Having said all this I think that family practitioner committees must take sympathetic note of special conditions and circumstances such as the difficulties and expense of getting out of hours cover from locums during holiday periods; the problems of elderly singlehanded GPs; and in particular those of inner city doctors in unsalubrious areas, who, for reasons such as concern about their children's education, tend to live further from their practices.

Any rigidity is apt to cause hardship and resentment and a hardening of views against any reform of the present use of these services in some areas; this would not be in the interest of either the public image of GPs or patient care.

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SIR,—The Minister of Health has at last decided that new arrangements are needed to limit the increasing frequency of use of deputising services by general practitioners and to ensure that those services are staffed by adequately trained doctors. It is time somebody did so.

His proposals seem sensible and moderate to me but quite the contrary to most of the doctors who use the deputising services. Out of hours responsibility for patients has always been part of general practice and is acknowledged by the payment of supplementary fees and allowances for the work. These are paid automatically to all doctors unless they notify the family practitioner committee that they want to opt out of the work. When about half of all doctors regularly subcontract their out of hours work to deputising services whose standards of care are thought to be lower than those of the family doctor service the government is bound to wonder if it is getting value for money. The independent contractor status of general practitioners has always been disliked by administrators because it exempts them from much of the bureaucratic control to which all other workers in the NHS are subject. The deputising services are even less subject to administrative control, and questions are likely to be asked about the financial relations between contractor and subcontractor. Is the contractor making a profit out of the transaction? Would it not be cheaper for the NHS to run the deputising services itself? These pose a threat to the independent contractor status.

The logical solution is to accept the fact that there are now two kinds of general practitioner: those who are willing to accept out of hours responsibility for their patients and those who are not. Those who are not should opt out and, of course, forego supplementary payments. The money saved could be used to employ the deputising services, which could be administered by the minister's proposed new deputising subcommittees of family practitioner committees. It would create two sorts of contract, one for those who undertook out of hours responsibilities and the other for those who did not, but the independent contractor status of general practice would no more be questioned than it is at present by opting out of midwifery or family planning services.

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SIR,—I spent eight years working in Canada, where few urban family doctors offer, or are

expected to offer, out of hours emergency services. If people fall ill during the night or at weekends they telephone the hospital emergency department. In rural Canada the family doctor tends to centre his activities round the GP hospital, and patients who fall sick out of office hours telephone the local hospital. If they have viral infections or other minor ailments, as up to half the callers may do, the nurse can dispense advice appropriately. If it is more complicated she calls the patient into the hospital, assesses the problem, and telephones the GP on call, who will then go into the hospital.

Why couldn't we operate a similar system here? In our community we have 12 doctors operating two separate emergency services for about 25 000 patients. All the doctors use our local GP hospital, but, with only 14 beds, nurses operating the night shift are underemployed. The public service planners are questioning the cost effectiveness of GP hospitals, while at the same time, paradoxically, a private hospice is being planned for our community. My proposal is simply that the GP hospital could become the focus for a night and weekend emergency GP service. One reason that these hospitals have become vulnerable to cuts is that while they used to meet the medical needs of all age groups they now primarily serve the old. The old form a growing but relatively non-vocal and undervalued part of the community. But what would happen if 25 000 patients, including the community leaders, all became aware of their local hospital?

Currently two doctors, their wives, children, or receptionists are available during evenings and weekends to receive emergency calls for two separate groups of doctors, and the doctors' families, and the single doctor bound to the telephone, may all find this a time consuming and emotionally draining ordeal. Out of hours calls could be directed to the GP hospital. Here, as in the Canadian model, a nurse could assess the problem. Many callers would be seeking advice about minor ailments and the nurses could provide this. Patients with more serious problems could come to the hospital for medical assessment. If the patient was bed-bound or lacking transport, the GP could be contacted direct for a home visit.

How could this be funded? A year ago when my partner and I tried to get our calls merely answered and relayed on to us by a commercial agency we were charged £60 a month. If each doctor in our local emergency scheme subscribed this sum it would generate £4320 a year. This could be used for additional nursing hours at the hospital, or current staff and facilities could be used more effectively with a net gain for the GP hospital.

LEONE RIDSDALE

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Huntington's chorea

SIR,—In his leading article (26 November, p 1567) Professor P S Harper suggests that there are about 3000 people suffering from Huntington's chorea in Britain at any one time, implying a prevalence of about 5.5 per 100 000. Our work in Scotland (unpublished) suggests the overall figure for Scotland will be about 9 or 10 per 100 000, more probably 10 as, for various reasons, retrospective surveys in Huntington's chorea give a higher figure than current ones. This would give a total of

around 500 patients with Huntington's chorea in Scotland and suggests that the estimate for England and Wales should perhaps be higher, especially since some surveys, such as that by Walker *et al*, have yielded higher prevalence rates.¹

Professor Harper rightly emphasises the need for adequate counselling of those at risk before they undergo a predictive test and of subsequent support for those who are probably carrying the gene. There is also a major need for continuing, long term support and help for most of those affected and their families.

It must be remembered that Huntington's chorea will be with us for many years, and at present these unfortunate patients and their families tend to fall between the medical and social services, inadequately supported by either. Furthermore, there is a tendency today to see problems as acute episodes, with the patient or client being discharged after the immediate difficulty has been dealt with, whereas the need here, as with other chronic disabilities, is really for a continuing contact that can provide more definite support when needed.

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¹ Walker DA, Harper PS, Wells CEC, *et al*. Huntington's Chorea in South Wales: a genetic and epidemiological study. *Clin Genet* 1981;19:213-21.

Whooping cough and pertussis vaccination

SIR,—Dr A H Griffith's accusation (17 December, p 1881)—for the second time—that I misquoted a statement from a Public Health Laboratory Service report on vaccines in my reply to him (12 November, p 1470) is transparently untrue.

I acknowledged the minor difference in results obtained with his company's vaccine. If he believes that Wellcome vaccine is so superior to that made by similar methods by other companies in Britain, North America, Holland, and West Germany, why does he not produce results to prove his point instead of prevaricating with mine? What concerns us now is the efficacy and safety of current vaccines, not those made before 1968 even if, as we knew then better than now, some were more effective than others or, should I say, less ineffective: none was free from toxicity.

This leads to a further prevarication in his letter. He knows that I support him in his wish to obtain relevant data on adverse reactions on this and other products from the Department of Health and Social Security. His quarrel in this respect is with that Department, with the Committee on Safety of Medicines, with the National Biological Standards Laboratories, and with other regulating agencies more familiar to him than to me. He might also, very reasonably, ask the Vaccine Damage Payments Unit for anonymous disclosure of awards made to children who have received his company's vaccines, for there is an obvious responsibility of all concerned to share relevant information about incidents investigated by them.

Dr Noel W Preston (17 December, p 1881) is an expert on the serotype antigens of *Bordetella pertussis* and I do not dispute his point about variations in their distribution, temporally and geographically, though I doubt if our information in this respect is more than patchy. The point is that—unlike certain serotype variants in some other organisms—these antigens of *B pertussis* appear to be of little or no importance immunologically or epidemiologically. In saying this I attached credence less to my own opinion than to those of

pioneers like Pearl Kendrick, Margaret Pittman, L B Holt, and members of the WHO group, and I gave relevant references. If Dr Preston objects, he should refute their work not mine. He might also take issue with Dr Griffith, who says (and I do not doubt it) that his company's vaccine contained all the main antigens from 1964 or earlier. Addition of antigen 3 to all British vaccines did not stop the major outbreaks of 1967-8, 1970-1, or 1974-5 during a period when vaccine acceptance was much higher than it is now; nor did it bring any change in the fact that 25-35% of proved cases, now as well as then, occur in fully vaccinated children not only in Britain but also in the United States,¹ Canada,² Australia,³ Sweden,⁴ and elsewhere. In parts of Scotland in 1977-9 the incidence of whooping cough in fully vaccinated children exceeded this range,⁵ and it could be argued that the loss of immunity two to three years after vaccination is the main determinant of the otherwise unexplained 44 month periodicity of outbreaks of whooping cough in Britain (personal report to Department of Health and Social Security).

I imagine that most of your readers are bored, confused, or exasperated by the continuing controversy about this one, relatively unimportant infection and its relatively ineffective vaccine, which is disproportionately important because so many children receive it. Might I therefore state yet again the central plea in my own argument for the need to repair "the national deficit in epidemiological data and intelligence,"⁶ because without some improvement, and not only about pertussis vaccine, we are abusing the trust of the public in medical judgment and in medications.

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¹ Pertussis surveillance, Georgia. *MMWR* 1977;26:307-8.

² Mathias RG. Whooping cough in spite of immunization. *Can J Public Health* 1978;69:130-2.

³ Bennet NM. Whooping cough in Melbourne. *Med J Aust* 1973;ii:481-7.

⁴ Eksmyr R. Kikhosta-en oppnevdsstudie. *Lakartidningen* 1979;76:4818-21.

⁵ Stewart GT. Vaccination against whooping cough: efficacy versus risk. *Lancet* 1977;i:234-7.

⁶ Ditchburn RK. Whooping cough after stopping pertussis immunisation. *Br Med J* 1979;ii:1601-3.

*.*This correspondence is now closed.—ED, *BMJ*.

Effects of pirbuterol and sodium nitroprusside on pulmonary haemodynamics in hypoxic cor pulmonale

SIR,—I would like to take issue with the methodology used by Dr W MacNee and others (22 October, p 1169) for measurement of pulmonary vascular resistance and the effects on this variable of the two drugs pirbuterol and sodium nitroprusside.

The authors state: "Left atrial pressure is not always easy to assess from pulmonary artery wedge pressure in these patients." The formula for measuring pulmonary vascular resistance is therefore invalid, as it includes no estimate of the pressure difference across the pulmonary circuit. Whether one believes in the concept of "resistance" in the pulmonary circuit or "impedance," I am sure that most workers would argue that the formula used in this study was wrong.^{1 2}

Furthermore, it is not valid to assume that long term administration of pirbuterol had beneficial effects, when no estimate is made of cardiac output. Thus a reduction purely in systolic pressure does not seem clinically

important in the pulmonary circuit when there is no estimate of flow through it.

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¹ Foex P. *Pulmonary dynamics*. In: Prys-Roberts C, ed. *The circulation in anaesthesia*. Oxford: Blackwell Scientific Publications, 1980:258-9.

² Kaplan JA. *Immunodynamic monitoring*. In: Kaplan JA, ed. *Cardiac anaesthesia*. New York: Grune and Stratton, 1979:95.

*.*The authors reply below.—ED, *BMJ*.

SIR,—Dr D W Green is, of course, technically correct. In any segment of the circulation the resistance is defined as the drop in pressure across the segment divided by the rate of volume flow through it. Thus the pulmonary vascular resistance is expressed as the difference between the mean pulmonary arterial pressure and the mean left atrial pressure divided by the cardiac output.

In stating this, however, Dr Green has ignored the controversy that has existed for the past 20 years or more as to whether measurement of pulmonary capillary wedge pressure accurately reflects mean left atrial pressure in patients with severe chronic bronchitis and emphysema, such as those studied in our paper. Fishman states: "Pulmonary capillary wedge pressure and pulmonary diastolic pressure are unreliable indexes of left atrial pressure" in such patients as "either venoconstriction or anatomic change in the intervening vessels may invalidate these measurements."¹ Recent authors support the view that the measurement of pulmonary capillary wedge pressure should be interpreted with caution in patients with disordered pulmonary mechanics,² and in such patients "a simple mean of the wedge pressure can be raised secondary to positive intrathoracic pressure during expiration."³ Although Lockhart has not ascribed the rises in pulmonary capillary wedge pressure in patients with obstructive lung disease to a generalised rise of intrathoracic pressure, close scrutiny of this paper (fig 4) shows a variability of 5 mm Hg between pulmonary capillary wedge pressure and left ventricular end diastolic pressure measured at rest.⁴

It is for these reasons that we chose to measure *total* pulmonary vascular resistance, so avoiding the effect of a possible error in the measurement of resistance. As pulmonary artery pressure in our patients was high and we have assumed that left atrial pressure was low (as all of our patients had well preserved left ventricular function) this is not dissimilar to the approach in calculating systemic vascular resistance, where the right atrial pressure is not usually considered. It was for similar reasons that *total* pulmonary vascular resistance was measured in the MRC long term domiciliary oxygen trial⁵ and that the largest single series of measurements of pulmonary haemodynamics in patients with obstructive lung disease does not mention measurements of pulmonary capillary wedge pressure.⁶ We cannot therefore agree with Dr Green that "most workers would argue that the formula we used in this study was wrong." We have defined *total* pulmonary vascular resistance (kPa s/cm⁵) as the mean pulmonary artery pressure divided by the cardiac output times 8—a definition used by many other workers.⁷

We would also point out that the beneficial effects of long term administration of pirbuterol were based on a reduction of systolic pulmon-

ary artery pressure and an increase in right ventricular ejection fraction. Cardiac output was not measured in order to reduce the number of invasive measurements made during the chronic study. As the increase in right ventricular ejection fraction paralleled the increase in cardiac output during the acute study and as a consistent increase in right ventricular ejection fraction occurred in the chronic study, we have assumed that cardiac output would also increase.

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¹ Fishman AP. *Pulmonary diseases and disorders*. New York: McGraw Hill, 1980:856.

² O'Quin R, Marini JJ. Pulmonary arterial occlusion pressure: clinical physiology measurement, and interpretation. *Am Rev Respir Dis* 1983;128:319-26.

³ Rice DL, Awe RJ, Gaasch WH, Alexander JK, Jenkins DE. Wedge pressure measurement in obstructive pulmonary disease. *Chest* 1974;66:628-32.

⁴ Lockhart A, Tzareva M, Nader P, Leblanc P, Schrijen F, Sadoul P. Elevated pulmonary artery wedge pressure at rest and during exercise in chronic bronchitis: fact or fancy. *Clin Sci* 1969;37:503-17.

⁵ MRC Working Party. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981;i:681-6.

⁶ Bishop JM, Cross KW. Use of other physiological variables to predict pulmonary artery pressure in patients with chronic respiratory disease. *Eur Heart J* 1981;2:509-17.

⁷ Morrison D, Goldman S, Wright AL, et al. The effect of pulmonary hypertension on systolic function of the right ventricle. *Chest* 1983;84:250-7.

Recognising placental steroid sulphatase deficiency

SIR,—The determination of the steroid sulphatase activity in leucocyte homogenates (23 July, p 293)¹ certainly meets the demand for an "easy diagnostic method for X-linked ichthyosis suitable for routine clinical biochemistry laboratories" put forward in the leading article by Dr R A Harkness and others (2 July, p 2). Unfortunately, we observed a considerable variation of the steroid sulphatase activity among obligate carriers of the steroid sulphatase deficiency trait. This limits the ability of our simple method¹ to recognise heterozygotes, which can be needed urgently in cases of low urinary steroid excretion during pregnancy as this may be caused by steroid sulphatase deficiency.

The results of the steroid sulphatase activity determination proved the heterozygosity for this trait in only 14 out of 18 carriers (figure). The much greater variation in the carrier group than in the healthy normal group is not likely to be due to technical differences, as all assays were carried out in the same period by one person.

Scientifically, these results are most interesting as they indicate that no complete escape from X-chromosomal inactivation of the steroid sulphatase locus seems to occur in leucocytes. This, however, differs from the results with cultured fibroblasts initially reported by Shapiro *et al* for four obligate carriers² and reported by Müller *et al* for five obligate carriers,³ which suggested complete escape of inactivation. The reported steroid sulphatase activity in leucocytes⁵ and in lymphocytes⁶ of normal persons and further work of Shapiro's group on cultured fibroblasts from carriers of the steroid sulphatase deficiency trait⁷ indicate, though, a differential expression of the steroid sulphatase locus.