

variable improvement in mental function, but we do not know which patients (if any) will benefit from them or for how long treatment should continue. Whether a doctor prescribes them or not is therefore largely a matter of temperament.

It cannot be overemphasised that drug treatment is only a minor part of the management of dementia. Indeed, by making the doctor feel that he is doing something, the administration of these drugs may actually deflect him from the really important tasks: providing the patient and family with sympathy, practical advice, and social support.¹³⁻¹⁵

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¹¹ Gedy, J L, Exton-Smith, A N, and Wedgwood, J, *Age and Ageing*, 1972, **1**, 74.

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¹³ Aric, T H D, *Age and Ageing*, 1977, **6**, suppl, 81.

¹⁴ Hodkinson, H M, *British Medical Journal*, 1975, **2**, 23.

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Gastric and duodenal ulceration after burns

Although ulceration of the stomach and duodenum after burns was first described by Swan in 1823 (and described by Curling in his classic account in 1842), we still know little about how this occurs. As early as five hours after burning the lining of the stomach and duodenum may show congestion, oedema, mucosal haemorrhages, and multiple superficial erosions¹—while from three days onwards true gastric and duodenal ulceration may be present. Serial gastroduodenoscopy has shown that ulceration is a more advanced stage of a disease which began soon after burning.

The condition is common. In 1965 in Birmingham a fifth of burned patients examined at necropsy were found to have ulcers in the stomach or duodenum, or both,² while in 1970 at the United States Army Burns Centre just over a tenth of all treated burned patients had clinical or necropsy evidence of this.³ In a subsequent prospective study of 54 burned patients examined by early and serial fiberoptic gastro-duodenoscopy Czaja et al⁴ found acute gastric erosions in four-fifths of patients and acute gastric ulceration in a quarter. Acute duodenitis was present in three-fifths of patients, and acute duodenal ulceration in a fifth. The frequency of ulceration increases with the total area burned: the peak incidence is 40% in those patients who have 70% of the body surface burned.³

An unexplained difference exists between the commonness of haemorrhage and perforation from these ulcers in the United States and their infrequency in Britain. Thus in Pruitt's series ulceration was clinically evident (as shown by haemorrhage) in two-thirds of the patients and showed no clinical manifestations at all in only a quarter of the cases.³ In Sevvit's series of

64 cases of ulceration noted at necropsy, on the other hand, only three were clinically evident.² This contrast persists.

Surgery is indicated for uncontrollable haemorrhage or perforation, the usual operations being subtotal gastrectomy or vagotomy and antrectomy. At operation the surgeon has to remember that gastric and duodenal ulcers coexist in 15% of cases.³

Gastric acid secretion is not increased in states of stress but it must be present for stress ulcers to form. In rats subjected to cold-induced stress pretreatment with cimetidine reduced the number of gastric mucosal erosions;⁵ however, intragastric administration of hydrochloric acid after pretreatment with cimetidine abolished this favourable effect. Hence the benefit of cimetidine could be due to a reduction of gastric acid production. Certainly clinical regimens that reduce gastric acid are considered essential in the United States for preventing haemorrhage, which typically presents about 15 days after the patient is burnt. Solem⁶ treated 109 patients with extensive but non-lethal burns with one of three regimens (intensive antacid therapy, an elemental diet, or both) all designed to reduce gastric acid, and all of these protected the patients from clinically evident ulceration—that is, from haemorrhage and perforation. Whereas the results of studies before the use of antacid therapy suggested that 14-26 of these patients would have been expected to develop clinically evident ulceration, in fact only three patients did so. Nevertheless, the regimens did not prevent clinically occult ulceration, and we need further studies to indicate their role in routine clinical practice.

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³ Pruitt, B A, Foley, F D, and Moncrief, J A, *Annals of Surgery*, 1970, **172**, 523.

⁴ McAlhany, J C, Czaja, A J, and Rosenthal, A, in *Burns—a Team Approach*, ed C P Artz, J A Moncrief, and B A Pruitt, p 512. Philadelphia, Saunders, 1979.

⁵ Levine, B A, et al, *Surgery, Gynecology, and Obstetrics*, 1979, **148**, 399.

⁶ Solem, L D, Strate, R G, and Fischer, R P, *Surgery, Gynecology, and Obstetrics*, 1979, **148**, 367.

Acquired pulmonary stenosis

Pulmonary stenosis is usually congenital but may be acquired, though sufficiently infrequently to be described in case reports.¹ The stenosis may be extrinsic, from compression of the low-pressure right ventricular outflow tract and pulmonary artery, or intrinsic, from obstructing lesions of the pulmonary valve and infundibulum of the right ventricle. The most common extrinsic causes are anterior mediastinal tumours, often lymphomas, although others include secondary carcinoma and thymoma.²⁻⁴ Any mass in the anterior mediastinum may compress the right ventricular outflow tract, and minor obstruction is not uncommon with aortic aneurysms, particularly those of the right sinus of Valsalva.² Pericardial disease may be localised, and constrictive pericarditis may present as pulmonary stenosis.⁵

The best-recognised cause of acquired stenosis of the pulmonary valve is the malignant carcinoid syndrome.⁶ The valve is rarely affected by rheumatic fever, although this complication may be more common in those living at high altitudes.⁷ Among the other intrinsic causes of pulmonary stenosis are cardiac tumours—for example, myxoma⁸—and hypertrophic cardiomyopathy. The latter affects the ventricular septum and usually presents with the symptoms and signs of obstruction of the left ventricular outflow tract. The right