Letters and Comment

Contributors to this section are asked to make their comments brief and to the point. Letters should comply with the Notice printed on the inside back cover. Tables and figures should be included only if absolutely essential and no more than five references should be given. The Editor reserves the right to subedit contributions to ensure clarity

The surgery of mitral stenosis 1898–1948: why did it take 50 years to establish mitral valvotomy?

I read with great interest the recent article by Treasure and Hollman (*Annals*, March 1995, vol 77, p145) on the history of the surgical treatment of mitral stenosis. Unfortunately, they gave the wrong dates for the first successful surgical treatment of mitral stenosis by Bailey and Harken. Instead of 5 June 1948 and 9 June 1948 that were cited in the article, Bailey did his first successful mitral commissurotomy on 10 June 1948 *(1)* and Harken followed with his successful mitral valvuloplasty on 16 June 1948 *(2)*, as I discussed in my recent book on percutaneous balloon valvuloplasty *(3)*.

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2 Harken DE, Ellis LB, Ware PF, Norman LR. The surgical treatment of mitral stenosis. I. Valvuloplasty. *N Engl J Med* 1948; **239:** 801–9.
3 Cheng TO. *Percutaneous Balloon Valvuloplasty*. New York/Tokyo: Igaku-Shoin, 1992.

Peptic ulcers can now be cured without operation

We read with interest the recent article by Dr J H Baron (*Annals*, May 1995, vol 77, p168) suggesting that the ideal method of detecting *Helicobacter pylori* is the carbon-labelled urea breath test using ^{14}C or ^{13}C. Although the ^{13}C urea breath test is practical, non-radioactive and non-invasive to the patient, reports have shown false-positive results *(1,2)*. This may be owing to the presence of *H. pylori* in other parts of the gastrointestinal tract.

We agree with the author that antral biopsies with histology does not exclude *H. pylori* infection in the body when the result of endoscopy is negative. The presence of *H. pylori* has been shown to be present in the oral cavity by various methods, for instance bacteriological culture using selective medium *(3)* and polymerase chain reaction *(4)*. Other urease-producing bacteria, for instance *Actinomyces viscosus* and *Streptococcus vestibularis*, found in the oral cavity *(5)* can also rapidly degrade urea-forming ammonia and carbon dioxide. It is therefore likely that the oral *H. pylori* and other urease-producing bacteria may be responsible for the false-positive results of the ^{13}C urea breath test. We are investigating the feasibility of the urea breath test in detecting oral *H. pylori*.

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2 Johnston BJ, Celestin LR, Reitmayer R *et al.* *^{13}C urea breath test as a practical test to monitor Helicobacter pylori status. VII. Workshop on Gastroduodenal Pathology and Helicobacter pylori.* Houston Texas, 1994.
3 Cheng LHH, Webberley M, Evans M *et al.* *Helicobacter pylori* in dental plaque and gastric mucosa. *Br J Oral Maxillofac Surg* 1993, **31:** 258–9.
4 Mapstone NP, Lynch DAF, Lewis FA *et al.* Identification of *Helicobacter pylori* DNA in the mouths and stomachs of patients with gastritis using PCR. *J Clin Pathol* 1993; **46:** 540–3.
5 Marsh P, Martin M. *Oral Microbiology*, 2nd Edition. London: Chapman & Hall, pp 92, 207.

Use of split-skin grafting in the treatment of chronic leg ulcers

Skin grafting for ulcers caused by arterial disease certainly is a difficult area, and we would concur with Wood and Davies (*Annals*, May 1995, vol 77, p222) that results are often disappointing. However, their outcomes for arterial ulcers are somewhat difficult to interpret in the face of such small numbers and in the absence of any information of Doppler pressure measurements.

Collaboration between plastic surgeon and vascular surgeons is fundamental in these cases. After arterial reconstruction ulcers may heal spontaneously, or if they are large, skin grafts may then take well. A short delay between revascularisation and the skin grafting is often helpful to maximise the chance of success.

In our experience pain is the primary indication for attempts at skin grafting in this difficult patient group. If skin cover can be achieved then pain is relieved. For patients with intolerable discomfort an aggressive approach to revascularisation and skin grafting seems worthwhile.

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We read with interest the recent article by Ward and Davies (*Annals*, May 1995, vol 77, p222). They describe their experience with the use of split-skin grafts in a variety of patients ($n = 26$). With relatively small numbers, subsequent subgroup analysis can be unreliable and hence it is difficult to be sure if those patients with arterial ulcers

($n=3$) 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.