Living wills and treatment refusal

SIR,—Dr Roger Higgs (14 November, p 1221) has well summarised current views on living wills, but it is a pity that he draws on "the rising numbers of the elderly suffering from dementia" to justify their use. Although the linking theme between all the groups he mentions, demented patients being the largest, is loss of mental competence, there are important differences in practice. Dementia does not arrive fully fledged, causing a "vegetable" state. It is therefore misleading to consider the implications for the demented patient in the same fashion as those for someone with, say, acute stroke or a terminally confused victim of carcinoma.

Those with dementia suffer the usual illnesses of the elderly. When these are life threatening or devastatingly disabling—for example, a major stroke—I think most psychogeriatricians take a conservative approach after discussion with relatives. Euthanasia in the proper sense—peaceful death—would be practised. It is hard to see what extra protection a living will would offer unless, say, relatives pressed for heroic measures—which is unlikely in my experience. With less serious conditions, such as a simple chest infection, withholding treatment, as would presumably be more likely with a living will lurking in the casenotes, leads to protracted discomfort and the patient frequently survives anyway. With dementia we are a long way from Dr Higgs's hoped for prognostic precision.

The application of living wills to dementia could render more likely the wrong form of euthanasia: assisted death. Would we be compelled, at some arbitrary point, to withhold nursing care, long term medication, or feeding and wait for death by inanition? If so then it could be argued that a little "help on the way" would be a more humane option.

Health professionals often impute suffering to demented patients, even when none is apparent. I suspect this is our own countertransference—that is, the distress is ours. This should be recognised and not submerged beneath apparently humane attempts to shorten life such as have previously been argued in these columns.¹ Dr Higgs raises some important dilemmas, but they are not clarified by the inclusion of patients with dementia. Until effective treatments have been found, the key issues for them will continue to be the provision of quality long term care.

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1 Robertson RG. Ethical dilemmas of brain failure in the elderly. *Br Med J* 1983;287:1775-7.

Deaths in children with inguinal hernia

SIR,—As Dr H G Pledger and colleagues did for acute appendicitis (14 November, p 1233), we examined deaths in children aged 15 years and under who died with a diagnosis of inguinal hernia between 1980 and 1984.

We obtained copies of 11 death certificates with this diagnosis from the Office of Population, Censuses, and Surveys and requested hospital notes for those children dying in hospital. We reviewed the notes to see whether we could learn anything to help future management of these patients.

Eight of the 11 deaths were of babies aged less than 1 year old and one occurred in a severely handicapped adolescent. Ten patients were boys, one a girl. One 2 month old child's notes were not traced.

Two young children (one aged 15 months, one 32 months) died at home; both were found at necropsy to have strangulated inguinal hernias, with inhalation of vomit as the cause of death. Two patients died shortly after birth, in hospital. Both had severe congenital malformations with associated hernia. Of the remaining children dying in hospital, only one died in a specialist paediatric centre. Three babies (aged 4 weeks, 6 months, and 10 months) died on arrival at hospital or shortly thereafter from cardiorespiratory collapse. All had been unwell at home for several days before admission with gastrointestinal symptoms, and in two the presence of inguinal hernias had been noted on previous hospital examinations.

Two babies, one of 6 weeks and the other aged 2 months, died in hospital after repair of irreducible inguinal hernia. One had been sent to the ward after the operation breathing spontaneously with an endotracheal tube in place because of respiratory difficulties. Unfortunately, the tube blocked and the child suffered a cardiorespiratory arrest. He died after several weeks, having suffered irreversible brain damage. The other baby was sent back to the surgical ward, although noted to be cyanosed; he subsequently suffered a cardiorespiratory arrest from which he could not be resuscitated. A severely mentally and physically handicapped boy was difficult to examine because of hip contractures and was treated for constipation until he suffered a cardiac arrest and died; he was found at necropsy to have a strangulated hernia with perforated bowel.

Between 1980 and 1984 about 9000 hernia repairs per year were carried out on children aged under 15.¹ Disaster is rare but does occur. In common with death from appendicitis, it is most often due to difficulty in diagnosis and delay in appropriate treatment, both at home and in hospital. It is thus frequently preventable. We believe that the importance of gastrointestinal symptoms in children with previously diagnosed hernia should be emphasised to parents. Equally, the importance of appropriate monitoring and adequate fluid and general resuscitation should be underlined for medical staff in hospital. It is a matter of debate whether specialised paediatric centres should deal with all young infants, in whom the risks of operation for hernia repair are considerably greater than in older children.

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1 Department of Health and Social Security. *Hospital in-patient enquiry*. London: DHSS, 1980-4.

Penile cancer and smoking

SIR,—Dr Dan Hellberg and colleagues (21 November, p 1306) report an association between smoking and penile cancer. Their table V, which presents the key results, appears to contain several errors: (a) the numbers of smoking, ex-smoking, and non-smoking cases do not add up to the total number of cases; (b) the numbers of smokers broken down by amount smoked do not add to the total numbers of smokers for controls or cases; (c) the relative risk for smokers of more than 10 cigarettes a day is shown as statistically significant but calculations based on the numbers of cases and controls given in the table do not remotely suggest significance. It would be valuable to see the correct table to understand the data better.

The authors report a strong association between phimosis and penile cancer and note an association

between phimosis and smoking. After adjustment for phimosis (and balanitis) the relative risk of penile cancer from smoking was noted to reduce from 2.22 to 1.53. Adjustment for inaccurately measured variables tends to be incomplete and to leave a residual artefactual association. How accurately is phimosis recorded by questionnaire? Might the association of smoking to penile cancer be such an artefact?

Recording of smoking habits seems to have been made without reference to the time of onset of disease. Might not the presence of disease have affected smoking habits or have affected the accuracy with which smoking habits were reported? Was any attempt made to validate reported smoking habits or to assess whether smoking habits changed after the onset of cancer?

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AUTHORS' REPLY—In the group of cases and controls who smoked more than 10 cigarettes a day the figures were wrongly printed, and we hope that Mr Lee's questions are clearly answered by the table below.

Mr Lee has two other major questions. Firstly, after adjustment for phimosis and (of less importance) balanitis the relative risk by smoking was reduced. In the questionnaire phimosis was explained in easy terms so that any man with a normal mental capacity would understand the question. If some did misunderstand results on phimosis in general are unlikely to have been affected more than marginally and the subsequent logistic regression analysis would probably have reduced the relative risk further. The potential bias of the question not being understood should have worked equally on both cases and controls.

Secondly, smoking habits were calculated from the onset of smoking to the onset of disease. Thus, presence of disease could not have affected the results on smoking habits. We have therefore had no reason or need to validate smoking habits before and after the onset of cancer.

Finally, the crude additive relative risk for men who both had phimosis and smoked more than 10 cigarettes a day was calculated to be 52.7

Distribution of smoking habits among men with penile cancer compared with that among matched controls. Values are numbers of men (percentages) (corrected version of table V, 21 November, p 1306)

Smoking habit	Men with penile cancer (n=223)	Controls (n=201)	Relative risk (95% confidence interval)
Non-smoker	67 (30)	82 (41)	1.00
Ex-smoker	28 (13)	21 (10)	1.71 (0.88 to 3.30)
Smoker:	128 (57)	98 (49)	1.61 (1.08-2.41)
1-10 cigarettes/day	57 (26)	59 (30)	1.18 (0.72-1.92)
>10 cigarettes/day	71 (31)	39 (19)	2.22 (1.34 to 3.69)

Contingency $\chi^2=4.89$, $df=3$, $p<0.05$, for comparison of smokers with non-smokers.

(unmatched analysis, not included in the paper). An analysis to determine whether there was in fact a synergistic effect between phimosis and smoking gave a relative risk of 55.5. This increase is, of course, not significant, and we cannot conclude therefore that there was such a synergistic effect. Smokers of more than 10 cigarettes a day who had not had phimosis, however, were so few ($n=14$) that we cannot exclude the possibility of a true synergistic effect being found in a larger study. In that case a logistic regression analysis adjusting for the "confounding effect" of phimosis might not be