figures are in fact quoted in that report, but it can be inferred that they have been obtained from *Statistics of Smoking in the United Kingdom* published by the Tobacco Research Council. The latest edition for 1972 contains the figures quoted in your article but these relate to the proportion in 1971 of smokers of any kind (that is, including smokers of cigars or a pipe) and not just to cigarette smokers. In 1971 the proportion of men in the United Kingdom who were smokers of manufactured cigarettes was 51% and the proportion of women was 42%.

When dealing with statistics of the prevalence of smoking it is important to distinguish between all smokers, all smokers of cigarettes (including hand-rolled cigarettes), and smokers of manufactured cigarettes. The difference between the percentage of manufactured cigarette smokers and that of all smokers can be as large as 15% for men, though the difference is negligible for women.

If the three quantities become confused (as appears to happen in your article), then a very misleading picture can be given.

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Vitamin C and the common cold

SIR,—Some of the trial evidence to which you refer[1] in your leading article (13 March, p 606) was subsequently discussed.[2 3] Repetition of the trial[4] demonstrated that vitamin C administration produces a significant beneficial effect on cold symptoms induced by intranasal instillation of the common cold virus.[5] You discuss our trial in schoolchildren[6] and comment on the criticisms of Dykes and Meier.[7] They drew our attention to an error of transcription in table I of our paper.[6] The mean ± standard error of the symptom intensity of catarrhal colds in boys who were receiving placebo tablets was 0·31 ± 0·09, not 0·18 ± 0·05. This provides a t value of $-0·96$ as stated in our table. The description of our trial was also criticised in that we did not comment on the make-up of the placebo or provide any evidence on the maintenance of double-blind conditions. We described these details fully in another paper to which reference was given.[8] White tablets, identical in colour, taste, and consistency, were provided as active and dummy tablets to the students. During the trial it was never suggested by the students to the school matrons that the tablets differed in any way from each other. The school matrons who administered the tablets were unaware that they differed in formulation. The trial was therefore fully double-blind with respect to both tablet constitution and administration. Results obtained from prophylactic trials on the effect of vitamin C on common cold symptoms indicate that daily administration of vitamin C within the dose range 200-2000 mg exerts a significant effect against the common cold. The evidence from these and other recent trials which you omit to mention was reviewed last year.[5]

When vitamin C is used for cold prophylaxis the raised ascorbic acid tissue concentrations during periods of normal health may induce greater tissue utilisation of the vitamin, thus increasing tissue metabolic activity and defensive efficiency. If viral penetration occurs despite high tissue ascorbic acid levels, no excess of ascorbic acid above the elevated tissue requirements is then available. This may be why some individuals on prophylactic ascorbic acid develop severe colds, and continue to have cold symptoms, when the defences have been breached.[9] In these circumstances, as you imply, further supplementation of the dose may be capable of controlling symptoms by further enhancing body defence. It probably exerts its prophylactic and therapeutic effects by enhancing defence mechanisms against viral attack and spread through the itssues and by promoting subsequent tissue healing.[10] In the absence of ascorbic acid, rhinovirus acts as a poor inducer of interferon, but it is susceptible to ascorbic acid.[11] Ascorbic acid may exert its benefit by promoting cyclic AMP production[12] and by enhancing interferon production[13] and reducing prostaglandin synthesis[14 15] during colds.

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Ischaemic heart disease in epileptics

SIR,—We note with interest three reports in the *BMJ* concerning ischaemic heart disease (IHD) in patients with epilepsy. Professor V Lindén (12 April 1975, p 87) suggested that IHD was rare in patients with chronic epilepsy. Dr S Livingston (6 March 1976, p 586) was also of the opinion that patients with epilepsy have an exceedingly low incidence of IHD. In both these letters the suggestion is made that long-term anticonvulsant therapy reduces the risk of IHD. On the other hand, Dr R Pelkonen (11 October 1975, p 85) reported that "an increase in serum cholesterol may be regarded as an untoward effect of long-term phenytoin treatment because it increases the risk of coronary heart disease."

We recently completed a study of IHD in a cohort of 310 patients with epilepsy.[1] This cohort is composed of residents of Rochester, Minnesota, with epilepsy newly diagnosed during the period 1935-67. Since the age- and sex-specific incidence for the mortality of IHD are known in this population it was possible to determine the numbers of IHD deaths and cases expected to occur in this cohort.

The mortality from IHD was not significantly different, as 20 deaths from IHD were observed while 16 were expected. With respect to the incidence of IHD, the expected number of cases was 15·7, while 25 such cases were observed. This difference was of borderline statistical significance ($P = 0·05$), and might be attributable to factors not directly related to epilepsy itself.

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Temperature rhythm in manic-depressive illness

SIR,—Drs Georgia Nikitopoulou and J L Crammer (29 May, p 1311) adduce some interesting evidence in favour of time discordance in depression. They postulate that the disruption of normal temperature swing found in their subjects might be due to desynchronisation relative to other subsidiary circadian rhythms.

However, some advantage might be seen in a model wherein the time conflict is shifted centrally. This would have the depressed subject revert to a "basic" endogenous diurnal rhythm, specific for each individual.[1] Such a time scale could be controlled by an archaic central biological clock out of step with the 24-hour solar day. An inherent central rhythm of this kind may have complete dominance at nights and only hesitatingly entrain with the 24-hour clock when the subject wakes. The more severe the illness, the less effectively will the time-markers of the advancing solar day pull the subject back into his premorbid circadian rhythm.

Expressed in a homely way, the patient aroused in the morning by the solar alarm clock may be unkindly drawn back towards oblivion by his personal central clock indicating a different hour of the day. Hence derive, perhaps, the well-known subjective and variable morning torpor and also such objective data as, for example, that of temperature disturbance described by the authors.

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Fetomaternal haemorrhage and fetofetal transfusion syndrome

SIR,—In their interesting article on "Small infant thymus in cases of fatal fetomaternal transfusion" (20 March, p 694), Drs J Ellis and H G Kohler compare the findings in their cases of fetomaternal haemorrhage with those in the anaemic (donor) twin in the fetofetal transfusion syndrome.

While agreeing that cellular immunity may well be depressed in the fetofetal transfusion syndrome, as suggested by Naeye's finding of low-weight thymuses in donor fetuses,[1] we feel, however, that the situation regarding humoral immunity is not comparable. As the immunoglobulin IgG is almost entirely of maternal origin the fetal condition in itself, unless associated with abnormal placental pathology, should not influence the amount of IgG present in the fetus.

In a pair of twins of 33 weeks' gestation with dichorial placentation, of whom only one had suffered from a maternofetal haemorrhage,[2] we found that the intrapair discrepancy in cord serum IgG concentration was not greater than