

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.<sup>4</sup> 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 speciality. 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,<sup>5,6</sup> but help may be provided by selective angiography, computed tomography, and transhepatic 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.<sup>7</sup> Overall 10 year survival rates thus exceed 90% in many centres,<sup>6</sup> 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.<sup>8</sup>

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,<sup>1</sup> and maternal infections. Chorioamnionitis associated with preterm rupture of the membranes is now recognised as important,<sup>2,3</sup> especially if there is colonisation with group B  $\beta$  haemolytic streptococci.<sup>4</sup> 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,<sup>5,6</sup> and those under psychosocial stress.<sup>7</sup> The risk also increases with increasing parity after the second baby.<sup>8</sup> 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.

Several scoring systems have been developed to try to predict premature delivery—for instance, one by Fedrick<sup>9</sup>—but none is sensitive enough to identify most patients who will go into labour preterm while avoiding falsely labelling as high risk many patients who will not. Thus only a quarter of patients with a Fedrick score of five or more will go into labour preterm, but even this high risk group contains only a quarter of the multigravidas and 9% of the primigravidas who go into spontaneous preterm labour. Measurements of effacement and dilatation of the cervix in the latter half of pregnancy and uterine activity are also poor predictors.

Preventing preterm labour depends on understanding better the aetiology in cases where there are no clear contributory obstetric factors, and it may be that the aetiology is different in those whose labour starts with the membranes rupturing and in those who start with uterine contractions. Arias and Tomich found significantly higher morbidity rates in infants when labour had been heralded by premature rupture of the membranes.<sup>10</sup> Recently White *et al* studied 254 primigravidas delivered between 28 and 36 weeks' gestation and excluded those whose fetus had already died and those who had had antepartum haemorrhage, multiple pregnancy, an elective caesarean section, or an induced labour.<sup>11</sup> They compared those in whom labour was preceded by membranes rupturing (57%) with those in whom labour had started with uterine contractions (43%). When mothers of unclassified social class were excluded, there was no difference between the two groups in socio-economic class, maternal height, weight, and smoking habit. Premature rupture of the membranes was associated with babies of lower birth weight, and in the group in which labour started with contractions there was an excess of unmarried mothers and teenagers (these factors being independent) but no association with babies that were light for dates. This suggests that there may, indeed, be two different pathological processes, and further studies relating risk factors to these two modes of onset may be very productive.

Meanwhile, the clinical dilemma is when to try to defer labour. Rest in bed is the most important factor, and if the membranes are intact uterine activity will diminish and labour will be deferred in 30-40% of cases. Although cervical encirclage performed at 14-16 weeks' gestation is effective in cervical incompetence, its value later in pregnancy—for example, when multiple pregnancy has been diagnosed—has not been proved. A trial by the Royal College of Obstetricians and Gynaecologists is investigating the question. Pharmacological options include reducing oestradiol concentrations by giving betamethasone<sup>12</sup>; inhibiting prostaglandin activity,<sup>13</sup> although indomethacin may lead to premature closure of the ductus arteriosus<sup>14</sup>; and giving  $\beta$  sympathomimetic agents such as ritodrine.<sup>15 16</sup> Combining sedation and inhibition of myometrial activity with ritodrine is a logical treatment, but there is little convincing evidence of benefit.

Deferring labour may be counterproductive if there is unrecognised placental insufficiency, and looking for failing placental function, particularly by cardiotocography, is important. Anderson has suggested that  $\beta$  sympathomimetic agents to try to stop labour should be considered only if the fetus is healthy, gestational age is less than 34 weeks, the estimated fetal weight is less than 1500-2000 g, the membranes are intact, and cervical dilatation is less than 3 cm.<sup>17</sup>

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## An uncompromising report on health visiting for the elderly

The case for preventive care of the elderly is based on four propositions: (a) the elderly are often admitted to hospital; (b) they are often admitted because of a crisis; (c) the events leading up to the crisis usually have a long history; (d) therefore it would be profitable to prevent the crisis occurring. Williamson's group quantified the third proposition in their study of 200 people aged 65 or over selected randomly from three Edinburgh general practices.<sup>1</sup> Each old person had an average of three medical problems, many of them serious, of which only half were known to the doctor. Since that study was published in 1964 the case for surveillance has been made virtually irrefutable—notably by Barber in Glasgow using an annual postal questionnaire sent to all patients over 75 years old,<sup>2</sup> by three randomised trials of screening,<sup>3-5</sup> and by several other studies.<sup>6-11</sup>

Nevertheless, since the pioneering Edinburgh work case finding—the systematic search for symptomatic disease (screening looks for asymptomatic disease in addition)—has not become widespread. Does it generate too much work for doctors? Barber says that in the long run it does not.<sup>2</sup> Do old people like it? Burns showed that they do.<sup>6</sup> Are there enough professionals to do it? Barber claims that a health visitor can do the case finding in an average practice in 11-18 hours per week.<sup>2</sup> This would mean that unless health visitors stopped doing something else between 3000 and 6000 more would be required nationally.

In the latest report on the subject the British Geriatrics Society and the Health Visitors' Association, although quietly recognising that general practitioners would not welcome a national case finding endeavour, draw some startlingly bold conclusions.<sup>12</sup> Following the example of the Cumberlege report's dry disregard of doctors' likely objections,<sup>13</sup> they argue that health visiting should promote the