principle, if it moves give it drugs, and if it stands still give it ECT. I would like to think that was just a very very bad and unpleasant joke, but I'm afraid it's not."

By now, sir, you have probably printed most of that particular programme in your columns and I hope it has served a useful purpose. It's certainly one way of making sure that doctors are aware of what is to be found on television if they are unable to watch.

One final point. I did not say that I feel a need to worry people. I said that the BBC could not take as its guiding principle "Thou shalt not worry people." The two statements are rather different.

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Life with spina bifida

SIR,—The management protocols adopted by some paediatricians, as described by Professor R B Zachary in his article on spina bifida (3 December, p 1460), raise important ethical issues. In cases where curative medicine has nothing more to offer it is accepted as good medical practice to see that patients die in comfort and with dignity. Furthermore, it is also accepted that when a patient's life is merely being prolonged by the sophisticated techniques of intensive care, with no prospect of restoring health, those techniques should be withdrawn.

However, actively, wilfully, and deliberately to employ a regimen, from whatever motive, designed to cause death introduces an entirely new ethic into medicine. It is one which will be regarded by many patients and doctors with profound disquiet. Once it becomes established in one area, what logical grounds are there for not extending such a seemingly compassionate approach to other areas?

One argument against voluntary euthanasia is that once doctors are allowed to end life on request they will eventually do it at their discretion. If the regimens described by Professor Zachary are in fact prescribed that argument is incorrect—it is already happening.

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SIR,—The considerable interest aroused by Professor R B Zachary's statement (3 December, p 1460) that many children born with severe spina bifida are sedated or starved to death, or both, is rather belated, since I made the same point myself almost two years ago,¹ adding for good measure that the Department of Health and Social Security in its booklet "Care of the Child with Spina Bifida" gives its tacit approval to this policy. I did so to demonstrate the ethical confusion which prevails in this area, since the DHSS ostensibly follows a policy of "preserve at all costs" in the case of late abortions in the grey area between fetushood and infancy. Professor Zachary shares this confusion, though he tries hard to conceal it.

There are patients of all ages who prompt their doctors, their relations, and society as a whole to ask whether their death will come as a release or whether it is a tragedy which should be actively avoided or postponed if at all possible. Sometimes the patient himself can

ask and answer the question, but modern medicine keeps many people alive who cannot have any say in what is done to them. It is evident that Professor Zachary does not believe that all cases of spina bifida merit active therapeutic intervention, but unless his clinical judgment is infallible some of those whom he rejects will surprise him by living on with very severe physical and mental handicaps. If this outcome gives him even qualified pleasure, let him say so, especially to the parents, and let him come out unequivocally against the antenatal diagnosis of any disease.

I strongly suspect, however, that Professor Zachary, like most people, believes that some patients should be allowed to die and is not against eugenic abortion. If this is so, it means that he must make distinctions between those whom he wants to see survive and those whom he hopes will die. The major difference then is the manner of death: slow, uncertain, and distressing versus quick, certain, and somewhat less distressing. The argument that the active killing of a patient is a crime, while encouraging him to die by deliberate inactivity is not, is as wrong as it is prevalent. Let anyone who disagrees try it out the next time he causes the death of a child with meningitis by refusing—or even failing—to give treatment. It is sad to see Professor Zachary falling into this venerable trap.

I quite agree that, as with neonatal circumcision, sedation in these cases is therapeutically superfluous, since what is not remembered cannot be called suffering. The sedatives are clearly given to reduce the anxieties of the doctors and nurses, but surely there is nothing very new in that.

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¹ Brewer, C, *Guardian*, 4 February 1976.

Indications for prenatal chromosome analysis

SIR,—Professor J Philip and his colleagues claim (29 October, p 1117) that they have found similar incidences of chromosomal abnormalities in amniotic cell cultures from women with increased risk of giving birth to a child with such abnormalities and from those without (1.2% and 1.4% respectively). They suggest that current criteria for the selection of pregnant women for amniocentesis may need revision, though they do recognise the need for a larger study. Although there will doubtless be some refinement of criteria in the future, we consider that the implication that any major revision may be necessary is not justified by their findings, which can be interpreted quite differently. It would be unfortunate if clinicians were to be deterred from offering amniocentesis to their patients as a result of accepting the authors' interpretation of their results.

The difficulties in the interpretation of their data are threefold. Firstly, they pool several disparate "high-risk" groups to obtain their overall incidence of chromosomal abnormality of 1.2%. This is misleading because both their observed incidences and those that would have been anticipated from earlier studies vary considerably. If, for example, the two groups with the lowest expected risk—namely, mothers of 35-39 years and those with a previous chromosomally abnormal child—are excluded, then the incidence among the remaining groups becomes 6.25%. In fact the incidence of 0.2% among these two groups

with only slightly increased risks does not differ greatly from that which would be expected from either retrospective or prospective studies, about 0.5-1.0%.

Secondly, the 1.4% incidence in their low-risk groups must be attributed to chance. It derives entirely from five instances of sex chromosomal anomaly, three of which occurred in their "Down's syndrome in the family" group which could arguably be placed with the increased-risk groups. The figure is higher than would be expected from surveys of consecutive neonates or from the limited figures available from prospective studies of amniocentesis on women not at increased risk of a fetus with a chromosomal abnormality.

Thirdly, the authors quote several such prospective studies in support of their own figures and in doing so have again pooled different groups at high risk to arrive at an overall incidence of 2.6%, including their own data, compared with 0.96% for patients with no increased risk. The assignment of high- and low-risk groups of patients from these and other studies can be juggled to give figures more or less favourable to the authors' case.

In conclusion, while strongly endorsing the plea for larger series to be reported, we would in the meantime advise caution in proposing even tentatively suggestions for revising the criteria for amniocentesis.

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False-negative results in urine testing for glucose

SIR,—Three patients in our group practice (two of them known diabetics) have recently been found to have definite glycosuria on testing with Clinistix strips, but no glycosuria was indicated with Diastix and Labstix. I understand from the manufacturers that similar findings have been reported by other doctors. One of the causes of discrepancy may be synergistic action between various drugs currently being prescribed for the patient.

I would suggest that Labstix and Diastix are not suitable for use as a screening test for glycosuria.

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Survival of infants of 1000 g birth weight or less

SIR,—We read with great interest the paper by Dr R R Gordon on neonatal mortality in England and Wales (5 November, p 1202). It was of particular interest to us to learn that the neonatal mortality rate for infants of 1000 g birth weight or less was 77.33% in England and Wales in 1975. As the figure for 1964 was 83.97% Dr Gordon rightly comments that on a community basis super-intensive care of the very-low-birth-weight infant does not appear to have improved survival rates. We are, however, not told how many of the 1416 infants weighing 1000 g or less received expert care in a perinatal or neonatal centre.

Our recent experience in San Francisco has been that intensive management of these small infants (500-1000 g birth weight) results in survival of more than 60% (see table). Infants born outside the regional perinatal centre

Neonatal mortality of infants of 500-1000 g birth weight admitted to intensive care unit, San Francisco Children's Hospital

Year	Inborn		Outborn		Total Survival
	No	Lived	No	Lived	
1972	15	2	12	3	5 (18%)
1973	10	0	9	4	4 (21%)
1974	6	1	9	1	2 (13%)
1975	4	2	12	4	6 (37%)
1976	13	7	20	12	19 (58%)
1977 (to November)	11	9	12	7	16 (70%)

("outborn") fare less well (during 1976-November 1977 59% survival compared with 67% for those inborn). As regards morbidity, we have been able to follow up 13 of 17 inborn infants in this weight group for periods ranging from six months to six years and found 12 to appear normal. Of 29 outborn infants, 22 have been followed up to the same age and 14 of these were considered normal.

Our mortality figures up to 1975 are similar to those for England and Wales reported by Dr Gordon, but in 1976 and 1977 there appears to have been a marked improvement in outlook for these tiny infants. This we attribute to more thorough intensive care involving mechanical ventilation for considerable periods of time and careful use of intravenous feeding, though it is possible that other factors have played a part.

The problem is not that intensive care cannot improve survival for these small infants but rather that these facilities are not available to all mothers about to deliver prematurely at any time after 26 weeks of gestation. We are firmly convinced that all high-risk mothers should be delivered in regional centres where intensive care facilities are available. When it is not possible to use the uterus as the transport incubator, then early transfer of these immature babies to a regional newborn intensive care unit in well-equipped ambulances is the next best alternative.

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Perinatal mortality rates by birth weight

SIR,—Dr Ronald R Gordon has drawn attention recently (5 November, p 1202; 8 October, p 939; 21 May, p 1313) to the disproportionately greater fall in the number of "small" (less than 1000 g) live births than total births which has occurred since 1962 in Sheffield and to a lesser extent in England and Wales.

Drs R Newcombe and I Chalmers (8 October, p 925) have shown, however, that in Cardiff the trend appears to be different from that in Sheffield. Using data on firstborn singleton infants born to primiparous mothers during the decade 1965-74, they have demonstrated a 12% increase in the proportion of infants weighing less than 2500 g during the second quinquennium. This increase in the proportion of low birth weight (LBW) babies in Cardiff would have tended to increase the

perinatal morbidity, and a previous study by Dr Chalmers and his colleagues has shown that there has been little reduction in the perinatal mortality rates there during the decade 1965-74.

The local experience in Wolverhampton appears to follow the Cardiff rather than the Sheffield trend when the total births among residents in the Wolverhampton Health Area are examined using the LHS27 returns made annually to the Department of Health and Social Security. The accompanying figure uses the two-year moving means because the annual numbers are small and shows that, although the total number of births began to fall from 1968, the number of LBW babies did not begin to fall until 1972, whereas in Sheffield this fall began in 1962 well in advance of the fall in total births.

Live births to Wolverhampton residents (two-year moving averages).

Furthermore, perinatal deaths among Wolverhampton residents have been monitored through a local study since 1970 and it is clear that the perinatal mortality rates for babies of mothers born abroad and for babies of mothers born in the UK do not differ greatly. For example, when births and perinatal deaths are aggregated for 1970-6 the stillbirth rates are respectively 17 and 17 per 1000 (all mothers: 17 per 1000) and the perinatal (stillbirths and first-day deaths) rates are 27 and 24 per 1000 (all mothers: 24 per 1000). Using the data from the Office of Population Censuses and Surveys for the same period, the perinatal rate for all mothers is 30 per 1000 and it would seem reasonable to assume that within this population the 75% of babies from mothers born in the UK have a perinatal mortality rate of 30 and the 25% of babies from mothers born abroad (mainly India) have a rate of 33 per 1000.

At a national level the decline in annual births during the 1970s (a drop of 25% in six years) has been the result of a very substantial decrease in births in the lower (manual) social classes and a smaller decline in the higher (non-manual) social classes.¹ As the proportion of socially disadvantaged mothers is greatest in social classes IV and V it would be expected that both the proportion of LBW babies and the associated perinatal morbidity would be reduced during the period of falling births. It is paradoxical, however, that in some areas like Wolverhampton and Cardiff there has been

little change during the last decade in either the proportion of LBW babies or the perinatal mortality rate.

The obvious conjecture is that there are factors operating to maintain the raised level of LBW babies and the perinatal mortality associated with them, so that as the LBW babies attributable to socially disadvantaged mothers are reduced they appear to be replaced by babies whose mothers come from higher social classes. These factors, as yet unidentified, could be a reason why the proportion of LBW babies and the associated perinatal mortality have not fallen generally in England and Wales as much as, for example, in Sheffield.

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¹ Pearce, D, and Brittain, M, *Population Trends*, 1977, No 7.

Still dissolving discs?

SIR,—Mr E Shephard (26 November, p 1419) says that "95% of our patients" recover with conservative treatment "at the proper time." Lucky man! Prolapsed intervertebral disc is ideally an operative diagnosis; otherwise the prolapse may be located by myelography, but the latter is usually reserved as a preliminary to operation. Therefore most patients treated conservatively have no objective evidence for the diagnosis, however suggestive the clinical signs may be. Can Mr Shephard say what happens to his patients after they recover? How many attacks do they have later, how long do they last, and how much working time is lost in total? What degree of disability qualifies as "recovery"? And how long is "the proper time"?

I think that no answers to these questions have been published. The painful disc with no nerve supply¹ and disc prolapse remaining when the patient has recovered² are only two points which make this a difficult subject to analyse. Very little has been reported about the natural history of low back pain, and without that knowledge it seems difficult to argue that any method of treatment is superior or even effective.

E C CAWTE

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¹ Kumar, S, and Davis, P R, *Journal of Anatomy*, 1973, 114, 47.

² Falconer, M A, McGeorge, M, and Begg, A C, *Journal of Neurology, Neurosurgery and Psychiatry*, 1948, 11, 13.

Ganglia

SIR,—Your leading article (26 November, p 1370) ignores a number of advances in the understanding of the mechanism and treatment of ganglia.

As long ago as 1840 Adams¹ suggested that popliteal cysts were connected to the joint by a valvular opening and in 1970 Jayson and Dixon² demonstrated valvular connections allowing effusions to be pumped from the knees into the cysts but not to return. They suggested that the valve might be of the Bunsen type, consisting of a narrow curved passage the walls of which collapse under direct cyst pressure. A similar pathogenesis for ganglia seemed likely, but this was not