The association is now piloting sex education modules in schools. It will certainly draw strength from Mellanby and colleagues' work, particularly with regard to the use of specific targeted methods and systematic evaluation.

Sex education has a formidable task to achieve. It is not just about sex: it is also about comprehensive education in family life. This includes interpersonal and decision making skills and positive attitudes about self and family, with the hope that the young person will develop a sense of responsibility for his or her actions.

Critics of sex education claim that evaluations of outcome must show change in the social and sexual behaviour of students outside the classroom. This is a tall order: it is like evaluating civics classes according to their ability to make students into better citizens. I am delighted that the work of Mellanby and colleagues proves the critics wrong. Promoters of sex education programmes around the world must persevere in their efforts. More importantly, they must constantly evaluate their programmes and publicise their findings to dispel myths and misconceptions about sex education.

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Migraine runs in families . . .

. . . but is it inherited?

EDITOR,—Michael Bjørn Russell and Jes Olesen report an increased familial risk of migraine.¹ Before migraine is accepted as an inherited disorder, as it is in the heading to the paragraph about the paper on the "This week in *BMJ*" page, however, all studies and possible biases should be considered.

The paper concentrates on the high prevalence of migraine in first degree relatives of probands with migraine. Surprisingly, however, the figures show that the first degree relatives of probands who had never had migraine had a prevalence of migraine with aura of 5.3% and of migraine without aura of 17.2% (table II). This latter figure is higher than that expected in the general population (table IV). If migraine does aggregate in families one would expect the first degree relatives of probands who had never had migraine to have a significantly lower prevalence than the general population.

Telling all probands in writing that "the object was to study the frequency and heredity of migraine" could have biased the familial study, which seems overall to have shown a higher prevalence of migraine than did the population study, which used the same interviewer, methods, and population.² In Russell and Olesen's study the response rates were high, but some bias may have arisen as the proportion of probands who did not allow their family to be interviewed was 3% for migraine without aura, 6% for migraine with aura, but 11% for those who never had migraine; of the probands in the study, those with migraine had an average of 2.8 relatives whereas those who never had migraine had an average of 3.1 relatives (calculated from table II).

I undertook my study on familial prevalence³ long before the diagnostic criteria of the International Headache Society were available.⁴ Russell and Olesen are wrong to imply that my study suffered "from lack of direct interview of relatives." A trained interviewer administered a questionnaire to over 99% of first degree relatives living in a defined area (south Wales).³ Several large twin studies, also completed before the

new diagnostic criteria were available, gave surprisingly little evidence of heredity.⁵ Perhaps the jury assessing the importance of heredity and environment in the prevalence of migraine should still be considering its final verdict.

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Authors' reply

EDITOR,—Since migraine without aura aggregates in families of probands with migraine without aura, it is expected that migraine without aura is less common in families of probands who have never had migraine. We calculated the population relative risk, adjusting for sex and age, since migraine without aura depends on these factors. If the population relative risk significantly exceeds one an increased family aggregation is implied, whereas a result significantly below one implies a decreased family aggregation.

W E Waters states that the risk of migraine without aura among the first degree relatives of probands who had never had migraine was expected to be considerably lower than the figure we found. This is not correct. We calculated that the expected population relative risk was 0.87 (95% confidence interval 0.62 to 1.12) and found that it was 1.11 (0.83 to 1.39). These results are not significantly different. Thus random variation may explain our result. The calculation of the expected population relative risk was based on the increased risk of migraine without aura in first degree relatives of probands with migraine without aura, the overall prevalence of migraine without aura, and a similar number of first degree relatives of probands who had never had migraine to that in our study.

Waters suggests that the disclosure of the objective of the family study could have biased the study, since the overall prevalence of migraine was higher in the family study than in the general population.¹ Firstly, these prevalences are not comparable, since migraine aggregates in some families and the prevalence of migraine in first degree relatives depends on the relative proportion of probands with migraine and probands who have never had migraine. Secondly, responders and non-responders were not significantly different regarding migraine. A minority of probands did not allow their families to be interviewed, but this is not likely to have caused bias in the family study since migraine as assessed by probands' report was not significantly different in these families and those included in the family study. Probands who had never had migraine had a slightly higher average number of first degree relatives than probands with migraine, which is probably due to chance.

Although Waters pioneered epidemiological studies of migraine, the methods section in his study that we cited is not completely clear. It states that "relatives were visited and a short administered questionnaire on headache was completed."² This can easily be taken as indicating that the relatives, rather than the trained interviewer, filled in the questionnaire. Our results indicate the importance of genetic factors in migraine without

aura and migraine with aura. This is supported by previous twin studies.³ Studies of unselected twins interviewed blind by a physician will, however, be important, and the final confirmation will be identification of the gene(s).

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Screening for intracranial aneurysms

Short natural course makes screening impracticable

EDITOR,—Jacoline E C Bromberg and colleagues report an increased risk of subarachnoid haemorrhage in first degree relatives of patients presenting with this condition and suggest that these relatives might be screened for unruptured aneurysms.¹ Though the accuracy of telephone interviews to diagnose aneurysmal subarachnoid haemorrhage may be questioned, the authors' findings are not surprising. Subarachnoid haemorrhage generally occurs sporadically in the population, but most neurosurgeons have encountered families with a history of the condition, and there is a large literature on such cases.² The question of whether to screen all first degree relatives of patients presenting with subarachnoid haemorrhage is more debatable.

Screening requires a knowledge of the natural course of unruptured aneurysms. This is lacking in at least two respects. Firstly, what are the risks of an asymptomatic unruptured aneurysm bleeding? The figure of 1% a year has been quoted but is based on a retrospective analysis of data.³ The likelihood of aneurysms bleeding is currently being investigated by the international study of unruptured intracranial aneurysms. Secondly, over what period does an aneurysm develop? This is complex: the size of an aneurysm measured angiographically varies depending on the intraluminal thrombus it contains, as well as with the absolute volume of the aneurysm sac. Certainly, we have seen an aneurysm increase in apparent size in a worrying and dramatic fashion over two weeks. Screening is pointless if the period over which an aneurysm develops and ruptures is short, and without such knowledge we cannot say what the interval should be between repeat screening tests.

The practicality of screening also depends on the sensitivity, specificity, and risks of the screening investigation. The gold standard remains cerebral angiography, which requires catheterisation of the carotid and vertebral arteries. This may carry a risk of causing a persistent neurological deficit of 0.5%.³ In the series of Bromberg and colleagues, if 1259 first degree relatives were investigated to identify 10 people who would develop subarachnoid haemorrhage, persistent deficits might be caused in 6. To this the morbidity and mortality associated with treating aneurysms (which might never haemorrhage) would have to be added. The alternative screening investigation, magnetic resonance angiography, is non-invasive but may be insensitive in detecting aneurysms less than 5 mm in diameter.⁴

We suggest that the hazards and limitations of current screening techniques, combined with the present ignorance about the natural course of intracranial aneurysms, make screening of first degree relatives of patients with subarachnoid haemorrhage impracticable.

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- 1 Bromberg JEC, Rinkel GJE, Algra A, Greebe P, van Duyn CM, Hasan D, *et al.* Subarachnoid haemorrhage in first and second degree relatives of patients with subarachnoid haemorrhage. *BMJ* 1995;311:288-9. (29 July.)
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Paper is ambiguous about number with proven aneurysm

EDITOR,—Jacoline E C Bromberg and colleagues report that the estimated relative risk of subarachnoid haemorrhage in first degree relatives of patients with subarachnoid haemorrhage compared with the general population was between 2.7 and 6.6.¹ This is similar to the 4.1-fold increased risk that colleagues and I found in first degree relatives of patients with aneurysmal subarachnoid haemorrhage in the population of Rochester, Minnesota, in the United States,² despite the dissimilar methods used in the two studies.

Not all subarachnoid haemorrhages are due to aneurysmal rupture, and we limited our study to patients with a proved ruptured intracranial aneurysm.² Bromberg and colleagues do not state how many of the 163 patients with subarachnoid haemorrhage had an aneurysm. To compare the different studies on the familial aggregation of subarachnoid haemorrhage^{1,4} it would be interesting to know the proportion of patients with a proved intracranial aneurysm and the location of these aneurysms in the study population reported by Bromberg and colleagues.

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Authors' reply

EDITOR,—Jeremy Rowe and colleagues are under the impression that subarachnoid haemorrhage in the relatives of our patients was diagnosed on the basis of a telephone interview alone, but, as we wrote, all diagnoses were verified by a review of medical documents. Furthermore, Rowe and colleagues are not surprised at the increased risk of subarachnoid haemorrhage in first degree relatives since most neurosurgeons have encountered

families with clustering of such haemorrhages. We found, however, that the increased risk in first degree relatives applies to the average patient with subarachnoid haemorrhage and is not confined to a few selected families. We agree, as we suggested in our paper, that one should not rashly screen these relatives but should study the risks and benefits. Indeed, conventional angiography is not the method of choice for screening, although in the study to which Rowe and colleagues refer the neurological complications occurred only in patients investigated for transient ischaemic attack or stroke (n=227) and not in those investigated for unruptured aneurysms (n=62) or subarachnoid haemorrhage (n=137).¹ Whether relatives should be screened with non-invasive methods such as magnetic resonance angiography should be studied prospectively; we are about to embark on such a project.

Schievink *et al* found an increased risk of subarachnoid haemorrhage in first degree relatives of the same order of magnitude as the risk that we found.² The design of their study differed from ours: it was community based and concerned a retrospective series of patients, and a telephone interview was performed with the patient or next of kin to find first degree relatives who might be affected. We approached all relatives personally and found that we would have missed a quarter of affected relatives if we had used only the family history obtained through the patient or next of kin. Therefore, Schievink *et al*'s estimate of the incidence of subarachnoid haemorrhage in first degree relatives may be too low. Despite this, the similarity of the results in the two studies suggests that they can be generalised to first degree relatives of other patients with subarachnoid haemorrhage.

In reply to Wouter I Schievink's question, we can state that 123 of the 163 patients and six of the 10 relatives had a proved intracranial aneurysm. We chose not to exclude patients who did not undergo angiography because they were mainly patients in poor clinical condition. Since familial subarachnoid haemorrhage seems to carry a worse prognosis than sporadic subarachnoid haemorrhage³ this could bias the results. Full details about the patients and the location of aneurysms will be published shortly.⁴

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Career choices for generation X

EDITOR,—Clare Vaughan states that the medical career structure is rigid and outdated and that flexible career paths for general practitioners are needed, especially to allow for part time working.¹ Having worked in general medicine before moving to Orkney in 1988, I expected to find part time work in general practice, perhaps through the retainer scheme, after a career break while my children were young. Now I find myself ineligible for the retainer scheme owing to a recent European

directive that requires full vocational training for participation. I cannot complete vocational training as my previous experience falls outside the time limits required. A sufficient range of accepted specialties is not available for me to undertake training locally if this experience is disregarded.

It seems that flexibility is decreasing, and I find it wasteful to be thus retired while still relatively young. I hope that the desired flexibility will materialise soon.

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- 1 Vaughan C. Career choices for generation X. *BMJ* 1995;311:525-6. (26 August.)

Controversy over new data on oral contraceptives

Risk of delay was small

EDITOR,—The rapidity with which the Committee on Safety of Medicines released its advice about the risk of thromboembolism associated with oral contraceptives has caused much anger, concern, and confusion.¹ Critics of the committee maintain that (a) systems exist for the rapid dissemination of information to medical practitioners, (b) these systems could have disseminated information to medical practitioners within 12-48 hours, and (c) the short delay resulting from the use of these systems would have resulted in less confusion, concern, and anger. The committee seems, however, to have thought that the dangers were so great that no delay was acceptable.

How many women would have been harmed if Professor Michael Rawlins, the chairman of the committee, had delayed his announcement by 48 hours? Professor Rawlins's letter states that the maximum thromboembolic risk from oral contraceptives that do not contain desogestrel and gestodene is 10 cases per 100 000 per year. He estimates that the risk of thromboembolic events associated with desogestrel and gestodene is double this, the implication being that women taking contraceptives that contain desogestrel and gestodene have a maximum extra risk of 10 cases per 100 000 per year. This approximates to 0.5 cases per million per 48 hours.

A spokesman for the Family Planning Association has estimated that one and a half million women in Britain are taking oral contraceptives. We can therefore imagine a worst case scenario and assume that all of these 1.5 million women are taking a high risk pill. Calculations show that if Professor Rawlins had delayed his announcement by 48 hours less than one woman in Britain (0.8 at the most) would have been placed at risk of having an extra thromboembolic event. Had the delay been one week, at the most 2.8 women would have been placed at risk of having an extra thromboembolic event. Has a sledge hammer been used to crack a nut?

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GPs were swamped by calls

EDITOR,—The way in which the Department of Health handled the information from the Committee on Safety of Medicines on the risk of thromboembolism associated with combined oral contraceptives was inept and dangerous.¹ Any