there had to be a special training for the GP analogous to that of the specialist.

The need for specific training was echoed by the conference after a series of papers on the present place of the GP in the health care systems of the Nine and their common trends and future needs. Despite considerable differences in the current state of postgraduate training in the member States, UEMO was able to agree unanimously that by 1985 all those wishing to practise as general practitioners should have carried out a formal period of at least two years' postgraduate specific training for general practice, including elements of training both in hospital and in general practice itself. In the light of the obstacles that differing academic, political, and ethical attitudes posed to such an agreement this can be regarded as a major step forward within the community.

When most countries in the Western World, and certainly the Nine in the EEC, are concerned with the rising costs of health care, the cost benefits of developing general practice in the health care systems have to be examined very carefully. The second day of the symposium looked at these economic consequences and showed how much research is still needed in this area. Nevertheless, as one economist said, "Even if in hard cash terms we cannot show an overall saving we cannot disregard the human benefits which accrue from the care of a patient in his own surroundings by a family doctor." National social security organisations, as the main source of money for general practice in the EEC, need to promote high standards, but the problems inherent in training programmes may have accounted for the delays in some countries. With good will on both sides the close co-operation needed should prove possible.

Clearly, much has yet to be done to achieve the full flowering of general practice over the whole of Europe; but, with the Council of Europe's recommendation on the training of the general practitioner and this strong lead from UEMO, doctors in the European Economic Community appear at least to have made a start.

Looking inside arteries

Vascular surgery is a highly skilled, technically demanding specialty: even a minor error may lead to thrombosis at an anastomosis or suture line, possibly resulting in the loss of a limb or in death.

In general, operations on the blood vessels are possible only because atheroma is a patchy disease, so that the healthy sections of artery can be joined by a bypass of the diseased segments or an endarterectomy. Yet however painstaking the surgeon's techniques, some vascular procedures fail within a month of operation, and most of these failures are thought to be caused by technical errors. The two common causes of early postoperative thrombosis are stenosis at the anastomosis and loose flaps of intima that project into the blood stream. Surgeons have tried many methods for detecting such faults during the operation so that they can correct them. For example, stenosis can be detected by measuring the blood flow through the artery: a rate of flow below 60 ml min in a saphenofemoral bypass is associated with a high chance of early thrombosis.[1] A low rate of flow does not, however, indicate the exact site of the obstruction; nor can flowmeters detect non-stenosing flaps of intima.

At present most surgeons believe that operative arteriography is the best way of confirming the technical quality of a vascular operation. Some perform an arteriogram at the end of every operation, while others do it in selected cases. If arteriography is performed on every occasion 10-15% of patients will be found to have a remediable abnormality, correction of which will improve the early patency rates. On the other hand, good quality arteriography requires a special operating table, an *x*-ray machine in the operating theatre, and a radiographer—all expensive and not always available.

An attractive alternative is for the surgeon to inspect the inside of the arteries with a small endoscope. The first clinical studies of the value of arterial endoscopy were by Pinet[2] and Vollmar,[3 4] who tried to look inside blood-filled unclamped vessels by perfusing them with saline. The perfusing technique, however, makes the procedure messy and difficult, and it has not become popular. Towne and Bernhard[5] have recently described their experience of arterial endoscopy without perfusion in clamped vessels at the end of arterial surgery. The inside of the vessel was easy to see, and they found many small flaps of intima and pieces of thrombus that they considered worth removing. Unfortunately the endoscope cannot measure the size of an anastomosis and so cannot detect minor degrees of stenosis. They tried both stiff and flexible instruments, none of which were specifically designed for arterial endoscopy, and found the rigid instruments best, though all needed modification and further development.

Arterial endoscopy seems unlikely to replace operative arteriography, but, if the instruments can be further developed, so making possible the removal of loose strips of intima, they may improve the early results of arterial operations.

[1] Little, J M, *et al*, *Lancet*, 1968, **2**, 648.
[2] Pinet, F, *et al*, *Presse Médicale*, 1966, **74**, 2351.
[3] Vollmar, J F, and Junghanns-Heidelberg, K, *Langenbecks Archiv für Klinische Chirurgie*, 1969, **325**, 1201.
[4] Vollmar, J F, and Storz, L W, *Surgical Clinics of North America*, 1974, **54**, 111.
[5] Towne, J B, and Bernhard, V M, *Surgery*, 1977, **82**, 415.

Typhoid and its serology

Serology plays a minor part in the diagnosis of enteric fever: at times it can help, but at other times it may confuse. Fortunately there are few opportunities to study the serology of typhoid on an epidemic scale in Britain, but Brodie's extensive investigations of the 1964 Aberdeen outbreak[1 2] have recently been published. These highlight the clinical limitations of serology.

The commonly used Widal test reaction is an agglutination test using bacterial suspensions of *Salmonella typhi* and *S paratyphi* A and B, treated to retain only the somatic (O) or flagellar (H) antigens. These are used to detect the corresponding antibodies in patients' serum. The earliest serological response in acute typhoid fever is said to be a rise in the titre of the O antibody. The H antibody usually develops more slowly but persists longer than the O. Towards the end of the first week of the illness titres of either antibody may be as high as 1:160, but paired sera taken four to five days apart give

more reliable information, as in an acute infection they should show an obvious rise.

There are, however, several causes of confusion in interpreting typhoid serology. Previous TAB immunisation and earlier infections with salmonellae sharing common O antigens with the enteric fever group may have stimulated antibody secretion; patients from communities where typhoid is endemic have higher H-antibody titres than do those not previously exposed to the antigen. Some patients, including some of those previously immunised, show a poor or negligible antibody response to active infection.

S typhi and *paratyphi* C possess an additional antigen, the Vi antigen (so called because of its association with virulence for mice, not man). Vi antibodies are evoked by acute *S typhi* infections; their disappearance is often taken as evidence that the organism is no longer present, though they may not be detected in known excreters of *S typhi*. A report[3] from the Public Health Laboratory Service in 1961 showed that only 70% of known carriers had Vi antibodies at a titre of 1:5 or more.

During the Aberdeen outbreak 507 cases of typhoid were notified and 403 were confirmed by culture. Widal tests were performed on patients' sera for up to two years after discharge from hospital. H antibodies could not be detected in either the acute illness or during follow-up in 15% of patients tested, 83% of whom had bacteriologically proved typhoid; and O antibodies did not develop in as many as 41%, 79% of whom had bacteriological confirmation of their disease. Failure to produce Vi antibody at any stage was seen only in 9%, and in 33 patients only Vi antibodies were present.

One hundred and eleven of the group had had TAB immunisation, but this seems to have made little difference to antibody production. For O antibodies about 60% of the non-immunised patients showed a response at some stage, compared with 54% of those who had been immunised. For H antibodies the figures were 82% and 95%, and for Vi antibodies the difference was slight—91% and 89%. Furthermore, 21% of a non-infected non-immunised group had detectable Vi antibodies, a much greater proportion than the 1% quoted by Christie.[4] One possible explanation is that other organisms possessing closely related antigens might have provoked this Vi-antibody response.

These results confirm that the Widal test is of limited value in typhoid, in which diagnosis remains essentially clinical, confirmed wherever possible by bacterial culture. Brodie looked at other serological tests and found the results equally disappointing. The sensitivity of the Coombs test for typhoid was greater, but it had the drawback of taking two days. The complement fixation test was unhelpful, as titres were low—and curiously a high percentage of anticomplementary sera was found among the immunised patients, so that the test was considered worthless in this group. Fimbrial antibodies could be detected in immunised and non-immunised patients and in healthy individuals, and the test appeared to have little diagnostic value.

The conclusions to be drawn from this mammoth study must challenge the accepted serological guidelines for the diagnosis of typhoid. Brodie suggests that H antibodies are more reliable in diagnosis than O—a reversal of the traditional view. Vi antibodies were not found consistently in chronic carriers. The study did confirm two accepted beliefs: relapse can occur despite high antibody titres, and it may not be associated with a further rise.

Possibly the Aberdeen outbreak was caused by a phage type of *S typhi* that caused a peculiar antibody response, and further information on this point would be useful. But clinicians should now be more aware of the limitations of serological tests. Laboratory confirmation of a clinical diagnosis of typhoid depends essentially on bacterial culture.

[1] Brodie, J, *Journal of Hygiene (Cambridge)*, 1977, **79**, 161.
[2] Brodie, J, *Journal of Hygiene (Cambridge)*, 1977, **79**, 181.
[3] Public Health Laboratory Service Report, *Journal of Hygiene (Cambridge)*, 1961, **59**, 231.
[4] Christie, A B, *Infectious Diseases*, 2nd edn, p 92. Edinburgh, Churchill Livingstone, 1974.

Hyperparathyroidism in renal failure

Much of the ill health of patients with chronic renal failure is due to the secondary hyperparathyroidism that accompanies it.[1–3] At first the hyperparathyroidism does not produce symptoms, but if allowed to develop the complications may include irritable red eyes with conjunctival and corneal calcification; generalised pruritus; vascular calcification with all its dangerous sequelae, including occasionally severe ischaemic myopathic changes[4 5]; cutaneous ulceration with gangrene[4–6]; ectopic soft tissue calcification; increased bone resorption leading to pseudoclubbing, skeletal bone loss, and fractures; pseudogout; tendon ruptures[7]; and autonomous tertiary hyperparathyroidism. In children, uncontrolled azotaemic hyperparathyroidism also causes the rare complication of slipped epiphyses and metaphyseal fractures[8–10] and it is an important factor in the condition radiologists call azotaemic rickets.[10–12]

What are the pathogenesis and evolution of azotaemic secondary hyperparathyroidism, and how is it best treated? All the evidence we have suggests that the central and most important stimulus to parathyroid enlargement and hypersecretion is hypocalcaemia with a reduction in the ionised serum calcium concentration.[13–15] Recent claims that a reduced production of 1,25-dihydroxycholecalciferol may have a direct stimulating effect on parathyroid hormone secretion have not been substantiated.[16] On the Bricker-Slatopolsky hypothesis[17] the hypocalcaemia was attributed solely to retention of phosphate, but equally important may well be malabsorption of calcium, itself due to a lack of $1,25(OH)_2D_3$. This hormone deficiency results partly from loss of renal tissue and partly from hyperphosphataemia, which interferes in the normal production of $1,25(OH)_2D_3$ by the remaining intact nephrons.[15 18]

Since the serum calcium concentration is of central importance any measure intended to prevent or treat secondary hyperparathyroidism in this phase needs to promote a rise in the calcium concentration. Before they need dialysis azotaemic patients will benefit from oral calcium supplements, phosphate binders, and vitamin D metabolites such as calciferol, dihydrotachysterol, and $1a\text{-}OHD_3$.[19–21] For patients on dialysis selection of the appropriate dialysate calcium concentration is also critical.[22] A low calcium concentration (less than 1·425 mmol/l (5·7 mg/100 ml) as used in the early dialysis days) could result in a negative calcium balance and so lead to osteoporosis, progressive hyperparathyroidism, and fractures,[14 23] while a high concentration of over 2 mmol/l (8 mg/100 ml) might result in progressive metastatic calcification. A dialysate calcium concentration of between 1·55 and 1·75 mmol/l (6·2