matic treatment until spontaneous recovery begins; this may normally be expected within three to four weeks. Recovery usually proceeds in two recognizable phases. There is an initial phase of rapid improvement, which seems to correlate with remyelination of affected peripheral nerves, and a very much slower phase in which reinnervation of denervated muscle takes place by nerve regrowth and sprouting. In most cases recovery is complete, but about 5% of adult cases have some sequelae.[3] The prognosis is less good in children.[4]

In adult cases Ravn[5] concluded that no prognostic hints could be drawn from the initial course of the disease, but a recent report[6] has attempted to correlate various clinical features with prognosis for recovery in children suffering from this syndrome. Neither implication of respiratory or bulbar musculature nor early corticosteroid treatment showed any correlation with eventual complete recovery. Those patients who still showed weakness three years after the onset of their disease were the cases in whom severe weakness of distal limb musculature had occurred. The most reliable prognostic indicator was the time interval between maximal weakness and onset of recovery. In those cases in whom improvement was delayed more than 16 days beyond the time when maximal weakness was recorded there was a 96% probability that recovery would be incomplete.

The duration of maximum symptoms can usually be reliably estimated in the Guillain-Barré syndrome, except in those cases where a chronic relapsing course develops.[7] If, therefore, these findings are confirmed they may provide a useful indicator of prognosis in children, where as many as a quarter of the cases show weakness more than three years after leaving hospital.

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Hereditary Pancreatitis

Pancreatitis is an uncommon disease in children and is usually unexplained, though trauma, mumps and other infections, ascariasis, steroid therapy, and congenital malformations of the pancreas and biliary tree are recognizable aetiological factors. Recurrent pancreatitis is probably even less common, but it may occur in cystic fibrosis, hereditary hyperparathyroidism, hereditary hyperlipidaemia of the type I (fat-induced hyperchylomicronaemia) variety, or as an unexplained familial illness. Indeed hereditary pancreatitis has been claimed to be the most common cause of recurrent pancreatitis in childhood.[1]

Sibert has recently reported a family with hereditary pancreatitis,[2] the largest kindred to be described in Britain, bringing the total of affected families to over 20 with more than 100 established cases of pancreatitis. Clinically hereditary pancreatitis is very similar to chronic relapsing pancreatitis, and patients complain of recurrent bouts of acute abdominal pain with intervening pain-free periods. The pain is typically epigastric, radiates to the back, and may be accompanied by nausea and vomiting. The biochemical features are common to those found in any form of acute pancreatitis. Eventually exocrine and endocrine insufficiency supervenes and the patient may develop steatorrhoea and diabetes mellitus.[1 3 4] Features peculiar to hereditary pancreatitis are the early onset in childhood, equal incidence for males and females, a preference for Caucasians, and the common and early onset of pancreatic calcification. The pancreatic calcifications develop within the pancreatic ducts[5] and in children must be distinguished from cystic fibrosis. Possibly patients with hereditary pancreatitis may have a predisposition to pancreatic cancer.[3]

Different kindreds have had singular clinical features. The original families described by Gross[6] had lysine-cystine aminoaciduria, but these families probably had coexisting hereditary pancreatitis and incompletely recessive cystinuria. Gastrointestinal bleeding features prominently in some families,[2 4] and a characteristic of the family described by Sibert was the cessation of abdominal pain once the patients reached 30 to 40 years of age.

The disease is inherited as an autosomal dominant with incomplete penetrance, though the basic inherited disorder is unknown. No specific biochemical defect has been identified, and while hypertrophy of the sphincter of Oddi[7] and anomalous narrowing of the pancreatic duct[8] have been postulated the evidence is not convincing.

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Antibiotic Cover for Dental Extraction

The protection of susceptible patients from bacterial endocarditis after dental extraction has been a perennial subject of controversy, but informed medical opinion has recently agreed on two precepts. These are that the antibiotic used should be bactericidal, penicillin being the obvious and usual choice, and that its administration should be begun only shortly before the operation, since pretreatment selects a resistant mouth flora.

To ascertain the current usual practice Durack[1] sent a questionnaire to 117 dentists in Oxfordshire, of whom 71 replied. All but two believed antibiotics to be of proved value for the purpose, and all but five administered them, the great majority using penicillin. Though this was given in six different forms much the most popular was oral penicillin V, 250 mg 6-hourly. For patients allergic to penicillin 33 gave tetracycline, 20 erythromycin, and 9 cephalosporins—dentists find it much more convenient to write a prescription than to stock and administer an injectable antibiotic, though patients who feel perfectly well can probably not be depended on for self-medication. Timing and duration were very variable; six started treatment three or four days before extraction, 11 two days, and 31 one day before, and the duration of treatment was anywhere between two and five days.

In fact many of these practices are plainly misguided, and it