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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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 erosions[1]—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 fibreoptic gastroduodenoscopy Czaja *et al*[4] 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 Sevitt'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. Solem[6] 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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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, myxoma[8]—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

ventricular outflow tract may also be narrowed, pressure gradients being found in 10 of the 64 patients studied by Braunwald and his colleagues.[8] Nevertheless, pulmonary stenosis is not generally recognised as a feature of hypertrophic cardiomyopathy, since studies on patients always concentrate on the left side of the heart.

The symptoms and signs of acquired pulmonary stenosis differ from those of the congenital form. The symptoms are governed by the underlying cause of the obstruction: thus patients with tumours often have cough, chest pain, and dyspnoea. Symptoms of right ventricular obstruction such as dizziness on exertion are less common but do occur.[1] The fact that the stenosis is a relatively recent acquisition means that the physical signs are less florid than in the congenital form. A systolic murmur is invariable, but its behaviour may be unusual—for example, in a mediastinal tumour it may become quieter during inspiration because this lifts the mass off the pulmonary artery.[3] A rise in venous pressure, right ventricular hypertrophy, and delay of the pulmonary component of the second heart sound provide further useful clues. The electrocardiogram may show right axis deviation, while the lateral chest radiograph may show an anterior mediastinal shadow. The role of echocardiography is seldom mentioned in published reports, but this technique may be invaluable in diagnosing pericardial tumours,[10] and will undoubtedly become increasingly important. The definitive investigation is catheterisation of the right heart, including right ventriculography, which should be undertaken whenever the diagnosis is in doubt so that proper treatment may be advised.

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Monitoring plasma concentrations of psychotropic drugs

At a Ciba Foundation symposium in London on 3-5 July an international group of scientists and clinical research workers met, under the chairmanship of Professor Louis Lasagna of Rochester University, USA, to discuss monitoring drug concentrations in neuropsychiatry. During the meeting a statement on the present position for the various classes of psychotropic drugs was prepared and accepted unanimously by all the members of the group:

Plasma concentrations of drugs are now determined with increasing frequency in many patients, including those who receive psychotropic medicines. Modern chemical methods permit the measurement of most drugs in biological fluids. Like other technological advances, this one has proved a mixed blessing.

Properly performed, drug assays at the very least can identify some causes of failure of patients to respond to treatment.[1] Some patients may not comply in taking medicines as prescribed or may not get adequate amounts of drug in their body for a number of other reasons; low concentrations result, and the drug may be clinically ineffective. Conversely, taking too large doses may lead to adverse effects. Drug monitoring is also valuable at the extremes of life.

The overlap of therapeutic and toxic concentrations, however, demands the application of clinical judgment in assessing a given drug concentration in a given patient. Furthermore, laboratory errors are all too common; grossly inaccurate drug levels are worse than no drug levels at all, and expensive to boot.

Even with drugs for which the usefulness of monitoring is established there are difficulties. The formidable problems involved in the drug treatment of epilepsy include the high prevalence of the disorder; the poor long-term prognosis in many; the need for prolonged treatment; polypharmacy; chronic toxicity; and uncertainty of the relative efficacy and toxicity of individual drugs owing to the poor quality of anticonvulsant evaluation.[2] Fortunately, the plasma concentrations of several major anticonvulsants appear related to their brain levels, to antiepileptic efficacy, and to some of the side effects. General plasma level ranges for effective anticonvulsant action without intolerable toxicity have been ascertained for some antiepileptic drugs, but assays are necessary only in the more severely epileptic patients. Prospective studies in chronic epileptics suggest considerable scope for simplifying and rationalising treatment by reducing the number of daily doses of anticonvulsants and by treating patients successfully with fewer drugs. In many epileptics, too, plasma concentrations are useful to detect non-compliance.

The use of tricyclic antidepressants is an increasingly common approach to the treatment of depression. Many drugs are available and recommended dosages vary. An appreciable proportion of patients fail to show a satisfactory or sustained clinical response; others suffer adverse reactions. Drug compliance is alarmingly low in many patients.

Measurement of plasma drug concentrations may increase the efficacy of antidepressant therapy.[3] In spite of many studies carried out over the past 10 years on the relationship between plasma concentrations of antidepressant drugs and clinical effects, the value of monitoring these drugs is still not clear. There is some evidence for the existence of a therapeutic range within which optimal antidepressant action appears to be obtained.[4]

Concentrations above and below this optimal range may be associated with poor therapeutic response. In some clinical states measurements may be of value—eg, patients who show a poor response, patients who experience side effects, those who have a complicating medical condition or in whom poor compliance is suspected, and for the control of long-term therapy.

Monitoring of lithium concentrations is now routine, not only to help ensure therapeutic levels in the prevention of affective swings but also to avoid adverse effects, both short term and long term. The evidence relating serious adverse effects to high plasma concentrations is now so strong as to preclude the use of lithium except when laboratory facilities are available.

The monitoring of plasma concentrations of neuroleptics and of benzodiazepines is not routine because the relevance of such monitoring to obtaining the best clinical response possible and to minimising unwanted effects remains to be