the early work on its application to clinical problems was therefore Japanese,[4 5] but the first British paper has now been published.[6] A joint study from workers in London, Bristol, and Cardiff, it reports the results of duodenoscopy in 60 patients with undiagnosed persistent jaundice, recurrent biliary tract symptoms, or suspected pancreatic disease. The descending duodenum was visualized in all the patients. The papilla of Vater was seen in 54 (90%) and was cannulated under vision in 44 (73%), in whom retrograde radiography of the biliary and pancreatic systems was performed. These figures compare well with the early experiences of the Japanese pioneers.

When the tip of the cannula has been guided under vision into the orifice of the papilla 2-4 ml of contrast medium is slowly injected under fluoroscopic control. The pancreatic duct is usually first outlined, but sometimes the biliary and pancreatic duct systems fill simultaneously. When the biliary tract is not filled with the first injection the cannula is repositioned to enter the biliary duct inside the common channel. This is done by trial and error.

Various abnormalities were recognized in the British series. Among 30 patients with persistent jaundice two had neoplastic strictures due to carcinoma of the pancreatic head, one had complete blockage of the common hepatic duct and neoplastic distortion of the cystic duct, while seven were shown to have gall stones. Among the patients with recurrent biliary-tract symptoms a variety of abnormalities was observed, including two large stones in a grossly dilated common duct in a patient in whom both oral and intravenous cholangiography had been useless. The findings in patients with suspected pancreatic disease included a simple stricture of the pancreatic duct, which was subsequently treated by surgical resection. Cannulation of the papilla of Vater was the sole topic at a recent meeting of the new British Society for Digestive Endoscopy. By then the numbers of patients in the combined British series had mounted to 108, and cannulation had been successful in about 75%.

Cannulation seems to be reasonably safe in the hands of experts. A Gram-negative septicaemia had developed in two of the 108 patients, but responded rapidly to antibiotic therapy. Transitory rises of serum amylase are common after the procedure, but acute pancreatitis has not been reported. Clearly, the clinical applications and potential hazards of this new diagnostic procedure need further study before it can be recommended for general use, but this should not take long.

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Status Epilepticus

Major status epilepticus (status epilepticus) may be arbitrarily defined as a series of two or more major convulsions without intervening recovery of consciousness or as continuous major convulsions lasting for more than an hour. The mortality rate of this grave medical emergency was 30-50% at the beginning of the century.[1 2] Powerful anticonvulsant drugs and modern techniques of cardiorespiratory support have reduced this to 3-21%.[3-7]

Status epilepticus must be clearly distinguished from serial epilepsy (frequent fits), epilepsia partialis continua, and petit mal status. About 0·5% of the population in Europe and America suffer from epilepsy.[8] Statements about the prevalence of status epilepticus in epileptics vary according to the method of selection of clinical material. As many as 8% of epileptic children have status epilepticus,[4] but in a recent series of 2,500 epileptic patients over the age of 11 years J. M. Oxbury and C. W. M. Whitty[7] found that only 86 (3%) had developed it. These figures, based on the experience of the United Oxford Hospitals from 1947 to 1967, specifically exclude status epilepticus during the acute phase of head injury. The pathological basis for status epilepticus was identified in 54 of these patients. Tumours were the commonest cause, occurring in 19 patients; vascular lesions accounted for 13, and infection for nine. In the remaining 13 patients the causes were head injury more than six years previously, congenital abnormalities (due to prenatal or perinatal cerebral damage or to cerebral anomalies), or metabolic (anoxia, drugs). No pathological cause was found in 32 of the 86 patients with status epilepticus in Oxbury and Whitty's series.

This division of patients with status epilepticus into 60% with symptomatic epilepsy (most having tumours) and 40% with idiopathic epilepsy is consistent with the experience of others.[3 5] One of the common causes of status epilepticus in patients with idiopathic epilepsy is sudden cessation of anticonvulsant therapy. Oxbury and Whitty[7] found the mean interval between the onset of epilepsy and the first episode of status epilepticus to be 18 years. Patients whose first attack of epilepsy was an episode of status epilepticus usually proved to have cerebral tumours. One or other frontal lobe was the commonest site for a single, small, focal lesion.

The mechanism whereby major convulsions lead to death or brain damage is unknown. There is evidence against the view that it is due to anoxia caused by respiratory difficulties during the seizure. M. H. Epstein and J. S. O'Connor[9] maintained adequate artificial respiration and muscle relaxation in cats subjected to prolonged cerebral dysrrhythmia induced by electric shock, leptazol, or penicillin, and despite good oxygenation all animals died within 48 hours of their seizures. Their brains were moderately swollen but there was no other visible lesion. These findings indicate that in managing patients with status epilepticus stopping the cerebral dysrhythmia is as important as coping with its effects on the cardiorespiratory system. Paraldehyde is a safe and effective anticonvulsant, though intramuscular injections may produce sterile abscesses and intravenous infusion may lead to phlebitis. For adults a deep injection of 10 ml is still advocated by some.[8] Others[10] prefer intravenous diazepan (10 mg, followed by a continuous infusion of 100 mg in 500 ml of normal saline), and several recent studies support this view. Subsequently the patient should be maintained on large parenteral doses of conventional anticonvulsants such as phenobarbitone. Occasionally thiopentone may be necessary.

Recent advances in intensive care mean that the assistance of an anaesthetist is often required. Attention should also be given to such simple but essential factors as maintaining an airway and controlling pyrexia. When there is a treatable cause such as infection appropriate therapy should be instituted. At the earliest signs of cardiorespiratory complications curarization with intermittent positive pressure ventilation is indicated. D. Janz and G. Kautz[5] emphasized that the commonest errors in the management of status

epilepticus are: giving anticonvulsants initially in doses that are too small; being too hesistant in the subsequent administration of anticonvulsants; and switching over too soon from parenteral to oral anticonvulsants. If these pitfalls are avoided the continuing improvements in supportive therapy should reduce still further the current mortality.

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Bone Disease after Gastrectomy

That metabolic bone disease may follow gastrectomy has been known since the 1940's,[1] but little has been written on the practical significance of the condition. A recent paper from Sweden[2] reported a higher incidence of fractures in men at least 20 years after a Billroth II type of gastrectomy than in men who had not had a gastrectomy. There was a threefold and highly significant increase in fractures related to reduction in the quality of bone—the so-called "fragility fractures"—in gastrectomy patients compared with matched controls. There was also a significantly increased incidence of all other types of fracture compared with the controls. The authors attribute these findings to a gradual loss of quality in bone after gastrectomy.

Difficulty in defining the terms osteomalacia and osteoporosis confuses discussion on this matter. Osteomalacia occurs when there is a loss of calcium from bone and, since it responds to physiological doses of vitamin D, is said to be due to a lack of that vitamin.[3] The symptoms of bone pain and tenderness together with muscle weakness may be confused with an arthritis or some other musculoskeletal condition. The bones are partially decalcified and pseudofractures may be seen on *x*-ray examination. Blood calcium and phosphate levels are often low and the serum alkaline phosphatase (bone) raised. Diagnosis is best established by biopsy of a core of bone extracted from the ilium by a trephine under local anaesthesia. Bone which is not decalcified shows an excess of osteoid tissue, the seams of which characteristically exceed 15 μm in width. Other, more sophisiticated measurements such as of plasma vitamin D-like activity,[4] calcium balance studies, calcium infusion tests,[5 6] and urinary hydroxyproline[7] may be made, but they are more appropriate to metabolic departments.

In osteoporosis there is actual loss of bone tissue. What remains is normally calcified. There are no associated blood changes and the main clinical feature is a proneness to fractures. Since osteoporosis occurs normally in ageing[8] the changes in bone due to gastrectomy are superimposed on and inseparable from those due to advancing years, and because of the difficulty in separating osteoporosis from osteomalacia the term "postgastrectomy bone disease" has been coined. Estimates of its incidence vary widely, but it probably occurs in 5-15% of patients some 10 years after operation.[9] Some think the incidence to be higher.[10]

Until recently no case of osteomalacia had been reported after vagotomy and gastric drainage, though there have been cases after gastrojejunostomy alone.[11 12] Long-term follow-up studies in Britain have so far failed to discover significant osteomalacia or osteoporosis after truncal vagotomy and pyroloplasty or after truncal vagotomy and gastrojejunostomy.[13-15] But in a recent study in Australia early bone changes were suspected in a few patients after vagotomy and pyloroplasty.[16] As more patients live longer after vagotomy and gastric drainage cases of bone disease will probably be seen among them too.

The recognition of postgastrectomy bone disease will usually be a matter for the general practitioner, since after 5-10 years, when the condition may appear, most of the patients will no longer be attending hospital. Estimates of blood calcium, inorganic phosphate, and alkaline phosphatase should be the first step in diagnosis, and when these are abnormal arrangements for bone biopsy should be made. In cases of gross bone disease the diagnosis will be obvious at this point, but in many patients it will remain in doubt. There should then be a thorough metabolic assessment, but when this is impossible bone disease should be assumed to exist and appropriate treatment started.

Intelligent anticipation should prevent gross bone disease developing in susceptible patients. Thus those who have had a gastric operation and whose nutrition is poor should be given calcium and vitamin D at intervals or continuously, as appropriate. The compound tablet of calcium and vitamin D, *B.P.C.*, contains calcium sodium lactate 450 mg, calcium phosphate 150 mg, and calciferol 12·5 μg (vitamin D 500 units), and one tablet should be given daily. In cases of established osteomalacia supplementary vitamin D should be given. Osteoporosis presents a greater therapeutic problem, and the response to anabolic steroids, at least in terms of the bones, is usually disappointing.

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Next Step in Scotland

While the White Papers on N.H.S. reform in England and Wales are still over the horizon, Scotland is steaming ahead with its legislation, the N.H.S. (Scotland) Bill[1] having already had its second reading in the House of Lords.[2] It largely follows the Scottish White Paper.[3] The profession in Scotland has been discussing the Service's reorganization