in 20 000 live births and this includes both familial and non-familial cases. There were 1068 children born in Seascale between 1950 and 1983.[2] There would have to be a 20-fold increase in incidence before a single case was expected in this population. It is nonsense to state that retinoblastoma should be "generally more common in Seascale." Similar arguments apply to Millom rural district. It is the extreme rarity of retinoblastoma that makes the observation of five cases linked to Seascale so extraordinary and noteworthy.

The ideas in our paper are neither confusing nor contradictory. The data, in fact, strongly suggest a link between retinoblastoma and residence in Seascale. The nature of the association remains to be determined, but it is wrong to imply that radiation can be excluded as the causative agent.

J A MORRIS

Lancaster Moor Hospital,
Lancaster LA1 3JR

JENNIFER BUCKLER

The Surgery,
Kendal LA9 4BD

1 Gardner MJ, Hall AJ, Downes S, Terrell JD. Follow up study of children born elsewhere but attending schools in Seascale, West Cumbria (schools cohort). *BMJ* 1987;**295**:819-22.
2 Gardner MJ, Hall AJ, Downes S, Terrell JD. Follow up study of children born to mothers resident in Seascale, West Cumbria (birth cohort). *BMJ* 1987;**295**:822-7.
3 Gardner MJ, Snee MP, Hall AJ, Powell CA, Downes S, Terrell D. Results of a case-control study of leukaemia and lymphoma among young people near Sellafield nuclear plant in West Cumbria. *BMJ* 1990;**300**:423-9.

Sexual expression in paraplegia

SIR,—Although we welcome the prominence given to an important subject, Dr J M Kellett's editorial on sexual expression in paraplegia[1] contains misleading elements.

Lesions of the cauda equina do not "remove all except seminal emission"; men with complete lesions of the cauda equina often have fully stiff psychogenic erections.[2] The efferent pathway for these erections is presumably the sympathetic erectile pathway demonstrable by stimulation of the hypogastric plexus.[3]

The statement that "lesions above T11 allow reflex erections" is misleading, because lesions as high as T6 may abolish erections if there is descending damage below the primary lesion (as is common in spinal injuries), but complete transections as low as the L5 segment of the cord sometimes permit reflex erections if the sacral segments survive.

It is unfortunate that no mention was made of fertility and parenthood. Reduced fertility in men[4,5] and difficulties with pregnancy and delivery in women[6] are vitally important sexual issues to patients with spinal cord injuries. Important recent developments in both of these have greatly improved this aspect of sexuality.

G S BRINDLEY

MRC Neurological Prostheses Unit,
Institute of Psychiatry,
London SE5 8AF

B P GARDNER

Stoke Mandeville Hospital,
Aylesbury HP21 8AL

1 Kellett JM. Sexual expression in paraplegia. *BMJ* 1990;**301**: 1007-8. (3 November.)
2 Bors E, Comarr AE. Neurological disturbance of sexual function with special reference to 529 patients with spinal cord injury. *Urological Survey* 1960;**10**:191-222.
3 Brindley GS, Sauerwein D, Hendry WF. Hypogastric plexus stimulators for obtaining semen from paraplegic men. *Br J Urol* 1989;**64**:72-7.
4 Brindley GS. The fertility of men with spinal injuries. *Paraplegia* 1984;**22**:337-48.
5 Bennett CJ, Seager SW, Vasher EA, *et al*. Sexual dysfunction and ejaculation in men with spinal cord injury: review. *J Urol* 1987;**139**:453-7.
6 Verduyn WH. Spinal cord injured women, pregnancy and delivery. *Paraplegia* 1986;**24**:231-40.

Fetal and infant origins of adult disease

SIR,—Dr G A Kaplan and Professor J T Salonen found that adults who reported a lower socioeconomic status during childhood had an increased rate of ischaemic heart disease.[1] However, no explanation was proffered for the findings, and data that might provide an explanation, such as the relative weights of subjects in childhood and adulthood, were not reported. In many communities the poor tend to be underweight infants but overweight adults. Abraham *et al* found that such a change substantially increased the risk of cardiovascular disease.[2] A partial explanation may have been provided by Williams, who found that plasma concentrations of high density lipoprotein cholesterol were related more to weight change from the set point weight than to actual weight.[3]

Nevertheless, despite the absence of data on relative weights Professor D J P Barker argued that this study is further evidence that fetal and infant malnutrition manifesting as low birth weight and low infant weight predispose to cardiovascular disease in adult life.[4]

This hypothesis has at least three problems. Firstly, in developing countries infant weight is often considerably lower than it is in developed countries, yet cardiovascular disease is less prevalent. For example, in rural Bangladesh the 50th centiles for weight at birth and at 1 year are 2·5 kg and 6·5 kg; by contrast, the Harvard standard is 3·5 kg at birth and 10 kg at 1 year.[5] Secondly, there have been no reports of increased cardiovascular disease in people subjected to famine prenatally or during infancy. During the Dutch famine of 1944 birth weight and placental weight declined, but no subsequent cardiovascular effects have been detected. In fact, the adult survivors of this intrauterine exposure to famine seem to be indistinguishable in most respects from control cohorts.[6] Similar results were found after food rationing in Great Britain.[7] Thirdly, there is evidence that, except during famine, genetic factors play an important and often decisive part in determining weight at birth and during infancy.[8,9]

I believe that it is only infants who are overweight for genetic reasons who are relatively protected from cardiovascular disease[8-11] and that Professor Barker is wrong when he advocates nutritional intervention to promote a heavier infant weight.[12] Court and Dunlop found that infants who were overweight because of overnutrition had raised plasma lipid concentrations whereas infants who were overweight for genetic reasons had normal plasma lipid concentrations.[13] It seems overwhelmingly clear that overfeeding infants whose "underweight" is genetically determined will increase, not decrease, the risk of cardiovascular disease.

PATRICK J BRADLEY

PO Box 892,
Bondi Junction,
Sydney,
Australia 2022

1 Kaplan GA, Salonen JT. Socioeconomic conditions in childhood and ischaemic heart disease during middle age. *BMJ* 1990; **301**:1121-3. (17 November.)
2 Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. *Health Services and Mental Health Administration Health Reports* 1971;**86**:273-84.
3 Williams PT. Weight set-point theory predicts HDL-cholesterol levels in previously obese long-distance runners. *Int J Obes* 1990;**14**:421-7.
4 Barker DJP. The fetal and infant origins of adult disease. *BMJ* 1990;**301**:1111. (17 November.)
5 Khan M. Infant feeding practices in rural Meheran, Comilla, Bangladesh. *Am J Clin Nutr* 1980;**33**:2356-64.
6 Stein Z, Susser M, Saenger G, *et al*. *Famine and human development: the Dutch hunger winter of 1944/45*. New York: Oxford University Press, 1975.
7 Strachan DP, Hart JT. Fetal and placental size and risk of hypertension in adult life. *BMJ* 1990;**301**:552. (15 September.)
8 Bradley PJ. Weight in infancy and death from ischaemic heart disease. *Lancet* 1989;ii:985.
9 Bradley PJ. Fetal and placental size and risk of hypertension in adult life. *BMJ* 1990;**301**:551. (15 September.)
10 Bradley PJ. Obesity and risk of coronary heart disease in women. *N Engl J Med* 1990;**323**:1144.
11 Bradley PJ. Is obesity an advantageous adaptation? *Int J Obes* 1982;**6**:43-52.
12 Barker DJP, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. *Lancet* 1989;ii:577-80.
13 Court JM, Dunlop M. Obese from infancy: a clinical entity. In: Howard A, ed. *Recent advances in obesity research. I*. London: Newman Publishing, 1975:34-6.

Socioeconomic conditions and ischaemic heart disease

SIR,—Drs George A Kaplan and Jukka T Salonen attempt to deal with the possibility that poor socioeconomic state in childhood is followed by worse conditions in later life.[1] They follow the usual path of controlling for confounding factors by adjusting the odds ratio in a multiple logistic model and conclude that socioeconomic conditions in childhood and adulthood were "independent predictors" of ischaemic heart disease. We suggest that the analyses presented, and the methods used, do not allow this conclusion to be drawn.

The magnitude of the association between a risk factor (exposure) and a disease outcome depends to a large extent on the degree of imprecision in measuring the exposure. In cases in which there is only one risk factor random misclassification of exposure will lead to underestimation of the magnitude of the association between exposure and disease.[2] When two or more potential exposures are strongly correlated, such as childhood and adulthood socioeconomic conditions, the case is more complex. If the exposures are considered together in, for example, a multiple logistic model differential degrees of imprecision of measurement can lead to associations being overestimated, underestimated, or present when in reality they are non-existent.[3] In particular, even small degrees of imprecision can lead to considerable residual confounding even after other exposures have supposedly been adjusted for.[4]

In the study by Drs Kaplan and Salonen an odds ratio of 1·44 associated with markers of poor childhood socioeconomic conditions was reduced to 1·21 after adjustment for a summary index of adult socioeconomic conditions (this is not a 16% reduction as they claim). This gives the impression that the association between childhood socioeconomic state and ischaemic heart disease is largely independent of conditions in later life. Socioeconomic state, however, serves as a proxy measure for factors that directly increase the risk of disease. Improving the categorisation of socioeconomic state—by combining factors such as social class, car ownership, housing tenure, occupational level, etc—greatly enhances the differences seen in mortality risk.[5,6] Thus it is likely that a considerable degree of residual confounding remains after adulthood socioeconomic state has ostensibly been controlled for.

In their study after adjustment for adult socioeconomic state the 95% confidence intervals for the odds ratio for childhood socioeconomic state include 1·0, though the authors do not directly refer to this. But when they found that adjusting for prevalent disease led to a similar change in odds ratio they said that the association was substantially reduced and non-significant. Significance, however, is not a crucial issue in these cases: an association could remain strongly significant and still be entirely due to residual confounding.

Many investigations depend on isolating independent effects from an intercorrelated web of potential risk factors. The identification of these apparently independent effects often better reflects the measurement precision and adequacy of proxy measures of exposures than the underlying causal mechanisms. A more sophisticated analysis is