without the supervision of a doctor experienced in that field.

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Strengths and weaknesses of British medicine

SIR,—I greatly enjoyed reading the issue to mark the BMA's 150th Anniversary (5 July) on my return from holiday and would like to congratulate you on this. One recurrent theme was the excellence of clinical medicine in our hospitals and the emphasis on scientific work. Yet, as a general practitioner, I feel there has been a great fall in morale over recent years among my hospital colleagues, in contrast with work in general practice, which has become increasingly exciting and professionally satisfying.

This problem is worrying for the future of medicine and appears to be caused by the structure and content of consultant education, and the numerous and competing professional bodies to which specialists belong, so that there is no clear leadership toward possible solutions. In his article Professor Harold Ellis (p 9) suggests a future structure for education. The content of specialist training based on scientific research and clinical practice is no longer sufficient in our Health Service, and there is great need for the introduction of management studies and training to improve the delivery of specialist health care. Many consultants have no policy about outpatient clinics (which staff should see which patients, how often recurrent attenders should be reviewed by the consultant or senior registrar, when patients should be sent back to their family doctors, and so forth). They have varying and unanalysed criteria both for hospital admissions from waiting lists and length of stay in hospital for their patients and have largely and tragically vacated their personal responsibility for the administration of their workplace.

General practice has been fortunate in the recent origin and structure of its vocational training schemes, and above all by the opportunity that has been available through its organisation for able and determined men to influence its direction. Hospital doctors teach all future general practitioners. Can they learn from their former pupils who are now in family medicine?

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Malalignment of the shoulder after stroke

SIR,—The overall incidence of malalignment of the shoulder after stroke was reported by Dr R G Smith and others (24 April, p 1224)

as 39% on admission rising to 53% after 12 months.

Routine chest x-ray films of 300 consecutive admissions to a department of geriatric medicine were screened for subluxation.¹ Twenty-one patients (7%) showed evidence of subluxation, four bilaterally. Subluxation was measured using a method described by Poppen and Walker² and was shown to fall into two groups: 13 shoulders showed downward subluxation and 12 upward subluxation. One was dislocated. Five patients had suffered a stroke and four of these had bilateral subluxation. In all these patients subluxation was downward, with a mean subluxation of 11 mm. There was no significant correlation between loss of passive movements and degree of subluxation or between subluxation and activities of daily living. Eight patients in the group complained of occasional pain.

Upward subluxation is probably related to previous injury, an intrinsic shoulder abnormality, or lax musculature, or it may simply reflect the aging process.¹ Subluxation associated with stroke is almost certainly related to lax musculature, but those who develop subluxation may have had an underlying shoulder abnormality. Malalignment in itself is probably of little significance unless it is associated with pain. Physiotherapy remains the most widely accepted treatment for pain though it probably has a negligible effect on the course of subluxation.³⁻⁵

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¹ Carpenter GI, Millard PH. *J Am Geriatr Soc* 1982; 30:441-6.

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Introduction of 100-unit insulin

SIR,—It is common knowledge that Britain intends to introduce 100 U/ml insulin in 1983, but it has been brought to my attention that 40 U/ml and 80 U/ml insulins will continue to be available. In my opinion this would be a disaster, and, like Dr Peter J Watkins (26 June, p 1929), I think it will lead to great confusion. When Australia was exploring the possibility of changing to 100 U/ml insulin a sub-committee of the Australian Diabetes Society investigated the effects of the change in the USA and Canada and made the following points in their report: (a) a change to a single strength of insulin was desirable; (b) 100 U/ml was the logical strength; (c) a change should be made only if 40 and 80 U/ml insulins were withdrawn and suitable syringes for 100 U/ml insulin were available. The Australian Diabetes Society recommended the change, with these provisos, and it was accepted by the Commonwealth Department of Health. Insulin and syringe manufacturers were given more than 12 months' warning and 100-unit insulin was introduced on 1 August 1980. By 1 December 1980 40 and 80 U/ml insulins were no longer available in Australia.

With intense publicity to the profession, pharmacists, nurses, and patients for six months before 1 August 1980, the change went very smoothly and there were very few examples of confusion. I believe the present situation, with only 100 U/ml insulin available, is stable, and we are not encountering the mis-

understandings that occurred when 40 and 80 U/ml insulin were used.

I urge those concerned with the change in Britain to take note of this lesson and to look at the problems that exist in the USA, where 40 and 80 U/ml are still available (although in decreasing amounts). The reason for the change is to have a single strength of insulin. Do not lose sight of this reason by perpetuating the situation of having multiple insulin strengths.

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Reversible hypothyroidism in adrenal insufficiency

SIR,—The paper of Dr Anthony Barnett and others (17 July, p 172) reminds us that the results of conventional thyroid function tests require careful interpretation in patients with untreated adrenocortical insufficiency (Addison's disease or isolated ACTH deficiency). Their experience supports the data of Topliss *et al*,¹ who reported six patients with Addison's disease whose abnormal thyroid function tests were similarly reversible, as well as our own unpublished observations. We are aware of six patients found to have Addison's disease at this hospital in the last three years. Three had abnormalities of thyroid function on presentation (raised basal thyroid stimulating hormone (TSH), low free thyroxine index (FTI); exaggerated TSH response to thyrotrophin-releasing hormone, low FTI; raised basal TSH) and in all three these abnormalities were corrected within four months of corticosteroid replacement therapy. Like Dr Barnett and his colleagues we feel it is most likely that corticosteroid deficiency impairs thyroid function directly and leads thereby to a rise in TSH with or without a fall in serum thyroxine. Carpenter *et al*² produced other evidence of such a thyroid defect by showing an impaired thyroid response to exogenous TSH in untreated Addison's disease.

When Schmidt described the association between adrenocortical failure and thyroid disease in 1926³ he reported lymphocytic infiltration of the thyroid in two patients who had died of Addison's disease. A similar finding had already been reported by Dubois,⁴ and up to 1964 there had been 72 reports of such infiltration in a total of 127 patients studied. It is interesting to note that although lymphocytic infiltration was commoner (53 out of 65 patients; 82%) in those with atrophic (presumed autoimmune) Addison's disease, it was still frequent (19 of 62; 31%) in those whose adrenocortical failure was the result of tuberculosis. This supports the contention that thyroid function may be impaired as a result of corticosteroid deficiency per se, even though it occurs more commonly in those with an autoimmune diathesis.

But the question of treatment remains unanswered: should thyroxine be withheld in the expectation that the abnormality on thyroid function tests will correct spontaneously? The rise in TSH, with or without a fall in thyroxine, is not a biochemical artefact and represents true, albeit temporary, hypothyroidism. Two groups^{5,6} have reported a total of six Addisonian patients with reversible hypothyroidism and in these cases the diagnosis of dysfunction was based on clinical grounds as well as on serum cholesterol, basal metabolic rate, protein-bound iodine, and radioiodine uptake, and not

just on the results of radioimmunoassay. Since a proportion of Addisonian patients will go on to develop permanent hypothyroidism and since the return to normality in the remainder may be delayed by many months, it would seem wisest to give thyroxine initially to all who are biochemically hypothyroid, and to attempt cautious withdrawal in some of them at a later stage.

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Guillain-Barré syndrome associated with campylobacter infection

SIR,—Drs K M Rhodes and A E Tattersfield (17 July, p 173) reported the first published case of Guillain-Barré syndrome in association with campylobacter infection. We have seen a patient with Guillain-Barré syndrome and some similar features to those of their case.

A 42-year-old lorry driver developed the initial symptoms of Guillain-Barré syndrome nine days after the onset of four days' diarrhoea, which he experienced abroad. The weakness of the extremities peaked on the fourth day after the appearance of the first symptoms of the syndrome. At that time the patient was unable to walk but was not brought to hospital until the eighth day. He did not need artificial respiration and his condition started improving during the next few days. The cerebrospinal fluid showed a protein content of 830 mg/l. Routine blood investigations were normal, and the stool cultures for *Salmonella*, *Shigella*, and *Yersinia* were negative. No antibodies compatible with acute viral, salmonella, yersinia, brucella, or chlamydia infection were found. Stool was cultured only once for *Campylobacter* (23 days after the onset of diarrhoea), and *Campylobacter fetus* subspecies jejuni was isolated. Electroneuromyography 24 days after the onset of Guillain-Barré syndrome showed pronounced exaggerated insertion activity throughout the muscles, reflecting axonal involvement. The motor and sensory nerve conduction velocities were normal. Two months later considerable denervation activity was present as a sign of extensive axon damage. After six months' follow-up there was atrophy of the distal muscles and weakness in all extremities, and, unexpectedly, the patient also had a high-frequency tremor and slight rigidity of the extremities.

The common features of our patient and that of Drs Rhodes and Tattersfield were the clear-cut period of acute diarrhoea, the short incubation period of Guillain-Barré syndrome, and the extensive axonal damage with considerable residual neurological signs even six months after the onset of the disease. *Campylobacter* appears to be the most common bacterial cause of diarrhoea.¹ We agree with Drs Rhodes and Tattersfield that at least some cases of Guillain-Barré syndrome developing after "non-specific" enteric infection are connected with the occurrence of campylobacter infection. In the light of our patient's tremor we also noted that *Campylobacter* has been suggested as a causal factor in some diarrhoea-associated convulsions,^{2,3} and involvement of the nervous system has also been reported.⁴ The pathogenesis of

Guillain-Barré syndrome might be different in various cases,⁵ and tissue damage in the nervous structures might itself be related to the causal agent. In these two cases our attention was particularly drawn to the extensive axonal damage.

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Impact of maternal serum alpha-fetoprotein screening on antenatal diagnosis

SIR,—We are forced to take issue with the conclusions drawn by Dr David J H Brock (31 July, p 365). Dr Brock fails to indicate: (a) the number of terminations in which no significant abnormality was found; (b) the number of neural tube defects not identified by a raised maternal serum alpha-fetoprotein estimation; and (c) the morbidity induced by amniocentesis performed for alpha-fetoprotein estimation. Our experience¹ is that this test is insufficiently sensitive and insufficiently specific. It is usually positive in the presence of a gross fetal neural tube defect, when prospects for survival of the fetus are low. It is much less reliable in the presence of lesser degrees of neural tube defect, when survival is likely and may lead to long-term institutional care.

Dr Brock, reiterating the well-known inconclusive association between moderately raised maternal alpha-fetoprotein and fetal neural tube defect, does not provide sufficient evidence to justify national adoption of the screening test.

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- ¹ Standing SJ, Brindle MJ, Macdonald AP, Lacey RW. *Br Med J* 1981;283:705-7.

Left-handedness

SIR,—I would like to comment on the reply given to the reader who wished to know if there was any association between left-handedness and a variety of dextrocardia and transposition of thoracic and abdominal viscera (10 July, p 118). Your expert's answer, that "there is no association between the two," may not be correct. In a recent study on "Left-handedness: association with immune disease, migraine, and developmental learning disorder,"¹ we found the frequency of autoimmune diseases, migraine, and developmental learning disorders to be increased in left-handed subjects. In obtaining this information we did not ask

about dextrocardia or transposition of viscera, but unsolicited information from several families with left-handedness and autoimmune disease provided multiple examples of transposition of great vessels and of thoracic and abdominal viscera. The theoretical relation between developmental brain abnormalities, laterality, autoimmune disorders, and learning disorders is discussed in our paper.

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- ¹ Geschwind N, Behan P. *Proc Natl Acad Sci USA* 1982 (in press).

Inaccuracy of London School of Hygiene sphygmomanometer

SIR,—Dr Desmond J Fitzgerald and others (2 January, p 18) showed recently that the London School of Hygiene (LSH) sphygmomanometer, when compared with a Hawksley random-zero sphygmomanometer, underestimated the systolic blood pressure by 7.1 mm Hg and the diastolic pressure by 3.6 mm Hg (average of 20 patients in whom simultaneous duplicate readings were obtained by connecting two stethoscopes via a connection to the cuff). Dr J G Evans (13 March, p 823) pointed out that the random-zero sphygmomanometer in turn has been shown to underestimate systolic pressure by 1.2 mm Hg and diastolic pressure by 2.2 mm Hg compared with an ordinary sphygmomanometer.¹ Thus, the LSH sphygmomanometer can be expected to show pressures around 8 mm Hg lower for systolic and 5.6 mm Hg lower for diastolic than ordinary clinic readings made with a mercury sphygmomanometer.

We have compared an ordinary mercury sphygmomanometer with the Auto-Manometer,² an electronic device manufactured by Southern Computers Ltd (PO Box 6091, Dunedin North, New Zealand) which operates in a manner similar to the LSH sphygmomanometer. It was originally developed in the hypertension clinic of Professor Simpson at the Wellcome Research Foundation in Dunedin, New Zealand. We used the same methods as Dr Fitzgerald and others with the modification that we let the two observers make two recordings in each patient, between which they changed manometers. We picked 20 patients at random from those making routine visits to the hypertension clinic.

The results are shown in the figure. The linear regression lines based on the average values of the two observations for each apparatus were Auto-Manometer = 1.10 mercury manometer (systolic)—19.3 and Auto-Manometer = 1.18 mercury manometer (diastolic)—21.9. The average values showed a mean underestimation by the Auto-Manometer of 3.8 mm Hg for systolic pressure and 4.4 mm Hg for diastolic pressure (both $p < 0.001$) at a pressure level of around 150/90 mm Hg. The regression lines are not quite parallel to the lines of identity, so the error is probably smaller in higher blood pressures. When one trained observer (GN) checked Auto-Manometer readings against self-read mercury readings in 16 patients, Auto-Manometer readings were also consistently lower than mercury readings by 1.0 mm Hg systolic and 2.9 mm Hg diastolic (both $p < 0.01$).

At 10 calibrations with the cuff around a cylindrical object the mercury readings were higher by 0.5 mm Hg. When the two observers were checked against one another, although average differences were small, the difference between the two occasions could be very large in individual cases. For the mercury manometer, systolic pressure