for an infant the parents must ultimately decide"—with the doctor helping the parents to understand the choices.[5] In their responses to the present case (22 August, p 567) both the BMA and the British Paediatric Association have reiterated these principles. We believe that in the absence of a clear code to which society adheres there is no justification for usurping parents' rights, or for believing that the courts are any more likely to reach a more humane solution. We must beware of that slippery slope that would lead to the nonchalant taking of lives found to be substandard, inconvenient, or expensive; but the "existence-at-all-costs" view points to a terrain no less treacherous. Letting nature take its course in certain circumstances is to acknowledge that there might sometimes be a right not to live—but we badly need to clear our confusions about what these circumstances are.

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Pancreatic islet-acinar interactions

In current clinical practice, endocrinologists usually study and treat diseases of the pancreatic islets leaving gastroenterologists to deal with diseases of the "exocrine" pancreas. This administrative separation has, until recently, helped to obscure the functional implications of the intimate anatomical interrelation of these two parts of the human pancreas.[1] Not that islets and acinar tissue have to be anatomically associated in order to function satisfactorily: in molluscs and protochordates, for example, separate and individual endocrine cells and digestive cells are distributed in the mucosa along the alimentary tract in a "pancreatic gut," while in cyclostomes like the hagfish clumps of endocrine cells form islet organs in the wall of the bile duct but remain quite separate, anatomically, from digestive cells.[2] In mammals, however, the islets and acinar tissue have become combined within the pancreas and, indeed, apparently arise from common precursor cells.[3]

The recent recognition that the anatomical juxtaposition of islets and acinar tissue is of functional importance is based on the results of convergent studies. Animals which are spontaneously diabetic or have been rendered diabetic are known to have abnormal pancreatic exocrine function,[4] as do patients with diabetes mellitus. In rats with alloxan-induced diabetes both the synthesis and secretion of amylase are diminished but can be restored to normal by giving insulin.[5] Patients with insulin-dependent diabetes have appreciably impaired pancreatic exocrine secretory capacity[6] and morphological abnormalities of the acinar cells.[7]

In the normal pancreas the acinar tissue surrounding the pancreatic islets differs from the remaining acinar tissue by the cells being larger and having a greater content of zymogen granules, and a quite different content of enzymes, and showing both a more rapid synthesis of DNA and an increased number of mitoses.[1 8]

While the morphological basis for an intimate functional relation between pancreatic islets and acinar tissue has been recognised for a century, the intrapancreatic "portal" system of capillaries has been recognised only recently. Blood leaving the islets has now been shown to pass into the capillary meshwork within the exocrine tissue of the pancreas rather than draining directly into the pancreatic veins.[9] Studies in rabbits have shown that up to one-quarter of the total pancreatic blood flow goes to the islets and thence to the acinar capillaries before leaving the pancreas.[10] Since pancreatic venous blood contains concentrations of insulin and other hormones 20 times greater than in peripheral blood, the concentrations in the portal peri-insular circulation are presumably still higher.

The peptide hormones[11] produced by the islets influence acinar cell function. Insulin potentiates the actions of both acetylcholine[12] and cholecystokinin-pancreozymin[13] on pancreatic acinar cells, increasing both the flow of pancreatic juice and the synthesis and release of digestive enzymes, especially amylase. Specific high-affinity receptors for insulin have been shown on pancreatic acinar cells.[14] Similarly, glucagon stimulates the secretion of enzymes from the acinar cells, an effect which is potentiated by an interaction with cholecystokinin-pancreozymin and cholinergic drugs.[15] Larger doses of glucagon inhibit pancreatic secretion. Somatostatin (produced by the D cells of the islets) inhibits the function of pancreatic acinar cells,[1] as does pancreatic polypeptide, a product of the PP cells of the islets, which inhibits pancreatic exocrine secretion but also increases the synthesis of DNA in the pancreatic acinar cells.[16] Whether the insulin, glucagon, and pancreatic polypeptide which appear in pancreatic juice[17] have any regulatory function on the acinar or ductal tissues is not known. These hormones do, however, have a further wide range of indirect effects on the exocrine pancreas—for example, by modulating the intake of food. Insulin stimulates food intake,[18] and therefore (indirectly) stimulates pancreatic exocrine secretion, while pancreatic polypeptide inhibits the intake of food by its effects on the central nervous system.[19]

These interactions do not operate only in one direction, from islets to acini: important interactions probably occur among the four principal types of islet cells, since the different cell types are arranged within the islets in an ordered manner.[20] For example, somatostatin powerfully inhibits the release of insulin and glucagon; insulin inhibits the secretion of glucagon and somatostatin; while glucagon stimulates the release of the latter two hormones. Pancreatic acinar cells also have profound (albeit only indirect, so far as we know) effects on the function of the islet cells. For example, patients with exocrine pancreatic insufficiency have glucose intolerance because the islets release insufficient insulin in response to a meal, despite quite a normal capacity to do so. When the maldigestion is corrected by replacement treatment, the release of insulin and hence glucose tolerance return to normal.[21]

These indirect acinar-islet interactions depend on the "enteroinsular axis."[22] When food is broken down by the pancreatic enzymes in the intestinal lumen the pancreatic islets are stimulated to secrete hormones by the products of digestion, which elicit neural reflexes and the release of alimentary hormones from the small intestine. The most important islet-stimulant hormone is gastric inhibitory polypeptide. Gastric inhibitory polypeptide powerfully stimulates the secretion of insulin from the pancreatic islets in the presence of circulating glucose. In patients with chronic pancreatitis and exocrine insufficiency food is not satisfactorily digested; release of gastric inhibitory polypeptide from the small intestine by the breakdown products of fat and carbohydrate is therefore less than normal; and release of insulin is therefore

also less than normal, so that the patients develop hyperglycaemia. With replacement of the pancreatic enzymes digestion returns to normal, as does release of gastric inhibitory polypeptide and, in turn, the secretion of insulin, so that intolerance to glucose disappears.[21] More severe disease of the exocrine pancreas is associated with structural, as well as functional, abnormalities of the islets, ranging from hyperplasia to sclerosis,[7] with corresponding functional changes which may extend to destruction of hormone-secretory capacity. This secondary damage to the islets has been assumed to be due to "inflammation" or ischaemia, but the topic requires further study.

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Fractures of the carpal scaphoid

Generations of casualty officers have been warned to be wary of the fractured scaphoid. It is, indeed, the quiet fracture and may cause problems, especially if the injury is mismanaged. Scaphoid fractures take a long time to unite, and delayed union and even non-union are not uncommon. Avascular necrosis occurs, and secondary degenerative osteoarthrosis may follow with all the attendant pain and loss of wrist function.

The scaphoid may be fractured by minimal trauma—a fall on the outstretched hand—and may cause the minimum of symptoms and signs. In fact, these may be so trivial that the injury may be dismissed as a simple strain. This is the major pitfall: the wrist is rarely if ever strained. All such "strains" must be assumed to be a fracture until proved otherwise. Even radiography is not as reliable as usual. The standard views are not adequate. Four views at least are needed: posteroanterior, lateral, and one each in 45° of supination and pronation—and even these do not always show the fracture.

The wrist is fractured in several ways, the fall on the outstretched hand being the commonest. In the days when cars were cranked by hand a backfire was another frequent cause. The injury also occurs in victims of road accidents, and as such patients frequently have other, more spectacular injuries the fracture of the scaphoid is often overlooked.

The patient generally presents with pain and stiffness in the wrist and local swelling and tenderness in and around the anatomical snuff box. Radiographs generally show up the fracture; but if they do not any patient with these symptoms and signs should be treated as if there is a fracture for at least two weeks, when repeat *x*-ray films should confirm or disprove the presence of the injury.

The fracture occurs in three main sites in the bone—the distal pole, the waist, and the proximal pole. Healing of injuries of the distal pole is the most rapid, and of those of the proximal pole the slowest. In one-third of patients healing is further complicated by an anatomical quirk of the blood supply. These patients have scaphoids whose arterial supply enters the bone only through the distal pole, so that fractures of the waist or proximal pole are very likely to be followed by avascular necrosis of the bone proximal to the fracture. This appears in an *x*-ray film as a relative increase in density of the affected area. The white appearance is, in fact, due to the surrounding bone having lost mineral after injury while the avascular area has retained it. Eventually the affected part crumbles, and later osteoarthrosis is inevitable. Nevertheless, provided that the fractures are detected 95% of them heal when treated with simple plaster-of-Paris fixation, plastering the arm from the distal palmar crease to the elbow and including the thumb to beyond the metacarpophalangeal joint. The exact position of the thumb is not now regarded as critical, but Watson-Jones instructed that the patient should be able to hold a wine glass between the index finger and the thumb.

What about the remaining 5% of patients? They should be treated symptomatically. A patient with an ununited but symptom-free fracture requires no treatment. If he or she suffers pain or weakness in the wrist the fracture can be fixed internally with a screw or graft, or either the proximal pole or the whole bone can be excised. If severe osteoarthrosis has supervened arthrodesis affords the best chance of a pain-free, useful wrist.

Leslie and Dickson have recently reported a prospective study of 222 patients with fresh fractures of the scaphoid.[1] The fracture occurred mainly in men—190 of the series—and the commonest age group was 15-29. Time lost from work is important in this group, but unfortunately it is also the group with the worst fractures, which are slowest to unite. Many of the fractures slow to unite were those caused by the worst violence. Most fractures were visible in the first *x*-ray films. The senior house officer saw the fracture in 95% of cases; when