commonly used are gelfoam, polyvinyl alcohol (foam), and steel coils.

If the patient is not fit for surgery embolisation of the coeliac axis is an effective treatment for severe bleeding in the stomach or duodenum localised by endoscopic examination.² A good collateral blood supply makes occlusion of a single branch of the coeliac axis safe. If, however, findings on endoscopy are normal then superior and inferior mesenteric angiography is indicated for localisation. Infusion of intra-arterial vasopressin is sometimes effective in controlling this bleeding, but embolisation has been little used because of the risk of ischaemic bowel complications.³ In occasional extreme circumstances it has, however, been used safely.^{4,5}

Bleeding into the liver is often associated with haemobilia and may occur after trauma, an operation, and needling procedures as well as in various liver diseases. If after angiography the abnormal branch can be selectively catheterised then embolisation offers effective control.⁶ Oesophageal varices that are bleeding severely are a serious problem, and some centres have advocated portal vein catheterisation and embolisation when other treatments have failed.⁷ Severe bleeding may rarely occur from pancreatic lesions, and these can be effectively embolised provided the right arterial branch is adequately cannulated.^{2,8} Haemorrhage from the spleen is usually caused by traumatic rupture, and splenectomy is often necessary. Embolisation has been little used in the spleen, but it might be better as the spleen and its important immunological functions would be retained.

Severe bleeding from the kidney most commonly occurs in extensive malignant disease but may occur after an operation, trauma, and biopsy or other percutaneous procedures. Embolisation of selective branches will infarct only a small portion of the kidney and will often avoid a nephrectomy.⁹ Extensive bladder carcinoma and gynaecological malignancies may give rise to severe and persistent bleeding, and embolisation of the internal iliac artery or its branches can have a dramatic even though only palliative effect.^{8,10}

Embolisation is an effective treatment for severe haemoptysis in cystic fibrosis or other chronic inflammatory lung diseases.¹¹ In such cases the bleeding often recurs, but embolisation can be repeated with no further problems. Embolisation can sometimes be an effective treatment of severe and persistent bleeding in other parts of the body but particularly in the pelvis after trauma or an operation.^{8,12} Bleeding in some sites—such as the nose—may be catastrophic, and occasionally epistaxis is so severe that embolisation is considered as an alternative to an operation.¹³

The success and safety of embolisation depends on the skills and techniques of the interventional radiologist. Serious complications may result if adjacent structures or organs are inadvertently embolised.¹ If radiology departments are to offer an embolisation service then staff must be prepared to offer a 24 hour emergency service. At present this requirement causes real problems and such a service is at best patchy. When angiography and embolisation are available there will be many circumstances when they will be the first choice for managing severe bleeding.

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Pancreatic endocrine tumours

The rapid advance in our knowledge of pancreatic endocrine tumours must have left confused non-specialists struggling in its wake. Thirty years ago the picture was refreshingly simple. In his second edition of the classic *Companion in Surgical Studies* Aird wrote on insulinomas in the section devoted to tumours of the islet cells.¹ A small paragraph acknowledged that the pancreas might also contain inactive islet tumours, and a sentence mentioned (incorrectly) that α cell tumours of the islets might be associated with recurrent peptic ulceration. As a single author of an encyclopaedic book Aird can be forgiven for not discussing at length the seminal paper published two years earlier by Zollinger and Ellison which suggested a direct relation between non- β islet cell tumours and peptic ulceration.² Since then technological advances—notably electron microscopy, radioimmunoassay, and immunohistochemistry—have opened up the subject. So much so that our struggling non-specialist has been besieged by a plethora of novel endocrine and paracrine cells, has puzzled over putative new hormones without any apparent role, and has striven to remember the meaning of the acronym APUD (amine precursor uptake and decarboxylation) and its importance as a cytochemical means of linking together cells of a diffuse neuroendocrine system responsible for controlling digestion and carbohydrate metabolism.

Where are we now? There appear to be five types of islet cell normally present in the adult pancreas, each of which produces a distinct polypeptide or amine³: α cells produce glucagon, β cells insulin, Δ cells somatostatin, F cells (alternatively known as D2 or PP cells) pancreatic polypeptide, and enterochromaffin cells serotonin. Tumours may arise from each of the cell types, but, whereas glucagonomas, insulinomas, and carcinoid tumours usually give rise to functional upset, those that produce somatostatin or pancreatic polypeptide may be clinically silent. Unfortunately, the problem is complicated by pancreatic endocrine tumours also developing from cell types not normally resident in the adult pancreas. Such ectopically functioning tumours include those that produce gastrin (gastrinoma) or vasoactive intestinal peptide (vipoma), and even rarer forms may produce corticotrophin and polypeptides with activity like that of parathormone. These ectopically functioning tumours often produce more than one polypeptide, giving rise to a bizarre clinical picture; and in general they have a

higher potential for malignancy than tumours arising from cells normally present in the islets.

What does the non-specialist need to know about these pancreatic endocrine tumours? Given their rarity, he might answer "very little" and continue to keep his head down in the belief that they have attracted interest out of all proportion to their clinical importance. Insulinomas are the commonest tumour, but even their annual incidence has been estimated as something less than one case for every million of the population.⁴ Pancreatic endocrine tumours often, however, produce distressing and potentially dangerous functional upset, can often be cured by an operation, and carry a risk of malignant transformation that might be avoided by timely diagnosis and treatment. Even in patients with advanced malignant disease, treatment often results in many years of symptom free life.

The insulinoma best exemplifies the case for prompt diagnosis and management. Inappropriate insulin production results in neuroglycopenia, and many patients with an insulinoma are misdiagnosed as suffering from epilepsy, cerebral tumours, mental disorders, or alcoholism; our own experience suggests that on average more than two years still elapse between the onset of symptoms and diagnosis. The fault, if I can call it that, lies less often with the general practitioner than with the hospital doctor who fails to think beyond the confines of his specialty. Ironically, the diagnosis can usually be made simply by showing inappropriate circulating insulin concentrations in the face of fasting hypoglycaemia. Once the diagnosis is established the problem that remains is to localise and remove the tumour. Thorough mobilisation and bimanual palpation of the pancreas by an experienced surgeon remain the key to successful localisation of these small tumours,^{5,6} but help may be provided by selective angiography, computed tomography, and trans-hepatic portal venous sampling (to map the profile of pancreatic insulin secretion). About 90% of insulinomas are small single adenomas that can usually be cured by enucleation or distal pancreatectomy. Even in the 10% of patients with malignant insulinomas curative resection gives a median disease free survival of five years, and after palliative resection medical survival falls only to four years.⁷ Overall 10 year survival rates thus exceed 90% in many centres,⁶ although careful long term follow up is essential to detect "second primary" lesions or the late declaration of hepatic metastases.

Suspicion is the key to diagnosing pancreatic endocrine tumours. To attribute mild diabetes and anaemia to a glucagonoma requires a high index of suspicion, but the characteristic necrotising migratory erythema may give a vital clue. Similarly, diarrhoea may not immediately suggest a vipoma, but the association of watery explosive diarrhoea with hypokalaemia (and hypochlorhydria) should raise the suspicion. Fulminant peptic ulceration is more readily attributable to an underlying gastrinoma in patients with the Zollinger-Ellison syndrome, but as with all pancreatic endocrine tumours diagnosis is too often delayed until malignancy has disseminated.

An operation remains the best treatment for all these tumours, but even when metastases preclude cure worthwhile palliation can be achieved by removing tumour bulk. These tumours are slow growing and often kill because of the toxic effects of the peptides that they secrete. When cure is precluded in patients with the Zollinger-Ellison syndrome long term palliation can be achieved by suppressing acid secretion by medical and surgical means, even if this means

total gastrectomy. Cytotoxic treatment has been used with mixed success to suppress growth and activity of pancreatic endocrine tumours, but more specific measures are becoming available. For example, diazoxide can be used in patients with insulinomas to suppress insulin production, and symptomatic benefit has been achieved in patients with a variety of pancreatic endocrine tumours by reducing circulating peptide concentrations with the newly available long acting analogue of somatostatin.⁸

So the view is far from gloomy. Pancreatic islet cell tumours have advanced our understanding of physiology, are amenable to prompt diagnosis, and can be much better treated than the commoner pancreatic and alimentary neoplasms. The non-specialist should be encouraged to keep his head up if only to impress his specialist colleagues by promptly diagnosing the one pancreatic endocrine tumour he will see in his professional lifetime.

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The aetiology of preterm labour

About 5% of pregnancies end in spontaneous preterm delivery, but 70% of all perinatal deaths occur in preterm infants, particularly those with associated growth retardation. The causes of spontaneous preterm delivery include cervical incompetence, fetal abnormality, multiple pregnancy, polyhydramnios, reduced tensile strength of the membranes,¹ and maternal infections. Chorioamnionitis associated with preterm rupture of the membranes is now recognised as important,^{2,3} especially if there is colonisation with group B β haemolytic streptococci.⁴ Infection is more likely to occur if pelvic examination is performed after rupture of the membranes, but without interference the risk of chorioamnionitis does not increase in the latent period after the membranes have ruptured. Most preterm labours are not, however, associated with any recognisable cause.

Preterm labour is commoner in mothers at the extremes of reproductive life, those of low socioeconomic class, those with low weight and small stature, those who smoke,^{5,6} and those under psychosocial stress.⁷ The risk also increases with increasing parity after the second baby.⁸ The most important factor in assessing risk is the patient's obstetric history, which makes prediction poor in primigravidae. Particularly important is a history of the mother delivering prematurely, giving birth to a baby of low weight or one who died perinatally, or having had antepartum haemorrhage, cervical trauma, or an induced abortion.