the resected specimen confirmed a squamous-cell carcinoma, with metastases in one of three mediastinal lymph nodes. Radiotherapy was not given.

He remained well until February 1972, when he developed acute bronchitis. At that time minor symptoms of oesophageal reflux were noted and barium-swallow examination showed no recurrence or any delay in gastric emptying or screening. His bronchitis persisted with the development of asthma, necessitating admission and treatment with corticosteroids in October 1975. Two months later, while still receiving steroid treatment, he was admitted as an emergency with an intrathoracic perforation of the gastric remnant (figure (a)). After intrathoracic drainage and resuscitation right thoracotomy showed two benign peptic ulcers on the anterior wall of the stomach, away from the oesophagogastric anastomosis. Both ulcers had perforated. They were excised and the stomach closed. No recurrent tumour was seen.

Chest radiographs (a) at time of gastric perforation; (b) after recovery and discharge from hospital.

The patient recovered and returned to work with a small sinus persisting in the centre of the wound (figure (b)). In August 1976 the sinus discharge increased and he was admitted for further investigations and exploration of the wound. Before surgery was undertaken, however, he developed sudden left hemiplegia and died shortly afterwards. Postmortem examination was not performed.

Comment

Survival after oesophagectomy for carcinoma is rare beyond 10 years¹; two patients have been reported alive at 13² and 31 years.³ Perforation in the gastric remnant may be benign; and this usually occurs within a year, often after radiotherapy. Radionecrosis of the stomach wall is implicated as a cause. Perforation of the oesophagogastric anastomosis has occurred into the heart one year after surgery.⁴ Recently it has been shown that if the blood supply to the mobilised stomach depends on the right gastroepiploic artery the lesser curve is relatively ischaemic.⁵

Anastomotic leakage is a complication with a high mortality rate.¹ Gastric stasis predisposes both to leakage and to benign perforation, and the importance of a drainage procedure is accepted. Other factors associated with ulcer formation in the intrathoracic gastric remnant include the use of non-absorbable sutures, injury after instrumentation, and ingestion of drugs. In our patient we can discount stasis, ischaemia, and radionecrosis. He had non-absorbable sutures at the oesophagogastric anastomosis, but the perforations were not in the suture line. He had not been subjected to any instrumentation. Probably the corticosteroids contributed to the perforation of the

ulcers, since this event occurred within the relatively short period of eight weeks from the start of treatment.

We thank Miss D Nightingale for permission to report this case, Mr D Ellis for the photographs, and Miss J S Henshall for typing the manuscript.

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Diphtheroid endocarditis after electrolysis

Diphtheroid bacilli are an uncommon, albeit recognised, cause of endocarditis, most often affecting prosthetic heart valves.¹ Lipophilic diphtheroids form a large, incompletely classified group of bacteria commonly isolated from normal skin.² We describe a young woman who developed diphtheroid endocarditis during treatment with electrolysis for facial hair. We believe this to be a unique portal of endocardial infection by an organism that is rarely responsible for endocarditis.

Case report

A 19-year-old Greek girl had had rheumatic fever when aged 11 years. At age 17 she had been admitted to a major teaching hospital for orthodontic surgery. Although she had been asymptomatic, moderate aortic incompetence had been detected on clinical examination and the diagnosis supported by echocardiography. Some three months before her illness she had started a course of electrolysis for facial hair. She had continued twice-weekly treatments until a week after the onset of the first symptoms—and then presented to her general practitioner with a two-and-a-half-week history of night sweats, rigors, increasing lethargy, and dyspnoea of effort. She also had a rash over her legs.

Her general practitioner took three sets of blood cultures over a three-day period, all of which grew the same Gram-positive bacillus. This was eventually classified as a lipophilic group I bacillus by its lipid growth requirements and sugar fermentation, according to the classification of Smith.² Because these cultures were initially reported as skin contaminants she was not admitted to hospital until the third day after presentation. The same organism was recovered from a fourth blood culture taken at the time of admission. When she was examined on admission the temperature was 38.7°C. A sparse macular rash was noted over the legs. Blood pressure was 130/40 mm Hg and pulse rate 108 beats/min. There were peripheral and auscultatory findings of moderate aortic incompetence. A triple rhythm and an Austin-Flint murmur were also present. Haemoglobin concentration was 15 g/dl and white cell count $7.9 \times 10^9/l$ ($7900/mm^3$) with 59% polymorphs, 30% lymphocytes, 8% monocytes, and 3% eosinophils. Toxic granulation was present. The erythrocyte sedimentation rate was 61 mm in the first hour. Cryoglobulins were not detected. Examination of a mid-stream urine specimen showed no abnormality.

She was started on intravenous benzylpenicillin, 4 million units four hourly, oral probenecid 500 mg twice daily, and intramuscular streptomycin 0.5 twice daily. In-vitro sensitivity testing showed the organism to be sensitive to penicillin, tetracycline, and lincomycin. It was resistant to streptomycin, and synergism to streptomycin and penicillin was not shown. A trough penicillin concentration was 19 µg/ml and, as the organism was

extremely sensitive to this concentration, formal measurement of the minimum inhibitory concentration and minimum bactericidal concentration was not performed. Streptomycin was stopped. Twenty-four hours after the start of this regimen the rigors had stopped and the temperature returned to normal. Intravenous penicillin was continued for two weeks and she was discharged home taking oral penicillin 1 g four times a day for a further six weeks. One month after discharge she was well, without any changes in the cardiovascular examination or echocardiographic findings.

Comment

While the dangers of endocarditis in susceptible patients undergoing oral or genitourinary manipulation are well recognised,^{3,4} scant attention has been given to bacteraemia that may follow compression of affected foci such as boils or epithelial damage of many other kinds.⁵ In Western society electrolysis is a common method of depilation and no consideration is given to any possible serious complications. This report emphasises that the damaged heart valve is at risk from any procedure that may produce bacteraemia. Susceptible patients should be alerted to this possible hazard of electrolysis and consideration should be given to appropriate prophylaxis.

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Oral or parenteral iron treatment in chronic ulcerative colitis?

Iron-deficiency anaemia is a common complication of ulcerative colitis, but studies of iron absorption in this disease¹⁻³ have yielded conflicting results and hence differing guidelines on the best route for iron replacement. The aims of our study were therefore to establish whether iron-deficient patients with chronic colitis have an enhanced absorption of inorganic iron comparable with that found in other iron-deficient patients, and to assess the haematological response to, and the acceptability of, oral iron treatment in patients with colitis.

Subjects, methods, and results

We studied three groups of subjects: seven normal male volunteers, 10 iron-deficient controls without inflammatory bowel disease, and 13 iron-deficient patients with chronic active ulcerative colitis. Iron deficiency was diagnosed on the basis of hypochromic microcytic anaemia with low transferrin saturation, low serum ferritin concentration, and reduced or absent bone-marrow iron stores.

The absorption of inorganic iron-59 (⁵⁹Fe) was determined in all subjects using a whole-body counter⁴ and expressed as the percentage of ⁵⁹Fe given by mouth retained at 10-14 days. The normal volunteers retained 12.3 ± 9.3% (mean ± 1SD), the iron-deficient controls 66.7 ± 30.9%, and the iron-deficient patients with colitis 66.0 ± 26.5%. There was no significant difference in the mean absorption of iron between the two groups of patients, while both groups absorbed significantly more than the controls (p < 0.001; Student's unpaired t test).

Ten of the 13 patients with colitis were subsequently treated with oral iron supplements for up to eight months. Of the remaining three, one, the only inpatient in the study, underwent elective proctocolectomy, one defaulted from follow-up, and one was only mildly iron deficient. Of the 10 treated patients, eight responded completely, their mean haemoglobin concentration rising from 10.4 to 13.4 g/dl and mean corpuscular volume from 73 to 83

fl. One patient had no response despite enhanced absorption of ⁵⁹Fe, but he had persistent active disease with bloody diarrhoea. The remaining patient showed only a partial response, her haemoglobin concentration rising from 5.9 to 9.5 g/dl, until after a hysterectomy for coexistent menorrhagia.

Seven patients tolerated oral iron preparations without ill effects. Two patients attributed mild constipation or diarrhoea to their tablets, and a further patient stopped ferrous gluconate after six weeks because of nausea but subsequently took slow-release ferrous sulphate without symptoms.

Comment

Beal *et al*¹ measured the absorption of ⁵⁹ferrous sulphate in five iron-deficient patients with colitis including four inpatients: in three absorption was above the normal range. Subsequently Ormerod,² measuring ⁵⁹Fe concentrations in blood after oral administration of ⁵⁹ferrous ascorbate, showed impairment of iron absorption in 11 patients with colitis who were sufficiently ill to require inpatient treatment. He therefore proposed that iron should be replaced parenterally in such patients, although in practice they usually receive transfusions. In a third study of iron absorption in inflammatory bowel diseases Bartels *et al*³ using external whole-body counting, found normal or increased absorption of inorganic iron in nine iron-deficient patients with ulcerative colitis. Paradoxically their patients responded poorly to oral iron treatment, and these authors also recommended parenteral iron replacement.

The present study shows that iron-deficient patients with active colitis who are not acutely ill absorb inorganic iron as avidly as other iron-deficient subjects. Thus repletion should be as effective by mouth as parenterally and without any of the hazards and discomfort associated with intravenous or intramuscular administration. The good response to treatment in eight of our 10 patients with colitis confirms this and shows unimpaired use of iron by these patients. Moreover, they generally tolerated iron tablets well: side effects could as easily have been due to the colitis itself as to iron treatment.

We conclude that iron-deficiency anaemia in outpatients with chronic ulcerative colitis should be treated with oral iron, which is well absorbed, acceptable, safe, and effective.

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Liver and spleen candidiasis: imaging and verification by fine-needle aspiration biopsy

Patients receiving immunosuppressive agents are at risk of infection, and with improved treatment of leukaemia and lymphoma and widespread use of organ transplantation more such patients are being seen. Diagnosing disseminated fungal invasion is difficult before death,¹ but aspiration biopsy with microscopy and culture may be successful.^{1,2}

We report candidiasis of liver and spleen that occurred in a patient with acute leukaemia. The final diagnosis was established by sonographically controlled fine-needle aspiration biopsy of a liver lesion.