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Bleeding oesophageal varices

In cirrhotic patients bleeding from oesophageal varices is a critical event. About half of those admitted to hospital will die.[1 2] When a cirrhotic patient is admitted with gastrointestinal bleeding, the precise diagnosis must be made by fibreendoscopic examination because about one-third of patients are bleeding from lesions other than their varices.[3 4]

There are three main principles of management: (1) Adequate transfusion to replace blood loss. (2) Measures to minimise encephalopathy; purgation and neomycin are commonly used. (3) Efforts to control bleeding. The methods which are used to control bleeding vary from hospital to hospital. This variation is related to several factors—for example, availability of resources and the variation in the type of cirrhosis seen in different parts of the world. Variation between patients certainly makes comparison of the results obtained in different countries very difficult.

Temporary control of haemorrhage is nearly always possible with a modified Sengstaken tube, but it requires skilled nursing.[5] Unfortunately bleeding again is common within a few days of the tube being deflated or removed, or both. The infusion of pitressin either systemically or regionally also causes temporary cessation of bleeding, but bleeding again is common when the treatment is stopped.[6] Many different operations have been used to stop bleeding. Most of the patients in whom operative treatment is considered are the hard core who have bled repeatedly. They often have advanced cirrhosis. It seems reasonable (though unproved) to perform the least traumatic operation that will control bleeding. Perhaps the most attractive of these procedures is the direct injection of sclerosant solution into the varices through an endoscope; the published results are encouraging.[7] The other operations entail either laparotomy or thoracotomy. They vary in complexity but all place considerable demands on the patients. The operations are aimed at obliterating or removing the varices. The long-term rate of further bleeding is high. A few surgeons favour emergency shunt operations despite the immediate high mortality rate,[8] and one advantage of this policy is that any survivors have a long-term protection against further haemorrhage.

Whatever operative treatment is used the outcome largely depends on the severity of the patient's liver disease. For patients with jaundice, ascites, and encephalopathy gastrointestinal bleeding is usually an agonal symptom. The vital importance of good hepatic function is emphasised by the comparatively small risk to life in those patients with virtually normal livers who bleed from varices secondary to extrahepatic portal obstruction. In recent years there has been a reappraisal of the place of both elective and emergency surgery in portal hypertension. When shunt surgery was introduced over 30 years ago it was taken up enthusiastically. Any patient who survived a haemorrhage from oesophageal varices was usually advised to undergo a shunt operation to prevent further bleeding. It is only recently that this advice has been questioned. Recent trials show that, although shunt surgery may prevent further bleeding, it hastens the onset of liver failure. The net overall results show little advantage from elective operation.[9]

Opinion on the most effective method of controlling acute haemorrhage from oesophageal varices is at present confused. With the data available it is not possible to say that emergency surgery saves life: it may only alter the mode of death. The need for an adequate prospective controlled trial is obvious. Conservative treatment alone should be compared with various surgical procedures. Perhaps sclerotherapy or emergency transection might be the first treatment to be tested.

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Teaching of anatomy

Any surgeon may reasonably be expected to know the map of the territory he is about to enter before he is allowed to embark on a voyage of exploration within the human body. Indeed, one of the most unpleasant experiences in the operating theatre is to watch a man who has lost his way at the bottom of a deep dark hole. But it is not only the surgeon who needs to know the fabric of the human body: the neurologist, radiologist, and indeed every clinician apart from the psychotherapist employs anatomy in his everyday practice. It is alarming, therefore, that there is now real concern about the teaching of anatomy in Britain, reflected in a one-day conference held at the Royal College of Surgeons of England earlier this year, when both professional anatomists and surgeons discussed what is fast becoming a crisis.

Recent years have witnessed a progressive erosion of the time that our medical educators have assigned to anatomy in the undergraduate curriculum. But the revolt against the minutiae of anatomy, which once formed such a major part of preclinical teaching, seems merely to have resulted in the replacement of unnecessary topography by equally detailed biochemistry and subcellular anatomy—which probably have just as low an educational and vocational value as the finer points of the ossification of the sphenoid. In one new medical

school the time devoted to topographical anatomy has been reduced to 160 hours, whereas even 250 hours may be insufficient to present the topographical and living anatomy which the medical student needs in clinical practice, together with another 180 hours for such topics as neuroanatomy, embryology, histology, and cell biology.

The fact that there has been a sharp reduction in the amount of gross anatomy taught to undergraduates means that young people entering surgical training today, no matter how keen and intelligent they may be, inevitably possess less topographical anatomical knowledge than their predecessors of 20 or 30 years ago. Few trainees are going to apply themselves to the brain-bursting task of learning surgical anatomy unless they have the incentive of being faced by a stiff practical examination in this subject, and this must be at an early stage of postgraduate training.

The Royal Colleges of Surgeons ensure the maintenance of anatomical standards, as well as adequate knowledge of the other relevant basic sciences, by the hurdle presented by the primary FRCS examination. This exists also in many of the Commonwealth countries and South Africa, and spreads by the influence of the college examinations to many surgeons in training from the Indian subcontinent, the Middle East, Africa, and elsewhere. It is a matter for regret that anatomical training for surgeons is a neglected topic in many other parts of the world, where teachers look on with envy at our standards.

Though the examination may insist on a high standard, many candidates who present themselves are ill prepared, and the failure rate is depressingly high. Excellent courses are available, including the basic science course organised at the Royal College of Surgeons, but the best training a young man can obtain is a period as a demonstrator in an anatomy department, an experience that will prove invaluable throughout the rest of his surgical career. Perhaps one of the most serious problems to be highlighted by the conference was the approaching crisis in the recruitment of medically qualified teachers into our anatomy departments. Much of the difficulty is due to the poor pay in contrast with comparable posts in the NHS. Unless we are prepared to follow the American lead and to accept PhDs as anatomy teachers we shall have to pay more to encourage our colleagues to take up this important teaching and research discipline.

Immunological factors in pre-eclampsia

The cause of pre-eclamptic toxaemia (PET) remains the great enigma of obstetrics. Though careful management reduces the risks greatly, it is still a major cause of maternal and perinatal mortality. In the days when advocates of the various hypotheses had fierce public exchanges it was known as "the disease of theories." As their ammunition was mainly moulded out of prejudice, hearsay, and illogical conviction it was little wonder that almost nothing in the way of agreement or advance emerged. Today matters are different, and systematic studies by groups of epidemiologists, physiologists, pathologists, endocrinologists, coagulation experts, and others have shown that the disease is associated with many and profound alterations in the maternal state. There is no clear evidence, however, that any of these abnormalities is the primary event in the disease process. Long before the classical signs appear a difference can be detected between those women who are destined to go through pregnancy normally and those who will develop pre-eclampsia.[1–4] Gant and his colleagues in Dallas, who have been responsible for much of this work, have recently suggested that some of these changes detectable before the overt manifestations have an immunological basis.[5]

Most contemporary workers have judiciously refrained from speculation about how the various disorders may be interrelated in the production of the disease, no doubt having in mind the efforts of their predecessors. Formerly, strong claims were made for the uterus as a vital organ for initiating the process,[6] but these are less frequent today,[7] though in a series of elegant studies Brosens, Robertson, and Dixon have indicated that there are detectable abnormalities in the myometrial vessels and in their permeation by the trophoblast.[8–10] Crucial to the case for a vital initiating role of the uterus is whether advanced extrauterine gestation is associated with pre-eclampsia. In such a rare condition the evidence is essentially fragmentary, but it has steadily accumulated and at a recent tally there were no fewer than 29 instances of the coexistence of PET with ectopic gestation.[11–12]

Pregnancy is a homograft, and our knowledge of transplantation immunity has provided strong grounds for considering that immunological mechanisms may be the basis of a systemic disease occurring in these circumstances. The reason that more evidence has not accumulated on this theory almost certainly relates to the discovery of rhesus isoimmunisation. In rhesus disease the primary sensitising stimulus comes from an early pregnancy and the disease is manifest in a subsequent one. This is in obvious contrast to PET, which is commoner in first than later pregnancies. In fact, however, the extreme simplicity of the rhesus model is an immunological exception, and in many other diseases the processes concerned are highly complex. The discovery of immunological factors in hypertension[13] and in glomerulonephritis,[14] both diseases with obvious similarities to the pre-eclamptic syndrome, was therefore of particular relevance.

In a new generation of studies of immunological aspects of PET Stevenson and his colleagues[15] tried to assess the degree of fetal antigenic challenge by investigating consanguineous marriages (in which it should be low) and dizygous twin pregnancies (in which it should be high). Next, contrary to earlier reports, came evidence of immunoglobulin depositions in the placenta and kidneys of patients with PET.[16 17] Thomson and his colleagues have recently reported in the *BMJ*[18] a study of the complement system based on the possibility that PET, like nephritis, might be an immune complex disease. They interpreted their findings as evidence against circulating immune complexes being concerned in the pathogenesis of PET. It seems that in conditions of immune tolerance (which pregnancy may represent) antigen-antibody complexes probably activate complement minimally, if at all.[19] Anticomplementary activity was found in four out of five severe cases with raised concentrations of C3 proactivator; the importance of these changes is not clear. They noted that a maternal immune response with both cell-mediated and humoral immunity is a feature of normal pregnancy, thus raising the possibility that in first pregnancies (primary stimuli) this response may be inadequate, resulting in PET. Another recent study supporting this concept showed that in mild cases of PET there is diminished spontaneous lymphocyte transformation.[20]

With this broader outlook the role of the immune processes in PET should soon become clearer. In interpreting further studies it will be wise to bear in mind that fetomaternal incompatibility on the ABO blood group system may be