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trend towards mandatory continuing medical education has come to a halt."

The bland conclusion that "Continuing medical education in its various modes is a prerequisite for the competent practice of medicine" is a fair declaration of faith. What remains is not so much to prove it as to make it true, and if there are not more stimulating activities in this direction than are dreamt of in this report's philosophy, then there is something to be learnt from general practice in the United Kingdom. The key is the relation of continuing education to standards of practice—the integration of learning, and teaching with audit, so that continuing education becomes the means and the measure of improvement in the quality of medical care.

The report includes many facts about undergraduate medical schools in the United States and their finances. Overall there are two applicants for every available place, with women constituting 30% of students admitted and 25% of those currently qualifying. The drop out rate during the medical school course is only about 2%. The output of medical graduates increased by 54% in the 10 years up to 1982, but this rate of growth has now declined to about a third of that between 1972 and 1977. About 95% of United States medical school graduates take up first year residencies—almost 16 000 doctors a year. This number of first year residents is augmented by just under 5000 foreign medical graduates and a substantial number of United States citizens who are graduates of foreign medical schools.

There is little evidence of manpower planning at the post-graduate stage or of successful efforts to relate the numbers of training opportunities to the needs of the community. The generally passive approach is reflected in the comment, "The choice of location and the choice of specialty will undoubtedly be limited for many persons," and by the relative numbers of residency positions available—well over 9000 in surgery and urology, compared with under 2000 in anaesthesiology. Residency training in emergency medicine is singled out as a new specialty, with programmes first accredited in 1982.

The general picture this report presents is one of activity and awareness of problems that remain. It is always difficult to write specifically about medical education while acknowledging its interrelation with service and manpower. The report describes, for instance, current policies and attitudes on acceptance into postgraduate training programmes for graduates from foreign medical schools and United States citizens who have graduated elsewhere, but it makes no comment on the extent to which these doctors become absorbed into the practising profession and what views there are about this. The complex relations between the various bodies concerned with the control of medical education, particularly at the postgraduate stage, are well described. The importance of the autonomy of the specialty boards is rightly emphasised, and the comment that "any attempt to politicise the boards should be resisted vigorously" is well made.

There are many other chords of sympathy between the American way of thinking and our own. It is a question of how to reconcile the separateness and independence of individual specialties, the maintenance of proper educational standards, and the right of the profession to self determination and self control with the need for a system which can meet the requirements of the community. *JAMA* pleads for a single organisation—the American Medical Association Council of Medical Education—to take the initiative in identifying and addressing the major issues in medical education. Identifying and addressing issues is not, however, the same as solving problems, because the problems of medical education interlock with those

Stars and stripes—for ever?

Each year the *Journal of the American Medical Association* devotes an issue to medical education. Last December the 82nd annual report described what is now happening and what hopes there are for the future in this great American arena. The best general reading is in the section on future directions, where the comforting self assurance of good American prose clothes many right sentiments. The report speaks strongly, for example, of the danger that increasingly specialised training, even at undergraduate level, will leave too little room for the arts, the humanities, and the generality of medical practice: "If it is accepted that medicine concerns people and that knowledge of human behaviour is enhanced by an understanding of literature, art, history and philosophy" then medical schools should be selecting and planning accordingly.

How familiar the exhortations are: on the fallibility of examinations, the need to develop and assess qualities other than factual recall, the importance of social awareness and medical practice outside the hospital, the need to define the true purpose of the undergraduate course—not as a complete training but as a foundation—and the neglected art and practice of evaluation, of both students and teachers. Have they made any progress in the United States? Have we, since our own General Medical Council quoted T S Eliot to us: "Where is the wisdom we have lost in knowledge? Where is the knowledge we have lost in information?" On both sides of the Atlantic there are ways of looking at what medical schools are doing, and there are teeth that can be bared if need be; but it would have been interesting to hear more about how the desired changes are to be achieved, or even about what exciting developments are in progress.

Medical education sometimes seems to be pervaded by the spirit of the stick and the carrot. Continuing education in the United States exemplifies confusion about how to achieve the desired end—by awarding good conduct stars through schemes such as the physicians' recognition award, or by threatening various stripes for default. The *JAMA* report on continuing education has an enthusiastic account of what technology can do for informatics and, for the rest, a good many dull facts and sterile definitions. Two statements tell most of the story: "It has been difficult to document a positive correlation between a physician's participation in continuing medical education and improved patient care" and, "It is clearly evident that the

of the career structure and all the work of the profession. In this greater arena there are many interests, and the craft committees of a single hydra headed association are not inherently more likely to find sweet accordance than are quite separate bodies; but it is out of sweetness and not bitterness that strength can come.

JAMES PARKHOUSE

Postgraduate Dean and Director,
Regional Postgraduate Institute
for Medicine and Dentistry,
Newcastle upon Tyne NE2 4AB

Oesophageal varices: curiosities

The cause of oesophageal varices is usually obvious: most patients have liver disease or portal vein thrombosis, and a few have both. Very rarely, however, patients present with oesophageal varices who have neither liver disease nor portal vein occlusion,¹ and patients with occlusion of the portal vein may rarely bleed from varices elsewhere in the gut.

Obstruction to the normal portal blood flow raises the portal pressure, which provokes the opening of collateral pathways to return blood to the systemic veins. Of all the sites of collateral formation, the submucosal veins in the lower oesophagus are the most important, because it is from this site that serious and frequently fatal haemorrhage may occur. Once the portal pressure becomes greater by 10-12 mm Hg than the pressure in the vena cava then the risk of haemorrhage becomes substantial, though above this level there is no linear relation between pressure and either the severity or the frequency of bleeding.² Nor is there a linear relation between portal pressure and the size of varices, but the risk of haemorrhage is greatest with large varices.

Injection studies of the normal structure of the veins in primates show a unique venous plexus situated beneath the mucous membrane and superficial to muscularis mucosa in the distal oesophagus.³ This plexus predisposes to the formation of large submucosal collateral veins. Transection of the oesophagus with a circular staple gun provides histological specimens which show that the lumen of the oesophagus and submucosal vein are separated by only a few layers of epithelial and endothelial cells.⁴

The oesophageal hiatus through the diaphragm represents a watershed between the positive intra-abdominal pressure and the lower intrathoracic pressure. Each inspiration produces a pressure gradient which encourages the development of collateral vessels and blood flow in them. Hence not surprisingly recurrence of varices in the oesophagus is frequent after direct surgical attempts to deal with them whether by transection, devascularisation, or even resection.

Occasionally submucosal varices at other sites in the gut are responsible for serious bleeding in patients with portal hypertension—nearly always those who have had surgery for other conditions and are left with intra-abdominal adhesions. Such adhesions may become vascularised; bleeding from caecal varices may occur after previous appendicectomy, and enormous varices may develop around an ileostomy or colostomy.

The obstruction to normal portal flow is most commonly due to liver disease. The block may be presinusoidal, sinusoidal, or postsinusoidal, its site depending on the disorder present.⁵ Occasionally thrombosis or webs of the vena cava may cause

obstruction of the hepatic vein, which leads to portal hypertension (the Budd-Chiari syndrome). Extrahepatic (presinusoidal) obstruction of the portal vein due to thrombosis may occur at any age. In children there is sometimes a clear history of umbilical sepsis or exchange transfusion, though in many patients no cause is found. In adults portal vein thrombosis is often associated with blood dyscrasia such as polycythaemia or it may follow splenectomy done for such conditions. Thrombosis sometimes occurs in the portal vein of patients with established portal hypertension from liver disease.

Occasionally slow growing tumours of the pancreas such as cystadenoma or malignant endocrine tumours produce sufficient obstruction to the splenic vein to cause oesophageal varices and troublesome bleeding. Varices may develop in patients with extensive carcinoma of the body of the pancreas but the rapid progress of the cancer usually overshadows any problem of bleeding varices.

Another rare cause of portal hypertension is the presence of a portal arteriovenous fistula⁶; portal pressure rises because the portal inflow exceeds the outflow capacity. Most reported cases have originated from trauma, and the arteriovenous fistula has usually been present for more than four years before it produces splenomegaly and varices. In the first year or so after injury patients present with colic and gastrointestinal bleeding, which is attributed to congestive enteritis. Whether or not a high portal flow secondary to massive splenomegaly (Banti's syndrome) ever produces varices as such is still debated.⁵ These patients nearly always have some fibrosis of the portal tracts, and it is not clear whether this is the cause or the effect of the portal hypertension. Varices confined to the upper oesophagus have been reported in a patient with superior vena cava obstruction secondary to idiopathic mediastinal fibrosis.⁷ In such cases the portal system is presumably decompressing the congested azygos system in the chest.

Most curious of all is a rare group of patients with bleeding oesophageal varices in whom no evidence can be found of liver disease, portal vein occlusion, or vena caval occlusion.⁸ Possibly in such cases the varices may result from a congenital weakness in the lower oesophagus—a hypothesis supported by the presence of the condition in a pair of monozygotic twins.¹⁰ Treatment of these rarities should probably be limited to local treatment directed to remove or obliterate the varices.

JOHN L DAWSON

Consultant Surgeon,
King's College Hospital,
London SE5 9RS

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