study represents a step forward in the investigation of the possible carcinogenic effects of 50 Hz electromagnetic fields.

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Authors' reply (Finland)

EDITOR,—We are well aware that magnetic fields from sources other than power lines will, if carcinogenic, mask and decrease the observed association. Our exposure assessment was based on the assumption that magnetic fields of high voltage power lines usually increase the overall exposures of children living in the vicinity of the lines compared with other children. Calculation of magnetic fields generated by power lines provided no more than a tool for classifying study subjects according to their overall exposure levels. The subcohorts of children with maximal annual average exposure of $\geq 0{\cdot}20$ μT or cumulative exposure of $\geq 0{\cdot}40$ μT years were considered to be exposed in our study; the rest were investigated solely to compare observed cancer rates with an external reference. The magnetic field level is usually less than $0{\cdot}1$ μT in Finnish homes without external sources,[1] and it is thus likely to mask only part of the effect of power lines in the exposed group.

The possible carcinogenic property of magnetic fields, if not the average as such, may well correlate with the average. The Finnish 110-400 kV grid is electrically well balanced by using carefully designed transpositions of phase conductors. The zero sequence current from the distribution grid cannot be transferred to a 110-400 kV grid because 110/20 kV transformers are of phasor group star delta. According to our measurements, zero sequence currents in 110 kV lines are typically a few amperes, seldom more than 10. However, zero sequence currents may have an effect on magnetic fields of power lines, especially in the case of the distribution grid but slightly also in the transmission grid for residences situated at some distance from the line. As to the resonance phenomena involving the alternating power frequency field and the earth's local geomagnetic field, Mr Philips did not further specify how these resonance phenomena would affect risk estimates in epidemiological studies.

The pragmatic objective of our study was to estimate the excess cancer risk in children living close to overhead power lines of 110, 220, and 400 kV, presuming magnetic fields are the carcinogenic agent, and not to estimate the risk caused by magnetic fields from various sources. We did not exclude the possibility of a real cancer risk of magnetic fields in our paper, although the population attributable excess risk of childhood cancer due to the power lines apparently is small. If the data are analysed using higher cut off points of exposure, risk estimates seem to be higher but they pertain to a smaller fraction of the population. For example, the children and adolescents in our study with cumulative exposure of $\geq 1{\cdot}0$ μT years contributed a total of 28400 person years. The corresponding standardised incidence ratios were $2{\cdot}3$ for total cancer (nine cases; 95% confidence interval $1{\cdot}0$ to $4{\cdot}3$), $2{\cdot}8$ for nervous system tumours (three cases; $0{\cdot}6$ to $8{\cdot}1$), $3{\cdot}5$ for leukaemia (three cases; $0{\cdot}7$ to 10), and $1{\cdot}5$ for other tumours (three cases; $0{\cdot}3$ to $4{\cdot}3$)—that is, the risk estimates for the small group of children with higher cumulative exposure seem to be relatively high, with wide confidence intervals. An analysis combining the results of the three recently published Nordic studies taken together supported the link between magnetic fields and childhood leukaemia.[2] The emphasis should no longer be on blaming exposure misclassification for having decreased the observed relative risks but on further serious attempts to comprehend the effects of magnetic fields on human health.

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Enforced hysterectomies

EDITOR,—Guru Nandan's report of 11 women in India who were forced to have a hysterectomy highlights the appalling ethical standards of some Third World countries.[1] It is encouraging that some Indian doctors have protested against the decision to carry out these operations.

Over the past year I have received letters from 11 women who have been subjected to a hysterectomy against their will in British hospitals. They have written to me after learning that I underwent a similar experience. I am a medical writer and editor and therefore better placed than most women when it comes to standing up for myself. Two friends, one a consultant physician and the other a consultant gynaecologist, separately advised me to complain to the police. The Crown Prosecution Service decided, however, that there was insufficient evidence to justify charges of assault. Since then at least three other women have complained to the police. In one case, the woman's solicitor has commenced a private prosecution.

The women who have contacted me had consented to a lesser procedure, usually laparoscopy, and awoke to learn that the surgeon had removed their wombs without consent. Most have been told by the surgeon that he or she "found something" and performed what may have been a lifesaving procedure. On consulting a solicitor they discovered that the "something" was, at worst, fibroids or adenomyosis. One woman, a widow of 75, had been admitted to have a pessary checked under anaesthesia; the hospital has admitted that it knew that she did not want a hysterectomy but thought that she should have it.

Several women came forward after reading about similar cases in newspapers, particularly the *Independent* (eight women); only one came forward after an article in the *Sun*. I believe that there are many more cases than 12, but that most women, particularly those with little higher education, have believed what their surgeon has told them. Most of the women are suing, which takes several years and costs thousands of pounds; most will probably abandon legal action when they and their money are exhausted. So the problem goes on. I have also heard from women who have needed an emergency hysterectomy after other procedures have gone wrong: in at least one case the procedure—dilatation and curettage—was an odd thing to do for premenstrual tension. Other women have consented to hysterectomy and subsequently learnt that it was unnecessary. Two of these had the irritable bowel syndrome, which was eventually cured by advice about diet and managing stress.

All of the women have been distraught and are emotionally scarred often years later; some are unable to work; most are terrified of doctors and hospitals. All of them are concerned that other women should not suffer the same treatment.

I have not heard any protest from the medical profession in Britain such has occurred in India. I hope that any doctors who think that women deserve better than this will stand up and be counted. Meanwhile, we should not assume that what happens to mentally handicapped women in India is any worse than what happens in our own back yard.

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1 Nandan G. Women in India forced to have hysterectomies. *BMJ* 1994;308:558. (26 February.)

New building at Guy's Hospital

EDITOR,—In his article about the charitable funding for Philip Harris House at Guy's Hospital Owen Dyer quotes Rachel Daniels, spokeswoman for the Guy's and St Thomas' Trust.[1] She apparently said: "There never was a perfect agreed blueprint for the building." This is nonsense. Parts of the building are already finished, and it was due to be occupied later this year. The structure of the building (which has been planned since 1979), its services, and its contents were agreed down to the last detail.

As Dyer indicates in his article, Philip Harris House was to be the third and final phase of the modernisation of Guy's Hospital. It was completely integrated with the tower and other parts of the hospital and should have contained long awaited modern inpatient and outpatient facilities for several medical specialties. It is sophistry for the health ministers to suggest, as they do, that the building will largely be used for its original purpose. Proposals for its new function have yet to be completed, but this is certain to be very different from the original plan.

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1 Dyer O. Charities demand money back in Guy's changeover. *BMJ* 1994;308:935. (9 April.)

Nocturnal blood pressure

Measurement is subject to bias

EDITOR,—Though I agree with Martin Middeke and Joachim Schrader that many patients with secondary hypertension seem to have a reduced fall in nocturnal blood pressure,[1] assessment of the nocturnal dip is susceptible to considerable measurement bias. Variation in the definition of day and night can have an important influence on the calculated nocturnal fall,[2] and I would argue

that assessment of pressures while subjects are awake and asleep is both more reproducible and of greater physiological relevance.[2] I wonder why, in a prospective study, the definition of night was 2200-0600 but the frequency with which blood pressure was recorded was reduced, presumably to minimise disturbance of sleep, from 2400.

Previous studies showing a blunted diurnal rhythm in patients with secondary hypertension have been performed predominantly in hospital inpatients, while most studies of subjects with essential hypertension have been in outpatients. As admission to hospital may itself influence the blood pressure profile,[3] probably because of a combination of reduced activity lowering the value during the day and sleeping in an unfamiliar environment raising the value at night, it is important to know the circumstances in which the ambulatory monitoring was performed on the subjects with secondary hypertension in this study. If it was performed during inpatient investigation the results must be treated with caution. What criteria were used to determine that patients had a "normal sleep-activity rhythm?"

The nocturnal dip in blood pressure is normally distributed in a hypertensive population,[4] and many patients with essential hypertension will therefore have a blunted profile. As essential hypertension is some 20 times more common than secondary hypertension, most patients found to have a small nocturnal dip in blood pressure will turn out not to have secondary hypertension.[5] Furthermore, my experience and that of others has been that blunting of the diurnal profile is not consistent in either renovascular or endocrine hypertension.[5] I therefore doubt whether ambulatory monitoring of blood pressure has either the sensitivity or the specificity to "aid the differential diagnosis of secondary hypertension."

Observation of a reduced or absent nocturnal fall in blood pressure is of considerable scientific interest, but its importance in a clinical environment remains undetermined.

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Authors' reply

EDITOR,—We agree with Michael J Stewart that the definition of day and night will have a considerable influence on the calculated variation in blood pressure between day and night. The effect of the frequency of measurement, on the other hand, is much less, as long as the measurements are not taken too infrequently (>40 minutes) at night. Our experience has shown that the period 2200 to 0600 is compatible with the actual sleep phase of most patients. Our patients are instructed to rest at this time if possible. At the time of our study we could not take the actual sleep phase into account because of technical circumstances.

As Stewart correctly assumes, we routinely take measurements between 2400 and 0600 at longer time intervals, irrespective of the definition of day and night, so that we do not excessively disturb patients' sleep. Measurements in our studies were made exclusively on outpatients, so that the possible influence of admission to hospital on the day-night rhythm can be excluded. The sleep-wake rhythm was determined from the patients' records.

The last point mentioned by Stewart is the most important and concerns the clinical importance of the day-night rhythm in the differential diagnosis of hypertension. Our experience shows that 78% of patients with secondary hypertension have a reduced nocturnal fall in blood pressure (<10% diastolic) compared with only 5% of patients with primary hypertension. This indicates a sensitivity and specificity that compare favourably with those in many other clinical tests.

In our view it is important not merely to refer to "dippers" and "non-dippers" but to differentiate to a far greater degree, since prognostic[1,2] and therapeutic consequences ensue[3,4] along with the diagnostic aspects.

Finally, the decision whether patients have primary or secondary hypertension is a matter of definition, which conceivably should also take the day-night rhythm into consideration in future. From a pathophysiological point of view we found it especially remarkable that patients with primary hyperparathyroidism and hypertension showed a normal circadian rhythm in contrast to patients with hyperthyroidism and hypertension. Patients with hyperthyroidism and normal blood pressure, on the other hand, had a reduced fall in their nocturnal heart rate: 9%, compared with 14% in patients with hyperthyroidism and hypertension.

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Effectiveness of cycle helmets

Remains debatable

EDITOR,—In their analytical epidemiological study of the effectiveness of cycle helmets, Steven Thomas and colleagues' discussion of biases was incomplete and possibly misleading.[1] In the second paragraph of the discussion they describe some sources of bias that would cause their data to underestimate the strength of the association between wearing a helmet and reduced risk of injury to the upper head. They then state: "Similarly, reduction in the estimated risk of upper head injury would have been greater if cyclists wearing helmets who did not have head injuries were more likely than non-helmet wearers to attend hospital."

This seems to imply that this source of bias would also cause their estimate to be an underestimate of the reduction in risk of upper head injury with helmet wearing. But it would cause the opposite—the authors' estimated reduction in risk would overstate any true reduction. If helmet wearers were more likely to attend hospital then the authors' estimate of the prevalence of helmet wearing in patients without head injuries would be too high, causing them to overestimate the odds ratio of upper head injury for people not wearing helmets. Restricting the study to children with injuries severe enough to require admission to hospital, when it could be assumed that all such children would attend hospital, would better control for this potentially important source of bias.

Secondly, the authors fail to mention one of the main disadvantages of using diseased controls in a case-control study. In this case the authors may have shown either that helmets reduce upper head injuries or that they are associated with injuries to other parts of the body. Possibly helmets do not change injuries overall but decrease severe injuries, in which case their universal use may be justified. But only a population based study can show whether helmets increase or decrease injuries overall or to specific parts of the body. (Incidentally, a cohort study in a population with high compliance with compulsory helmet wearing would not be an ideal study, as the small number of people not wearing helmets would probably differ in many ways from people wearing helmets, in particular in risk taking behaviour.)

This study is an important contribution on cycling safety, but, if science is to make the best contribution to public policy making, researchers and readers of research must be aware of the weaknesses as well as the strengths of the scientific evidence.

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1 Thomas S, Acton C, Nixon J, Battistutta D, Pitt WR, Clark R. Effectiveness of bicycle helmets in preventing head injury in children: case-control study. *BMJ* 1994;308:173-6. (15 January.)

Author's reply

EDITOR,—I and my colleagues generally agree with Patricia Priest's criticism and comments. Our discussion particularly aimed to emphasise the potential inadequacies of the case-control design, and we were careful to suggest that results from our study cannot stand alone and that a cause and effect relation might be suggested only by large population based cohort studies. By high compliance with helmet wearing we meant levels similar to those quoted in our results, with about half the population wearing helmets, to ensure adequate person time of experience in which to consider events of head injury. We agree that in the case of extremely high compliance such a study would not be ideal for the reason cited.

Priest rightly corrects our interpretation of the direction of the change in risk. Given the magnitude of the protective effect that we estimated, however, such a bias alone is unlikely to be large enough to nullify the noted association.

There was much internal debate about our choice of a suitable control group for this study. Certainly, a further restriction to include only non-head injuries to cyclists of a severity requiring admission to hospital would control for any bias in risk taking behaviour, although perhaps at the expense of statistical power, generalisability of the study, and other logistic considerations. There was little evidence in our study that helmets cause injuries to other parts of the body. Though we did not specifically address questions about vision and headturning, we asked for details of the cause of the accident and injury, which elicited no reports of poorly designed or poorly fitting helmets as a cause. Consequently, as Priest concurs, even though we chose disease controls, it is most plausible that the noted effect is a protective effect of helmet wearing against upper head injury. Thompson *et al* used similar controls and showed an even stronger effect.[1]

We do not agree that the trends of injury suggest that helmets may increase the rate of bicycle related injury overall, but we allow that no effect