local associations, sometimes even against the advice of local doctors. The monitors, usually abdominal respiration detectors,[7] are simple to use, requiring little instruction, and the parents have the common sense to realise that, although survival cannot be guaranteed, it is more likely with a monitor than without.

Although a controlled trial is not possible, steadily increasing use of monitors throughout the world will eventually produce a consensus about their value. After all, there has never been a controlled trial of the efficacy of life jackets.

B M WRIGHT

Ricksmansworth,
Herts WD3 2DQ

1 Minerva. *Br Med J* 1982;**285**:1362.
2 Wright BM. Near miss cot deaths and home monitoring. *Br Med J* 1982;**285**:1743 (correction **286**:61).
3 Southall DP, Richards JM, Lau KC, *et al*. An explanation for failure of impedance apnoea alarm systems. *Arch Dis Child* 1980;**55**:63-5.
4 Lucey JF. False alarms in the nursery. *Pediatrics* 1978;**61**:665-6.
5 Kelly D, Shannon DC. Neonatal and infantile apnea. In: Milunsky A, *et al*, eds. *Advances in perinatal medicine*. Vol 1. New York: Plenum Press, 1981.
6 Mackay M, Abreu FA, Silva E, *et al*. Home monitoring for central apnoea. *Arch Dis Child* 1984;**59**: 136-42.
7 Wright BM. An abdominal respiration detector. *J Physiol* 1977;**271**:11-12P.

Rifampicin in non-tuberculous infections

SIR,—Drs A J Davies and D A Lewis come down in favour of advising against the routine use of rifampicin in non-tuberculous infections (7 July, p 3), although it seemed a close run thing until their last paragraph. Their restriction should perhaps apply to non-mycobacterial infections since rifampicin is of such enormous value in both tuberculosis and leprosy.

The reasons for the same decision in most developing countries are somewhat different. While accepting that anxiety about emergent resistance in mycobacteria is a major reason to restrict the use of the drug, they admit that the evidence that this is a problem at present is slim. Only 0·2% of routine cultures from Zambian patients with pulmonary tuberculosis have shown rifampicin resistance, but concern about the possibility of an increase is valid. In addition the number of reported cases of leprosy with rifampicin resistant organisms is growing steadily. Unfortunately in many developing countries rifampicin is widely available over or under the counter and it has a widespread reputation as a cure all. This general availability of the drug is virtually impossible to curtail, and its long term effect on bacterial resistance can only be left to speculation.

An equally important argument, however, is the economic one. Rifampicin is undoubtedly costly. Many countries are unable to buy it as a first line drug for tuberculosis. Others have adopted short course regimens such as that now used for sputum positive adults in Zambia (two months of rifampicin, streptomycin, pyrazinamide, isoniazid, and thiacetazone followed by six months of isoniazid and thiacetazone). But even when used for only two months this is a considerable strain on the very tight allocation of funds for drug purchasing. In 1982 the World Health Organisation urged all member states to introduce multiple drug treatment for leprosy, and the most important drug in the two recommended regimens is rifampicin. Many countries have still been unable to implement this policy nationwide. There are so few effective drugs against *Mycobacterium leprae*, and rifampicin has such a vastly superior bactericidal action to any other known agent, that it is both therapeutically sound as well as economically necessary to restrict—as does the *Zambian National Formulary*—the use of the drug to tuberculosis and leprosy.

R DE SOLDENHOFF

Ministry of Health,
PO Box 30205,
Lusaka, Zambia

Which patients are cured of breast cancer?

SIR,—We were interested by the report from Mr I S Fentiman and his colleagues on patients surviving more than 20 years from diagnosis of breast cancer (27 October, p 1108). We are concerned, however, that they have drawn negative conclusions from data that have weaknesses and deficiencies. For example, they admit that 26 (14·8%) deaths within 20 years were from causes other than breast cancer. Also no histopathological data (table VI) were available in 30 (17·0%) of the patients who died within 20 years. Would they explain how an analysis of survival from disease has validity, in particular as a comment on histological characteristics and prognosis, when no allowance is made for these facts?

We have presented evidence for the importance of histopathology in long term prognosis of breast cancer from a similar study of 133 patients surviving 17 to 20 years after treatment for breast cancer in Edinburgh.[1] The same data were obtained for 200 consecutive patients treated in the same way and diagnosed from the same period but dying within 10 years from breast cancer. The findings in the two groups with long and short survival were compared by χ^2 tests, and our results are at variance with those in the report of the Guy's group in several respects. Some of our results are presented in the accompanying table for comparison.

We found that tumour grades differed significantly between the groups and that certain histological types of invasive cancer were significantly more common among the long term survivors. In addition to the lobular, medullary, and mucinous types recognised by Mr Fentiman and others, "special type" invasive tumours of cribriform, tubular, and papillary type were seen, but only in long term survivors. No significant differences were observed in age distribution or menstrual state of the two groups.

Different interpretations are applied to the term "cure" for breast cancer.[2] We are surprised that no reference was made to the findings of Brinkley and Haybittle that "statistical" cure could not be shown for their series, in which excess mortality persisted beyond 25 years of follow up.[3] As one quarter of women surviving 20 years in the Cambridge series had positive axillary lymph nodes at the time of initial treatment[4] it cannot be asserted that lymph node metastasis is a localised phenomenon, as Mr Fentiman and others have done. We also request substantiation for the statement "Premenopausal patients may develop tumours that are intrinsically less malignant, or possibly may mount a more effective response to their tumour," which appears under Results and is repeated in the Discussion. Indeed, there are several discrepancies and ambiguities within the text and figures which we find difficult to accept.

Our data, which will shortly be published, suggest a very different conclusion from that of Mr Fentiman and his colleagues. Thus we have not found age and menopausal state to be of value in predicting long term survival and, furthermore, have found that histological analysis of the primary tumour identifies many of those patients who will survive long periods after treatment for breast cancer.

THOMAS ANDERSON
DAVID PAGE

Department of Pathology

J MICHAEL DIXON
A P M FORREST

Department of Clinical Surgery

HELEN J STEWART

Department of Radiation Oncology

R A ELTON

Unit of Medical Computing and Statistics,
University of Edinburgh,
Edinburgh

1 Page DL, Dixon JM, Anderson TJ, Stewart HJ, Forrest APM. Long-term survivors from breast cancer: a histological review. *Lab Invest* 1983;**48**: 66A.
2 Haybittle JL. The evidence for cure in female breast cancer. In: Bulbrook RD, Taylor DJ, eds. *Commentaries on research in breast disease*. Vol III. New York: Alan Liss, 1983:181.
3 Brinkley D, Haybittle JL. Long term survival of women with breast cancer. *Lancet* 1984;i:1118.
4 Brinkley D, Haybittle JL. The curability of breast cancer. *Lancet* 1975;ii:95-7.

**Mr Fentiman and his colleagues reply below.—ED, *BMJ*.

SIR,—Dr Anderson and his colleagues have raised a number of questions. We will try to answer them.

Firstly, it is generally agreed that, owing to ambiguities and inaccuracies in death certification, both cancer related and non-cancer related deaths should be included in an analysis of death rates because this method introduces least bias.[1]

Secondly, histopathological review could not be conducted on 30 of those who died because the slides were unobtainable, but we

Comparison of results of Edinburgh and Guy's trials

	Edinburgh		Guy's
	No (%) of long term survivors	No (%) of short term survivors	No (%) of long term survivors
False positive diagnosis (histopathological review)	14	0	3
Non-invasive carcinoma	7	0	9
Total with invasive carcinoma	599		227
Studied "survivor" groups	112	200	51
Clinical T3/T4	11 (9·8)	58 (29·0)	9 (17·6)
Clinical node positive	32 (28·6)	78 (39·0)	24 (47·1)
Histopathology:			
Invasive ducta:	56 (50·0)	195 (97·5)	40 (78·4)
Grade 1	7 (12·5)	6 (3·1)	5 (12·5)
Grade 2	35 (62·5)	70 (35·9)	22 (55·0)
Grade 3	14 (25·0)	119 (61·0)	13 (32·5)
Invasive lobular carcinoma	6 (5·4)	1 (0·5)	6 (11·8)
Invasive special type carcinoma	50 (44·6)	4 (2·0)	5 (9·8)