and increasing numbers of students denied university places, no arguments can justify spending £15m of public money in this way, to say nothing of the appalling wastage of more than £10m of valuable assets at Charterhouse Square. To behave with such reckless abandon at this time of crisis within the universities of this country is surely immoderate.

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Hodgkin's disease and viruses

SIR,—Though I have been retired for eight years and am out of touch, your leading article (27 June, p 2079) on viruses in the aetiology of Hodgkin's disease strikes me with a familiar ring. The uncertainties experienced in "cutting through the forest of information" in Hodgkin's disease have gone on so long that we have either failed to bring out significant facts or are attempting to fit observation and experiment into an outmoded view. Perhaps we need a more compatible hypothesis as much as new information.

Cancerous reactions are most frequent where repeated demands are made for replacement, repair, and cyclic growth. Multiple initiating agents of varying potency making repeated proliferative demands may lead to disordered growth. This is generally accepted in epithelial neoplasia, even if by no more than the growing acknowledgment of the importance of environmental factors. I suggested that Hodgkin's disease might be in this mainstream of malignant development, attributing its origin to sustained lymphoproliferative demand and its manifestations to multicentric origin in patients and in lymph-node groups of varying susceptibility, the process not only affecting normal control but promoting immunologically competent tumour-versus-host reactions. To me Epstein-Barr virus in Hodgkin's disease seemed comparable to, say, naphthylamine in urothelial malignancy—just one of the more active pressures disrupting normal control. The search for *the* malignant cell in a mixed-cell tumour response seemed unlikely to bring about any great advance. Reed-Sternberg cells, for example, though characteristic of Hodgkin's disease, are sometimes difficult to find and are hardly good candidates for chief culprit, let alone for fitting any monoclonal hypothesis.

I summarised these views in a chapter on nature and aetiology in Hodgkin's disease.[1] Looking back on nearly 30 years of emphasising the need to understand normal growth control mechanisms and the means of their disruption in cancerous reactions rather than to cry "this is the cause" of cancers, I note that in Hodgkin's disease they do not seem even to rate dismissal.

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[1] Smithers D. In: Smithers D, ed. *Hodgkin's disease*. Edinburgh: Churchill Livingstone, 1973:3-10.

Management of patients after mastectomy

SIR,—I read with interest the article by Dr P B Clark and Mr D L Morris (27 June, p 2095) on the management of patients after mastectomy. They comment on the pick-up rate for recurrent carcinoma; however, there is no discussion of the development of a second carcinoma in the contralateral breast and its early diagnosis. The incidence is said to be of the order of 7·5%[1]—surely a good basis for screening an at-risk group.

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[1] Rains AJH, Ritchie HD, revisers. Bailey and Love's *Short practice of surgery*. 17th ed. London: HK Lewis, 1977:661.

Phantom breast sensations

SIR,—Although Minerva (13 June, p 1982) is quite correct in saying that women regard phantom breast symptoms as trivial she is wrong in thinking them to be rare.

We asked 100 consecutive women who had had a mastectomy at least one year previously about their experience of phantom breast sensations. Forty-one patients had experienced phantom symptoms at some time since their mastectomy. The patients with phantoms were younger—49 ± 11 *v* 56 ± 12 years—and more often married—34/11 (86%) *v* 39/59 (66%)—at the time of mastectomy than the others. There was no apparent relationship with the type of mastectomy performed, interval since mastectomy, or recurrent disease. Patients with phantoms did not appear to be more anxious or have greater psychiatric morbidity.

The phantom symptoms were usually transient, lasting a few seconds, and consisted of either an awareness of the breast or a tingling sensation; only eight patients described the experience as unpleasant and three as painful. The whole breast was involved in 32 patients and the nipple only in eight, and in one the symptom was confined to the site of the original cancer. In some premenopausal patients the symptom was experienced only in the luteal phase of the menstrual cycle.

Only two patients had considered the symptoms sufficiently important to mention them to their medical attendants, which is the likely reason why this common syndrome is so little recognised.

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Levodopa: long-term impact on Parkinson's disease

SIR,—Dr G O Tippett's letter (6 June, p 1875) is a timely reminder that symptoms of postural dizziness in a Parkinsonian patient should raise the possibility of the Shy-Drager syndrome or multiple system atrophy with autonomic failure. This applies even if the symptoms arise only after levodopa therapy and even if there are at that stage no other neurological signs. It should, however, be stressed that autonomic failure may also occasionally occur with otherwise typical idiopathic Parkinsonism; and since the prognosis for this variety is much better than that of the Shy-Drager syndrome and there is a better response to anti-Parkinsonian drugs, attempts at treatment are well worth pursuing.

Dr Tippett goes on to describe the problems of management of the postural hypotension and the abandonment of treatment with 0·5 mg of fludrocortisone, which raised his wife's systolic blood pressure so much that it was stopped for fear of causing a cerebral haemorrhage. The first line of treatment should be by means of head-up tilt at night, a technique which by reversing the normal loss of sodium and fluid in recumbency may well abolish postural hypotensive symptoms in such patients, at any rate for a time.[1] If this fails fludrocortisone has a beneficial effect in autonomic failure in a much smaller dose of 0·1 mg, which does not lead to fluid retention. The improvement in blood pressure control is probably due to increasing the sensitivity of sympathetic receptors to small amounts of noradrenaline.[2]

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[1] Bannister R, Ardill L, Fentem P. *Q J Med* 1969;38:377-95.
[2] Davies B, Bannister R, Sever P. *Br J Clin Pharmac* 1979;8:253-60.

New uncertainties in prenatal screening for neural tube defect

SIR,—Dr D J H Brock (6 June, p 1870) deserves the lion's share of the credit for the present system of screening for neural tube defect on the basis of maternal serum α-fetoprotein, and we do not wish to make him unhappy because we suggest that this may no longer be the only screening method to consider. We did not suggest in our review (2 May, p 1416) that α-fetoprotein screening should be discarded immediately and everywhere in favour of ultrasound. We have no doubt that well-organised α-fetoprotein screening in areas of high incidence of neural tube defect (for example, the Glasgow programme[1]) performs a valuable service. We do wonder how many screening programmes are adequately organised to keep maternal stress down to acceptable levels, and we feel that there is room for doubt about the wisdom of expanding screening services further in low-incidence areas, some of which appear to have tried and abandoned screening. We pointed out a number of factors suggesting that low incidence is increasingly the norm; and, while we agree with Dr Brock that a drop in the incidence of neural tube defect is unfavourable for any screening method, the effect is more serious for α-fetoprotein screening than for ultrasound, which has many other applications.

Dr Brock objects to our statement that when the amniotic fluid shows raised α-fetoprotein but normal acetylcholinesterase (or only a faint second band) the fetus is likely to have exomphalos, gastroschisis, etc. The UK Collaborative Acetylcholinesterase Study (to be published in the *Lancet*) has shown that about three-quarters of cases with exomphalos show a second band (acetylcholinesterase positive); but in our experience this band, when present, is so faint that these cases can usually be distinguished prospectively from cases of neural tube defect. Unlike Dr Brock, we do not find that congenital nephrotic syndrome is the most likely diagnosis in cases with raised α-fetoprotein but normal acetylcholinesterase or only a faint second band. In 17 women in 1980 who had raised α-fetoprotein and normal acetylcholinesterase (one band) or only a very faint second band on acetylcholinesterase gel, there were two cases of exomphalos, two of gastroschisis, one of