treated with neutrons and photons.² Results were significantly better, however, for neutron than photon treatment in node positive patients, but worse in node negative patients with the suggestion that these were caused by inadequate planning because of the fixed neutron beams. No opposition was expressed to continued trials now under way. The discontinuation of funding suggested by Dr Tobias refers to that of new cyclotrons rather than of existing units.

The arguments against neutrons have been well rehearsed. We still do not have clear cut answers. The proposed unit at St Thomas's would be for not only neutron therapy but also particle treatment of eye tumours and nuclide production for positron emission tomography. Cancer therapy depends on adequate planning of treatment obtainable only with the newer high energy neutrons. The earlier work at Hammersmith using fixed horizontal beams was a remarkable achievement, whatever the final value of neutron therapy.

The real interest in this debate lies not only in the true role of neutron therapy but in the political machinations of the funding process. These have no long term significance. We await with interest the results of clinical trials and the evaluation of data obtained by positron emission tomography.

KEITH HALNAN
KAROL SIKORA

Department of Clinical Oncology,
Royal Postgraduate Medical School,
Hammersmith Hospital,
London W12 0HS

- 1 Tobias JS. The cyclotron saga continues. *Br Med J* 1989;299:1294-5. (25 November.)
- 2 Griffin TW, Pajak TF. Mixed neutron/photon irradiation of unresectable squamous cell carcinoma of the head and neck: the final report of a randomised co-operative trial. *Int J Radiat Oncol Biol Phys* 1989;17:959-65.

Insertion of permanent pacemakers as a day case procedure

SIR,—Dr Guy A Haywood and colleagues report that the insertion of permanent pacemakers as a day case procedure is as acceptable to patients as conventional admission and that the incidence of complications is comparable in these groups. I wish to draw their attention to a few facts.

The incidence of lead displacement causing capture failure was reported to be 1/21 in the day case group and 1/19 among patients managed conventionally. Whether this was detected during the hospital stay or after discharge and the mode of presentation have not been mentioned by the authors. As only 37 patients received new permanent pacemakers (three having received only generators), the incidence of capture failure following lead dislodgement is 2/37—that is, more than 5%. Although there was no significant difference between the two groups, this is a significantly high rate of displacement considering the present lead designs. The rate has been reported to be as low as 0.4%,¹ and this problem has not occurred in my latest 73 patients. This is an extremely important complication: for patients who depend on pacemakers capture failure could be fatal, especially if the patient has been discharged from the hospital. May I therefore suggest that the authors review and if necessary revise their method of lead implantation before taking to implanting permanent pacemakers on a day case basis?

Also, the authors have mentioned that "standard implantation procedures" were used but did not specify the exact number of procedures performed by subclavian puncture or cephalic vein cut down. It is important to note that the chances of haematoma formation are considerable with subclavian puncture.¹ In cephalic venous cut down complete haemostasis is possible because the

same is done under vision. Thus in patients undergoing permanent pacemaker implantation as a day case procedure cephalic venous cut down would be preferable to prevent the complication of haematoma formation and subsequent infection.

BHARAT DALVI

Department of Cardiology,
King Edward VII Memorial Hospital,
Bombay 400 012,
India

- 1 Haywood GA, Camm AJ, Ward DE. Insertion of permanent pacemakers as a day case procedure. *Br Med J* 1989;299:1139. (4 November.)
- 2 Mond H. The pacemaker lead. In: Mond H, ed. *The cardiac pacemaker: function and malfunction*. New York: Grune and Stratton, 1983:76.
- 3 Janchuck SJ, Gill BS, Petty AH. Permanent cardiac pacing through the subclavian vein. *Br J Surg* 1974;61:373-6.

AUTHORS' REPLY.—Two patients were affected by lead dislodgement. One was assumed to have a microdislodgement as he had a low threshold before discharge but at one month follow up was failing to pace. A chest radiograph showed no obvious displacement, and increasing the pulse amplitude resulted in recapture. The other patient was one of the three who were to receive new generators, but an insulation break was noted in the old lead and a revision of system was carried out. Pacing was satisfactory before discharge, but at follow up at one month the chest radiograph showed lead displacement. Assuming that the threshold rise in the first patient was due to a microdislodgement, the rate was 2/38 (5.3%) for the series, which is within the range for electrode displacement quoted in other series (0.12-5% for ventricular and 0.9-6% for atrial leads) but is higher than the mean reported in these series (1.6% for ventricular and 3.2% for atrial leads).¹ The numbers are, however, small, and Dr Dalvi's suggestion that our displacement rate is significantly higher is statistically incorrect.

The important question is whether travelling after implantation increases the risk of electrode dislodgement compared with the risk in patients who remain in hospital for one or more days after implantation. As we pointed out in the article, calculations of sample size show that 2500 patients would have to be studied for a 90% probability of showing a doubling of the frequency of electrode dislodgement using a one tailed test at the 5% level of significance. We thought that it would be impracticable to attempt a study of this size, and we draw Dr Dalvi's attention to the studies referenced in the introduction to our article, which attempted to assess safety.

We agree that cephalic cut down is the method of choice for introduction of the pacing lead, and it was used in 20 of the 38 patients in our series. In the remainder the operator judged the cephalic vein to be unsuitable. We agree that this method is preferable for obtaining good haemostasis. It is our impression, however, that most haematomas form because of leakage from small vessels disrupted while the generator pocket is formed rather than from bleeding from the site of the subclavian vein puncture.

GUY HAYWOOD
A JOHN CAMM
DAVID WARD

Department of Cardiological Sciences,
St George's Hospital Medical School,
London SW17 0RE

- 1 Ohm J, Breivik K. Pacing leads. In: Perez-Gomez F, ed. *Cardiac pacing, electrophysiology, tachyarrhythmias*. Madrid: Grouz, 1985:971.

Child health surveillance

SIR,—Dr Leon Polnay's article highlights many important issues in child health surveillance.¹ I have recently researched the conditions and cir-

cumstances surrounding failure to thrive in children aged 12-24 months. The findings are particularly relevant to two of the issues raised—namely, the value of parental observations and the importance of growth monitoring.

The study was based in the two electoral wards of Newcastle most affected by socioeconomic deprivation. Fifty two children (mean age 20.8 months) with mild failure to thrive were identified. Each was matched by age and gender with a control child, growing normally, from the same communities. Failure to thrive is often associated by professionals with neglectful or abusing parents, but I found that the parents of children who were failing to thrive were clearly aware of their child's predicament and were concerned about it. Analysis of responses to a questionnaire on attitudes showed that parental anxieties over their child's health, growth, and eating patterns were significantly different from those of parents in the control group. Most of these families were not, however, receiving any input from health care professionals.

The mean number of weight measurements documented in the records of the children who were failing to thrive was 17, higher than for the controls (mean 12; $p=0.001$). More frequent attendance may represent an indirect expression of these parental anxieties. Evidently, listening to parents talking about their concerns will aid the identification of vulnerable children.

The article mentions the somewhat equivocal views in *Health for All Children* on the value of growth monitoring. My findings suggest, however, that growth charts represent a valuable tool for child health surveillance. Even in these deprived communities, where uptake of services is variable and often believed to be poor, 92% of all children had adequate growth charts (defined arbitrarily as those with at least six recorded weight measurements—that is, about every three months) held either by the health visitor or by the local authority clinic.

The prevalence of mild failure to thrive was found to be 20.6%. The mean body mass index of these children was 16.1 kg/m², significantly less than in the control group (17.4 kg/m²; $p<0.01$), and a wide range of adversity was identified among children who were failing to thrive. This included greater socioeconomic deprivation, nutritional insufficiency, more prevalent minor illnesses, and interactional difficulties between parent and child. They were thus a highly vulnerable group of children.

Monitoring children's growth is therefore an effective way of identifying children who need further assessment and intervention. It can be applied to most children and deserves a central part in child health surveillance.

ADRIAN EDWARDS

Newcastle NE2 4LR

- 1 Polnay L. Child health surveillance: new report highlights value of parental observations. *Br Med J* 1989;299:1351-2. (2 December.)

SIR,—Dr Leon Polnay¹ accurately summarises the conclusions in *Health for All Children*² in relation to growth monitoring. The recommendations to weigh at each clinic visit and measure height at 3 years and between 4 and 5 years seem unreasonable, however, from the evidence discussed.

Weight gain is a poor guide to health or healthy growth. Even in infancy, when it is of some clinical relevance because growth is very rapid, fluctuations largely reflect bowel and bladder contents as the report points out. In older children differences between normal and abnormal weight gain are smaller than the reproducibility of weight measurements some months apart, even on sophisticated apparatus. Weights are often inaccurate, but even accurately recorded weight gain reflects change in many tissues and can be seriously misleading as a growth indicator. Frequent weighing is thus

unlikely to be of value and often causes unnecessary anxiety. Nutrition is better assessed using skinfold calipers (which are also cheaper and more portable than weighing scales) to measure directly the thickness of subcutaneous fat.¹

Accurate height measurement (supine length in infants under 2 years) is a sensitive guide to child health.¹ Growth velocity (calculated from repeated measurements of height at intervals) represents the current dynamics of growth much better than a single measurement, which reflects previous growth. Regular, accurate measurement of children can identify those who would benefit from medical, social, or educational intervention.¹

Many height measurements in hospital and the community are inaccurate and misleading because of careless techniques and inadequate apparatus. Suitably accurate, cheap, and portable apparatus is now widely available for use in primary care, and measuring techniques eliminating postural drops and positional errors are readily learnt by motivated staff. Supine length in children under 2 years can generally be measured accurately with the help of an assistant.

Collected accurate growth (height) data in children have important benefits beyond those to the individual—as an index of the health of a population or a subgroup (for example, ethnic group or social class). British data are not available and would be valuable.

Many who care for children lack the skill to measure them accurately, plot measurements on a growth chart, and interpret the data obtained. As the report states, such understanding is essential for growth monitoring. More must be done to make those who look after children aware of the need to measure height accurately and regularly throughout childhood and to train them to do so.

CHRISTOPHER J H KELNAR

Department of Child Life and Health,
University of Edinburgh,
Edinburgh EH9 1UW

- 1 Polnay L. Child health surveillance. *Br Med J* 1989;299:1351-2. (2 December.)
- 2 Hall DMB, ed. *Health for all children, the report of the joint working party on child health surveillance*. Oxford: Oxford University Press, 1989.
- 3 Tanner JM, Whitehouse RH. Revised standards for triceps and subscapular standards in British children. *Arch Dis Child* 1975;50:142-5.
- 4 Tanner JM. *Foetus into man*. 2nd ed. Ware: Castlemead Publications, 1989.

SIR,—*Health For All Children*, discussed by Dr Leon Polnay¹ and Dr D M B Hall,² is the result of a working party set up by groups representing paediatricians and general practitioners, neither of whom is disinterested. The British Paediatric Association suggested some years ago that senior clinical medical officers in the child health service should be replaced by “community paediatricians” who would work part time as paediatricians in the hospital and would take part in the on call duty roster. Similarly, much of the interest in taking over child health surveillance by general practitioners has been tied to the proposal that extra payments would be made for such a service. Practitioners who have a real interest in this work provide such a service already for patients on their lists. Bodies that actually represent the medical officers who work in the child health service were not invited to join the working party.

Child health surveillance requires a different outlook from clinical medicine, and it is not easy for clinicians whose whole training has been directed to the diagnosis and treatment of disease to stop thinking in such terms and abandon their prescription pads. Clinicians are not the most appropriate group to advise on a child health service that they do not fully understand.

Developmental assessment and child health surveillance were pioneered by the former child health group of the Society of Medical Officers of

Health, which started running full time training courses of six weeks' duration for doctors some 30 years ago. In the early 1970s when the Faculty of Community Medicine was formed community health doctors were not eligible for membership. Fortunately, a number of medical schools started to run training courses in child development to fill the need that resulted. There was, however, no organisation or body monitoring the standard or content of those courses, which varied widely.

Following the formation of the Faculty of Community Medicine residual members of the Society of Community Medicine sought to promote the interests of community health as well as community medicine. In 1988 the society (which has since changed its name to the Society of Public Health) was instrumental in establishing a new Faculty of Community Health to produce syllabuses, set standards, and appoint examiners. In future, membership of the Faculty of Community Health should be evidence of eligibility for posts as senior clinical medical officer or as consultant in community child health—more appropriate to the needs of the clients and of the child health and education services than “community paediatricians.”

We hope that this faculty will provide training for general practitioners in child health surveillance and that appropriate diplomas will be established.

P A GARDNER
J S ROBERTSON

Society of Public Health,
London W1N 4DE

- 1 Polnay L. Child health surveillance. *Br Med J* 1989;299:1351-2. (2 December.)
- 2 Hall DMB. Child health surveillance. *Br Med J* 1989;299:1352-3. (2 December.)

Passive smoking and cardiorespiratory health in Scotland

SIR,—In an earlier letter¹ I claimed that misclassification of active smoking state can explain the fact that Mr David J Hole and his colleagues² found weak positive associations between passive smoking and a number of indicators of cardiorespiratory health in the Scottish prospective study. In their reply Mr Hole and colleagues presented calculations to justify their view that the effect of misclassification is to produce “only small biases in the relative risk for passive smokers,” with the reported relative risk “well in excess” of that produced by this form of bias.¹ Unfortunately, these calculations are grossly in error and therefore highly misleading.

The error lies in basing calculations on results for men and women combined without adjustment for sex. Table I of the original paper² shows a clear

TABLE I—“Observed” relative risks for passive smoking for varying denial rates of smoking*

Rate of denial (%)	Relative risks for passive smoking			
	Men	Women	Sexes combined	
			Adjusted†	Unadjusted‡
2	1.74	1.25	1.40	1.12
4	1.95	1.42	1.58	1.18
6	2.06	1.54	1.70	1.20
8	2.11	1.63	1.78	1.22
10	2.15	1.70	1.84	1.23

* Assuming “true” relative risks of 1.0 for passive smoking and 20.0 for active smoking.

† Adjusted for sex using weights $N_1N_2/(N_1+N_2)$, where N_1 and N_2 are the observed numbers of exposed and unexposed subjects. This is a conservative approximation to the true adjusted figure, which cannot be calculated precisely from the data provided by Hole *et al.*²

‡ As given by Hole *et al.*²

association between the smoking habits of the index case and the cohabitee, with the concordance (cross product) ratio being 2.32 for men and 2.19 for women. An appropriate estimate of the concordance ratio for the sexes combined with sex adjustment by the Mantel Haenszel procedure⁴ is 2.25. If, inappropriately, the concordance ratio is calculated from the pooled data, a much lower figure of 1.29 is obtained, and this masks most of the true association. This is important because it can readily be shown that the concordance ratio provides the upper limit to the extent of the observed relative risk from passive smoking due to misclassification of smoking habit (assuming a true relative risk of 1.0). Table I shows that when correctly calculated the observed relative risk can far exceed the value of 1.20 stated by Mr Hole and his colleagues to be “the largest risk to be among passive smokers due to this form of bias.”

The question arises as to the extent that this source of bias can explain all the reported relative risks for active and passive smoking seen in the Scottish study. Table II gives some insight into this question, showing “observed” and “true” relative risks assuming a 4% denial of smoking, a figure consistent with data from many studies of the issue.⁵ Comparing the “observed” relative risks of active and passive smoking with those given in

TABLE II—“Observed” relative risks for passive and active smoking for varying “true” relative risks for active smoking*

	“True” relative risks		“Observed” relative risks	
	Passive smokers	Active smokers	Passive smokers	Active smokers
1		30	1.70	9.17
1		20	1.58	7.88
1		10	1.39	5.62
1		5	1.23	3.63
1		3	1.13	2.50
1		2	1.07	1.81

* Assuming 4% of smokers deny smoking. Results are for sexes combined adjusted for sex as in table I.

table VII of the original paper² shows that there is no problem whatsoever in reconciling the data with the bias hypothesis for most of the cardiorespiratory endpoints. For example, relative risks of 3.77 (active) and 1.21 (passive) for hypersecretion are both very close to the values given in table II for a “true” active risk of 5 (1.23 and 3.63, respectively).

Only two endpoints deserve special comment. The first is death from lung cancer, for which risks of 10.64 (active) and 2.41 (passive) were observed. The confidence interval for the risk with passive smoking was enormously wide (0.45 to 12.83), and the point estimate of risk was higher than that in any of over 20 other, larger, studies on the issue.⁵ I have claimed elsewhere that misclassification of active smoking state can explain the average relative risk for passive smoking of about 1.3-1.5 seen in epidemiological studies.⁵ I retain this view but have never stated that it explained the figure in the Scottish study, of 2.41, to which chance has clearly contributed substantially.

The other endpoint is ischaemic heart disease, for which risks of 2.27 (active) and 2.01 (passive) were observed. Although the risk with passive smoking is significant (95% confidence interval 1.21 to 3.35) and the lower confidence limit is slightly above the bias expected, I do not find this convincing evidence of a true effect of passive smoking. This is partly because the significance level is not high, bearing in mind the number of endpoints studied, and partly because the point estimate of relative risk for passive smoking is difficult to reconcile with that for active smoking, bearing in mind that smokers have much higher active and passive exposure to the constituents of smoke, in the form of both mainstream and sidestream smoke, than do passively exposed non-smokers. More evidence is clearly needed here. The American Cancer Society million person study