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

beneficial, since it protects against rhesus isoimmunisation.[21] Possibly other immune processes are protective and beneficial in pregnancy. Furthermore, while the chances seem high that any immune disturbance has a primary place in the chain of events, possibly immunological processes are also implicated at some other point—for example, after tissue damage has been caused. Finally, we must remember that, as in diabetes, all cases of PET may not be caused in the same way.

[1] Gant, N F, *et al, American Journal of Obstetrics and Gynecology*, 1971, **111**, 555.
[2] Gant, N F, *et al, Endocrinology*, p 1026. *International Congress Series No 237*, Amsterdam, Excerpta Medica, 1972.
[3] Gant, N F, *et al, Journal of Clinical Investigation*, 1973, **52**, 2682.
[4] Gant, N F, *et al, American Journal of Obstetrics and Gynecology*, 1974, **120**, 1.
[5] Gant, N F, *et al, Obstetrics and Gynecology*, 1976, **47**, 319.
[6] Sophian, J, *Pregnancy Nephropathy*. London. Butterworths, 1972.
[7] *Lancet*, 1975, **2**, 487.
[8] Brosens, I, Robertson, W B, and Dixon, H G, *Journal of Pathology and Bacteriology*, 1976, **93**, 569.
[9] Brosens, I, Robertson, W B, and Dixon, H G, *Obstetrics and Gynecology Annual*, ed R M Wynn, p 177. New York, Appleton-Century-Crofts, 1972.
[10] Robertson, W B, Brosens, I A, and Dixon, H G, *American Journal of Obstetrics and Gynecology*, 1973, **117**, 294.
[11] Paterson, W G, and Grant, K A, *Obstetrical and Gynecological Survey*, 1975, **30**, 715.
[12] Paterson, W G, *British Journal of Obstetrics and Gynaecology*, 1976, **83**, 336.
[13] Mathews, J D, Whittingham, S, and Mackay, I R, *Lancet*, **2**, 1423.
[14] Peters, D K, and Lachmann, P J, *Lancet*, 1974, **1**, 58.
[15] Stevenson, A C, *et al, Lancet*, 1971, **2**, 1286.
[16] Petrucco, O M, *et al, British Medical Journal*, 1974, **1**, 473.
[17] Tribe, C R, Smart, G E, and Mackenzie, J C, *British Medical Journal*, 1974, **2**, 335.
[18] Thomson, N C, *et al, British Medical Journal*, 1976, **1**, 1307.
[19] Humphrey, J H, *British Medical Bulletin*, 1976, **32**, 182.
[20] Petrucco, O M, *British Journal of Obstetrics and Gynaecology*, 1976, **83**, 245.
[21] Woodrow, J C, and Donohoe, W T A, *British Medical Journal*, 1968, **4**, 139.

Symptomatic care in motor neurone disease

Motor neurone disease (MND) is an uncommon but devastating disease characterised by progressive wasting and weakness of the arms and spastic weakness of the legs, associated with progressive dysarthria and increasing difficulty in swallowing and breathing.[1] Two-thirds of those affected are men, many in the prime of life.[2] Guanidine may modify the course of the disease,[3] but no generally satisfactory treatment is available. Well-documented spontaneous remission is rare, and, though some patients have lived for more than 15 years after diagnosis, most die within three.[4]

The thought processes are unimpaired and the patient is fully aware of his increasing disabilities. The continuing deterioration lowers the morale of the patient, his family, and those caring for him.[5] Doctors will therefore welcome a recent article about the symptomatic care of patients with MND.[6] Smith and Norris emphasised that, despite his inability to treat the primary disease, the physician can treat symptomatically many of the problems. Help must, however, be given quickly: many patients spend their last years waiting for one thing or another which arrives too late to be of value.[7] Close co-operation is needed among neurologist, general practitioner, and medical social worker.

Difficulties in getting about are best met by providing an electric wheelchair early. Patients should use this to avoid fatigue, reserving their limited strength for occasions when a wheelchair is impracticable. Physiotherapy helps to reduce disuse atrophy and to prevent contractions. Exhaustion should be avoided, so that regular brief exercises are preferable to attempts at long periods of activity. At home relatives can be instructed to carry out exercises daily over the full range of movement. When wrist-drop develops, a splint may help maintain grip; foot braces should be obtained only if the disease appears to be following a relatively benign course. It is unusual for patients to develop bedsores unless there is gross neglect[8]—possibly because sensation and bladder control remain intact—but proper nursing care is still essential. Cramp is common and may be exacerbated by overactivity. Sometimes this can be relieved by heat or massage, but muscle relaxants such as baclofen or diazepam are often necessary, though the patient may dislike them if they also cause increased flaccidity. In such circumstances, quinidine, phenytoin, or calcium should be tried.

Progressive bulbar palsy results in gross disturbance of speech. At first the voice tires easily and becomes rasping and, later, monotonous and nasal. Eventually the patient can make only inarticulate noises. Letter and word cards may help, but a specially adapted electric typewriter allows greater freedom of expression and reduces the psychological burden that accompanies anarthria. A few exceptional patients have communicated by blinking in morse code or by shining a light attached to their foreheads on phrases on a screen placed in front of them.[9]

At first difficulties in swallowing are encountered with liquids, later with solids. As dysphagia increases the patient may need much help with eating to avoid aspiration. Though the tongue and lips may be appreciably affected, the oropharynx frequently continues to function relatively normally, so that the patient can swallow food more readily if it is placed on a spoon and deposited at the back of the mouth.[2] In undernourished patients 15 mg of neostigmine bromide by mouth 30 to 45 minutes before meals may temporarily improve food intake. Cricopharyngeal myotomy and cervical oesophagostomy, though recommended in certain patients by Smith and Norris,[6] are not generally appropriate; nasogastric tubes should also be avoided. If sialorrhoea (drooling) is a problem anticholinergic drugs such as belladonna, propantheline, or amitriptyline should be prescribed. If the problem seems intractable transtympanic neurectomy under local anaesthesia or irradiation of the salivary glands should be considered.[10]

Sexual relationships may present problems, particularly in younger men. Potency is commonly retained even when much of the power in skeletal muscles has been lost.[11] Given the opportunity, patients reveal a great deal of tension and anxiety on this score. If marital relations are to be maintained, it needs great mutual understanding between the patient and his wife, and it is often helpful for them to be able to talk to a doctor about the problems.

As the patient's disabilities increase frustration may lead to anger and anxiety compounded by depression. Both rage and tears must not only be expected but also accepted by the family and those in attendance. This commonly throws an increasingly intolerable strain on the wife and family if the patient is at home, and temporary or permanent admission to hospital may become necessary. Depression is best combated by maintaining real interest in the patient's life and by encouraging all possible expressions of his attitudes and concerns. If non-pharmacological measures fail to control these symptoms antidepressives or tranquillisers should be tried, though these do not always help.

Terminally, patients are usually bedridden. Opiates in small regular doses (for example, diamorphine and cocaine elixir (*BPC*), 2·5 mg four-hourly) are often necessary to