mentioned, nor are the criteria for transfer to oral anticoagulation. A difference in duration of treatment between the groups may be important in interpreting both the phlebographic findings and the incidence of complications. The data on total and daily heparin dosage suggest that those treated with subcutaneous calcium heparin had a mean of 13·8 days of treatment compared with only 10·7 days for the intravenous sodium heparin group—that is, 29% longer. Standard deviations of the dosages are large: that for the total dose of subcutaneous calcium heparin is 33% of the mean. This suggests that there was either a very wide variation in the daily dose, not indicated in the standard deviation of that figure, or that there was a considerable range of duration of treatment for the calcium heparin group.

In addition, the mean daily dose of calcium heparin was 20% greater than that of the sodium heparin, and the mean total dose given was 56% more. Thus the calcium heparin group seem to have had the advantages of more than three days longer receiving heparin treatment, with its fibrinolytic properties, and an appreciably greater total dose. It may well be that those in the subcutaneous heparin group were also more effectively anticoagulated, averaged over the day, than those in the continuous infusion group. Though the same criteria for control of anticoagulation were used in both groups at the sampling time, it would be interesting to know the anticoagulation profile over 24 hours in the group receiving twice daily subcutaneous injections.

The prospect of using subcutaneous heparin to anticoagulate patients with deep venous thrombosis is very attractive, and the reported results are encouraging. Phlebography has provided the scientific criterion of therapeutic advantage, while patient acceptability and convenience of management offer undoubted practical advantages. If, however, 14 days of subcutaneous heparin anticoagulation is the ideal it may be difficult to achieve in practice in many hospitals. Further studies will be needed to establish the relative importance of the type of heparin, the duration of treatment, the total dose, and the degree of mobility of the patient, as well as the route of administration.

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Children, bikes, and money

SIR,—Cyclists will welcome Dr Tony Smith's interest (16 May, p 1244) in their safety, but Dr Smith recommends that all cyclists should wear helmets, be separated from other traffic, and be given formal training in road safety. These worthy proposals seem to me to overlook the fact that it is the other road users that cause the cyclists' accidents.

Helmets have become more widely worn voluntarily by pedal cyclists recently, but we have no epidemiological evidence of their effectiveness because we do not know the prevalence of exposure to accidents among helmet users and non-users. Because of the confounding effect of risk compensation[1] even showing the benefit of crash helmet legislation for motor cyclists remains problematic.[2] Separation of cyclists from other traffic might reduce accidents, but who should be separated from who? In the study by Mr James Nixon and colleagues (p 1267) only 5% of the accidents injured a second person yet 80% involved a motor vehicle. The cyclists were injured, but surely it is the motor vehicles that should be regulated. I would welcome more road safety training, although its effectiveness is also unproved. Moreover, as one third of the children injured in the Philadelphia study were stunt riding or riding too fast[3] we must recognise that education for cycling could transfer necessary risk learning to other activities resulting in new accidents.

Dr Smith correctly states that all roads should be made as safe as possible. Half of the accidents in Nixon *et al*'s study, however, occurred on straight roads, and lighting conditions were unimportant: the roads themselves are not the problem. They are simply the setting for the everyday drama of travel, of the conflicting interests between car drivers and cyclists. Two proposals not mentioned by Dr Smith would substantially improve the position of cyclists: to reduce the speed of vehicles and to divert car travellers to public transport. This might not actually reduce the number of accidents[4] as we would see an increase in the number of cyclists, as has already happened in cities that have banned cars from their centres. What we must not do is limit further the freedom of cyclists, who do not usually cause accidents to other people, on the specious grounds of preventing accidents.

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Lower oesophageal contractility as an indicator of brain death in paralysed and mechanically ventilated patients with head injury

SIR,—Dr D J Hill (6 June, p 1488) suggests that our letter (16 May, p 1287) about the presence of lower oesophageal contractions in patients in whom brain stem death had been diagnosed supports his view that the clinical criteria of brain stem death are inadequate. It is unfortunate that Dr Hill has been so selective in his interpretation of the relation between spontaneous oesophageal activity and brain stem function. We agree with him that doubt would be cast on the adequacy of the clinical tests of brain stem death if it was established that lower oesophageal contractility depended on brain stem function. There is no evidence at all, however, that this is the case.

The references that Dr Hill cites in support of his contention that oesophageal activity is mediated through the vagal brain stem nuclei merely aired a hypothesis.[1 2] He has ignored the fact that we recorded brain stem auditory evoked responses in our patients. The finding that in three patients these recordings showed no peaks despite the presence of spontaneous oesophageal contractility supports our argument that spontaneous lower oesophageal contractions do not depend on a functioning brain stem but are due primarily to activity in the oesophageal nerve plexus. It is, of course, very probable that the frequency of these contractions is modulated normally by impulses generated in the brain stem.

The purpose of our letter was to contrast our observations with the findings of Drs Sinclair and Suter,[2] whose report might have encouraged others to rely on spontaneous oesophageal contractility as a predictor, or even a confirmatory sign, of brain stem death. We did not intend to enter the debate about the reliability of the clinical criteria of brain stem death. We do not believe, however, that spontaneous lower oesophageal contractility depends on the brain stem for its genesis, or that its presence or absence should be used as evidence to support either side in the brain stem death debate.

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Why do women live longer and is it worth it?

SIR,—Dr Alan J Silman presents some very interesting epidemiological evidence relating to the increased longevity of women compared with men (23 May, p 1311). I am sure, however, that he would be the first to admit that the combination of cigarette smoking, industrial accidents, heavy drinking, and general self neglect still cannot explain the pronounced difference in life expectancy between the sexes. The question of a protective role of female sex hormones, a subject touched on briefly, requires a closer look.

In addition to their receptor mediated effects, both synthetic and naturally occurring oestrogenic compounds probably have substantial radical scavenging ability, by virtue of their phenolic A ring.[1] Oxygen free radicals have been related to the aging process by several investigators.[2 3] Increased lipid peroxidation by free radicals leads to atheromatous plaque formation and consequent atherosclerosis. It is, of course, well known that women are largely protected against this disease (at least until the menopause). The laying down, in various organs including the brain, of age related pigments composed of malonyldialdehyde complexes is also free radical mediated. It therefore seems feasible that the scavenging of free radicals by oestrogens may help to slow aging and degenerative processes in women as compared with men. On the other hand, cigarette smoking, a potent producer of free radicals, has been shown visibly to acccelerate the aging process and is the major cause of arteriosclerotic disease in both sexes.[4] It has also been suggested that carcinogenesis may be stimulated by free radicals,[3] which may explain the increased incidence of many neoplastic diseases (with the exception of hormone dependent cancers) in men. Though the evidence for the radical scavenging activity of the oestrogens is still circumstantial, it goes some way towards explaining many of the differences in aging and other disease processes between the sexes.

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SIR,—Has Dr Alan J Silman (23 May, p 1311) thought that the reason why many elderly women lead socially isolated and financially impoverished lives is because their husbands, by smoking, drinking, and carelessness in avoiding accidents, have roistered themselves into an early death?

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