 17 patients with arterial insufficiency. Overall I find evidence of arterial insufficiency in 13% of patients referred to my clinic (although this includes ulcers of all different sizes), but I have only ever successfully grafted one old lady. The reasons for this are several: in the first instance, many of these people are very frail and I would hesitate to inflict even a femoropopliteal bypass on them. In the second place, they very often do not complain of claudication and as just about half of the ulcers can heal with conservative treatment anyway, I would think that surgery is not indicated in these cases—once the ulcer has healed, the patient has no relative complaint. It is possible that they are so frail and immobile and do not walk enough to notice claudication, but I can only recall a primary complaint of claudication in a leg ulcer patient in about half a dozen instances. The other problem arises as to the nature of the grafting. Any operation on a leg with an open portal of infection must carry a risk, but Balaji and Mosley do not mention that there are complications with the procedure and it is possibly outside the scope of their paper. I would certainly have grave hesitation in introducing a prosthetic graft into such a limb.

While I believe that the future for treatment of leg ulcers will be at the cellular and microbiological level, this may be a long time coming and in the meantime we need practical solutions to this intractable problem which has been greatly facilitated by the work in this paper.

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A 5-year audit of outcome of apicectomies carried out in a district general hospital

We read with interest the recent paper by Lyons *et al.* (*Annals*, July 1995, vol 77, p273). We are concerned, however, that the apparently high success rate in this audit, based on a very small number of cases (less than half of those actually performed), together with an inappropriately detailed analysis of results, may be giving the wrong message.

We consider that the prescription of apical surgery should take due consideration of the scientific literature relating to conventional and surgical endodontic treatment and retreatment.

The literature suggests that conventional endodontic treatment can have a success rate varying between 70% and 97%, depending on the definition of success *(1)*, that preparation technique and position of the apical seal influence outcome *(1)*, that coronal leakage can contribute to failure *(2)*, and that the prognosis for success of non-surgical retreatment, when an unfilled canal is discovered and treated, is good *(3)*. Surgical retreatment in a root harbouring an untreated canal carries a questionable prognosis *(4)*. Therefore, surgical treatment is not always appropriate or justifiable.

To suggest that "periapical sepsis is refractory to orthograde treatment, it may also be refractory to retrograde treatment", without reference to the quality of the orthograde treatment, demonstrates a lack of appreciation of the pathological processes involved in endodontic failure.

We believe that there should be key involvement of clinicians appropriately trained in restorative dentistry in the decision-making process and initial treatment for endodontic cases referred to secondary care providers. The increase in resources required for this would be offset by a reduction in surgical resource usage, and an accompanying rise in the quality of patient care.

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Some clinical aspects of reconstruction for chronic anterior cruciate ligament deficiency

The excellent long-term review of outcome after reconstruction for anterior cruciate ligament deficiency (*Annals*, July 1995, vol 77, p290) concludes that a patellar tendon graft supplemented by a MacIntosh extra-articular reconstruction is a reliable technique. The author indicates that the data supports the inclusion of the MacIntosh procedure; however, an alternative conclusion may be drawn from these figures.

Take VI presents the Lysholm scores at 1, 3 and 6 years for each operation; if an arithmetical error in the MacIntosh only group is corrected (satisfactory results at 6 years should read 10 years) it suggests that the proportion of satisfactory results remains virtually unchanged between 1 and 6 years. In the other groups in which the MacIntosh procedure has been used as an additional reconstruction, the Lysholm scores for the patellar tendon reconstruction are also well maintained for 6 years; however, the results for prosthetic ligament reconstruction progressively deteriorate, possibly an indication of fatigue failure or fragmentation of the prosthesis. It would appear that the utilisation of a MacIntosh reconstruction does not prevent deterioration of knee function if the prosthetic graft fails, but it is also unclear whether the presence of the patellar tendon graft improves the outcome of this operation. In order to address this point, it is necessary to look at the outcome of the only objective measurement of knee stability performed in this study, that is the presence or absence of a pivot shift. In Table IV, approximately half of the patients who underwent a MacIntosh repair alone, or a prosthetic ligament reconstruction with a MacIntosh repair, redeveloped a pivot shift by 6 years; however,