

satisfaction of the smoke. Indeed, some low-nicotine cigarettes are difficult to light and smoke, and the increased depth of inhalation or rate of puffing⁶ is likely to have little to do with nicotine itself. Recent studies of the effects of chewing gum containing nicotine on smoking suggest that if nicotine has a role in determining smoking behaviour it is small.⁷ In a crossover study the pattern of smoking was studied in volunteers who chewed chewing gum containing nicotine or a placebo chewing gum, or had no chewing gum at all. The results showed that chewing gum considerably reduced cigarette consumption, but that the chewing gum containing nicotine led to little more reduction than did the placebo chewing gum.

One study of the effects of intravenous doses of nicotine on smoking behaviour is often cited in favour of the nicotine-dependence hypothesis,⁸ but the results do not entirely support it. Four men received either 6 mg of nicotine by intermittent infusion or similar infusions of saline solution over six hours; they were not told which they were receiving. No statistically significant difference was shown between the number of cigarettes smoked during the control and the nicotine infusions. For five other volunteers the concentration of nicotine was increased to 22 mg over six hours. Now each man smoked on average 2.7 fewer cigarettes during the nicotine sessions, a small but statistically significant decrease. An infusion of 22 mg of nicotine is, however, equivalent to smoking about nine cigarettes. Thus if the pleasure of smoking or the craving for tobacco is due to the general effects of nicotine a much greater reduction in smoking frequency should have been observed. The results of the experiment therefore suggest that nicotine has a definite but small role in the smoking habit. More recently similar experiments have tended to support these conclusions.^{9 10}

One piece of evidence that weighs against the nicotine-dependence hypothesis is that in America and Britain the nicotine yields of cigarettes have declined by about half over the last 15-20 years, but cigarette consumption has not doubled. In Britain cigarette consumption in men has remained fairly steady, and in the United States it even appears to have declined. Even if nicotine does influence smoking behaviour, over several years it does not appear to be important.

Re-examining the nicotine-dependence hypothesis, a recent paper concludes that it is based on slender evidence.¹¹ The paper describes an experiment in which 12 volunteers who inhaled tobacco smoke under controlled conditions reduced their spontaneous smoking in a dose-related way—that is, the greater the intake of tobacco smoke during the control period the less they subsequently smoked when they could smoke at will. They were later given equivalent doses of intravenous nicotine; but this failed to affect their smoking, even though intravenous nicotine produced physiological effects similar to those that followed smoking. Possibly inhalation and intravenous infusion produce different effects; nevertheless, this study is one of the most direct investigations of the nicotine-dependence hypothesis.

If not nicotine, then what does mainly determine smoking behaviour? Tobacco dependence seems to be a complex phenomenon, and pharmacological agents are probably only a partial explanation. The other likely factors include the ritual of manipulating cigarettes in the hand and lips, taste and smell, relief of tension, and enhancement of sociability. But what makes people smoke is still largely a mystery.

³ Knapp, P H, Bliss, C M, and Wells, H, *American Journal of Psychiatry*, 1963, **119**, 966.

⁴ Ashton, H, *et al*, *British Medical Journal*, 1970, **3**, 679.

⁵ Russell, M A H, *et al*, *British Medical Journal*, 1975, **2**, 414.

⁶ Wald, N J, *et al*, *Lancet*, 1977, **1**, 110.

⁷ Russell, M A H, *et al*, *British Medical Journal*, 1976, **2**, 391.

⁸ Lucchesi, B R, Schuster, C R, and Emley, G S, *Clinical Pharmacology and Therapeutics*, 1967, **8**, 789.

⁹ Stolerman, I P, *et al*, *Psychopharmacologia (Berlin)*, 1973, **28**, 247.

¹⁰ Goldfarb, T, *et al*, *Clinical Pharmacology and Therapeutics*, 1976, **19**, 767.

¹¹ Kumar, R, *et al*, *Clinical Pharmacology and Therapeutics*, 1977, **21**, 520.

Teaching general practice

Two years ago when we reviewed the progress of general practice teaching in British medical schools¹ there were promising signs. Its academic credentials had become more mature, while the medical hierarchy showed a greater acceptance of its role. So on the surface today's virtual absence of medical schools without departments or subdepartments of general practice seems a gratifying continuation of previous trends—and all the more impressive because the latest developments have taken place in London and at Oxbridge.

Even so, there are more intransigent problems. These can be recognised by careful reading of parts of the daunting accumulation of evidence which the Royal Commission on the NHS is digesting. On the one hand, the University Grants Committee centres its evidence on support of the traditional base of the teaching hospital, including the view that the general practice contribution to teaching "cannot be more than a valuable but ancillary activity." It equates teaching hospitals with "the best possible medical practice," though how learning based on them aids students to "develop the flexibility to deal with changing patterns of patient care" is not made clear. The evidence goes on to equate weakening of the capacity of teaching hospitals to discharge their functions with the progressive decline in the standard of medical competence and practice—a view that many British doctors will find extraordinary.

The British Medical Association's evidence, on the other hand, welcomes university departments of general practice, but it is disturbed by the differences between medical schools in both resources and curricular time available for developing the teaching of general practice. This evidence refers directly to London, where some of the difficulties may be concentrated. No doubt London presents special problems. Often it is difficult to find practices suitable in terms not only of teaching potential but also of geographical convenience. Moreover, money for employing teaching staff is genuinely short, as are also resources for providing adequate back-up for them.

It may be that the difficulties are being unwittingly perpetuated by a vicious circle which will not be broken till it is recognised. The London teaching establishment and the University Grants Committee have many common denominators, and their attitude towards the place of general practice teaching is probably shared. Perhaps those promoting general practice teaching in London still have to overcome the sort of intellectual resistance that has subsided elsewhere. But those who are attempting to show the value of academic general practice might question whether they do not do their case a disservice by trying to do an impossible range of activities on the resources they have. It is hard enough simultaneously to teach well, to administer, to research, and to project the image of a new discipline; it is even more difficult without adequate support in terms of colleagues, finances, and time,

¹ Johnston, L M, *Lancet*, 1942, **2**, 742.

² Finnegan, J K, Larson, P S, and Haag, H B, *Science*, 1945, **102**, 94.

and almost certainly impossible while carrying a major clinical commitment. In such circumstances entrenched resistance may gain its apparent justification. Enthusiasts might consider that more respect will be gained in the long run by stating exactly what given resources can buy and ensuring that the quality of what is then offered is measurable in its depth rather than—as at present may be the case—in its unrealistic breadth.

If at length some principles do emerge on which to base the future development of academic general practice, the first must be that academic investment should not be made conditional on service-earning capacity. The second is probably that the "continuing genuine service role" for academic general practitioners provides a spurious and incomplete respectability if it is maintained at the expense of providing teaching and research of the standard the University Grants Committee would rightly like to see more generally evident before committing itself in print to a more progressive position.

¹ *British Medical Journal*, 1975, 4, 724.

Iatrogenic pancreatitis

In spite of considerable research a good deal of mystery still surrounds the pancreas. There is no simple and foolproof way of studying its appearance and behaviour in disease; our knowledge of the various forms of pancreatitis is limited, and treatment of them is far from satisfactory. Acute pancreatitis, for example, affects about 2500 people a year in Britain (twice as many in the United States), of whom some 500 die.¹ An association has been established with gall stones, alcoholism, hyperlipidaemia (especially types IV and V), and virus infections and this accounts for some three-quarters of the cases; but just how these various agents damage the pancreas is not always clear. Such factors as anatomical variations in the biliary and pancreatic ducts, a direct toxic effect of alcohol, increase of tissue-digesting enzymes, abnormal composition of the bile, and immunological reactions on the part of the pancreas have been postulated, but it may remain difficult to pinpoint a cause in an individual patient. This is particularly true in the case of drugs, where an association may not be considered because of the nature of the underlying disease.

Quite a number of drugs have been reported to cause pancreatitis, and a recent review² of 112 cases from English language papers up to 1975 is a useful guide for the clinician. Corticosteroids headed the list with 51 examples. Equal numbers of adults and children were affected, though males predominated. There did not seem to be any connection with the underlying disease, but patients with the nephrotic syndrome, asthma, and disseminated lupus erythematosus were common. The dose, duration, and type of corticosteroid were immaterial. Prognosis for an attack of pancreatitis was poor, probably because symptoms were masked by the drug. The contraceptive pill also caused pancreatitis (and gall stones) on rare occasions, and sometimes this was due to a pre-existing hyperlipidaemia. The next largest group was antibiotics, of which rifampicin was responsible in 20 patients, and almost as many cases were reported with different diuretics, where an allergic vasculitis might have been the cause. Phenformin,³ clonidine, salicylates, dextropropoxyphene, calcium (the association with hyperparathyroidism is well recognised),

warfarin, colaspase (L-asparaginase), and azathioprine were thought to be responsible in a few patients each, and recently pancreatitis has been reported after paracetamol poisoning.^{4,5} It may complicate any type of operation and may be a sequel to renal transplantation.⁶ Two diagnostic procedures—endoscopic retrograde cholangiopancreatography⁷ and trans-lumbar aortography⁸—may also be followed by pancreatitis.

The clinician faced with a patient with acute pancreatitis should always inquire about drugs, but it is unlikely that any single observer will see enough cases to be sure of an association. If a drug is suspected it should be reported to the Committee on Safety of Medicines, whose recently updated list of adverse reactions contains a section on pancreatitis⁹; and perhaps the Pancreatic Society of Great Britain and Ireland¹⁰ might bring new causes to light by undertaking a survey of iatrogenic pancreatitis encountered by their members.

¹ Myren, J, *Scandinavian Journal of Gastroenterology*, 1977, 12, 513.

² Nakashima, Y, and Howard, J M, *Surgery, Gynecology and Obstetrics*, 1977, 145, 105.

³ Graeber, G M, et al, *Archives of Surgery*, 1976, 111, 1014.

⁴ Gilmore, I T, and Tourvas, E, *British Medical Journal*, 1977, 1, 753.

⁵ Coward, R A, *British Medical Journal*, 1977, 1, 1086.

⁶ Fernandez, J A, and Rosenberg, J C, *Surgery, Gynecology and Obstetrics*, 1976, 143, 795.

⁷ Bilbao, M K, et al, *Gastroenterology*, 1976, 70, 314.

⁸ Imrie, C W, et al, *British Medical Journal*, 1977, 2, 681.

⁹ Committee on Safety of Medicines, *Register of Adverse Reactions*, 1977, 6, 451.

¹⁰ Secretary: Michael Knight, FRCS, St George's Hospital, Hyde Park Corner, London SW1X 7EZ.

Oust the louse

Head louse infestation is thought to be getting more common in children, with an incidence of 10% to 12.5% in some parts of Britain.¹⁻³ Infestation is by no means unusual in adults.⁴ Lice often go unnoticed since no symptoms arise unless the host is sensitised to their salivary antigens, when there may be pruritus of the scalp and an urticarial eruption over the neck and shoulders. Secondary pyogenic infection in the excoriated scalp causes enlargement of the cervical lymph nodes, and louse infestation must be considered a possibility in cases of impetigo. Silent infestations may be discovered only by close inspection of the scalp, when the ova (nits) may be seen fixed to the hairs, though the adult lice may not be visible.

A national campaign to eradicate the head louse started in September this year and will continue for several weeks. The area health authorities of England and Wales are mounting the project, which has been co-ordinated by the Health Education Council. The main priority is the inspection of millions of schoolchildren, though all age groups, including child play groups and the elderly, will be examined. The public will also be educated in how to recognise louse infestation and how to get treatment. So they may well ask their family doctors how it is picked up and how it can be treated. Head lice spread by close human contact, and infestation is most common in children below school age.⁵ In boys the incidence used to fall during the school years, but in 1969 Wilson¹ found a smaller difference between the rates of infestation of girls and boys at school. Possibly the fashion for long hair in both sexes played some part in this.

For some years pediculosis has been treated with DDT or gamma benzene hydrochloride, but in 1971 Maunder⁶ reported that in at least 20 areas in Britain lice had become resistant to organochlorine insecticides. Furthermore, they do