

LETTERS TO THE EDITOR

When does the risk of acute coronary heart disease in ex-smokers fall to that in non-smokers?

SIR,—The paper by Dr Robinson and his colleagues (1989;62:16-9) used such completely inappropriate methods that its conclusions should not be accepted. First, it notes that, among patients with acute myocardial infarction or unstable angina, those who smoked were a lot younger than those who did not (and had slightly lower serum concentrations of cholesterol and blood pressure). Next it notes that if smokers give up smoking, then after several years their other risk factors will, in aggregate, tend to get worse (one important reason for this being, of course, that the ex-smokers will inevitably get progressively older), until the ex-smokers' overall risk score resembles that of non-smokers. Finally—and this is what is completely inappropriate—Robinson *et al* infer from these two observations that it takes several years after stopping smoking for the risk of myocardial infarction among ex-smokers to fall to that among non-smokers.

Not only is the cited evidence irrelevant to this conclusion, but also the conclusion itself is wrong. Several large and epidemiologically appropriate studies of the effects of smoking cessation have already been done, and have indicated substantial benefit within a short time of stopping smoking.

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This letter was shown to the authors, who reply as follows:

SIR,—Dr Peto's difficulties with our interpretation of both our data and previously published research were ones that we discussed in our paper. Our paper rested on this assumption: that people who get heart attacks have, roughly speaking, all reached the same point in the development of their coronary disease. Smokers, because they smoke, have lower levels of other risk factors. If ex-smokers have a rapidly declining risk after stopping we would expect to see only those ex-smokers whose heart attack came shortly after giving up smoking to present with risk factor levels typical of smokers. The other ex-smokers would have risk factor levels similar to those who never smoked. Our paper examined data on patients after a first episode of myocardial infarction or unstable angina to see if this was so. Dr Peto's criticism would be valid if we had examined a group of people in the general population, but what he does not take into account is that we assumed that all the patients in our study had equivalent progression of coronary disease. If one rejects this assumption one must of course follow Dr Peto in rejecting our conclusions, but it is an assumption that we believe to be tenable, if simplistic, over large numbers of patients with coronary disease. We did not note that "if smokers give up smoking, then after several years their other risk factors will tend to get worse". This conclusion could not,

furthermore, be drawn from the data we presented and we believe the reverse to be true.

Nor is the published evidence on smoking cessation as clear cut as Dr Peto suggests. What are "substantial benefits" and what is "within a short time of stopping"? As we point out, some studies showing absence of added risk in ex-smokers were too small to detect the sort of risk we would expect, and although some large epidemiological studies have indeed shown substantial benefit, others, notably the report of Cook and his colleagues from the British Regional Heart Study,¹ have not.

It would be nice to think that a chronic insult to the cardiovascular system such as smoking, which leaves a permanent legacy in other physiological systems, leaves behind no lasting change in the risk of heart attack, but from the present state of the evidence this is unproven. To treat it as proven, furthermore, would lead us to regard smoking in younger people as cardiovascularly safe, because they can give up before they reach the age at which they might have a heart attack. The question of a lasting residual risk from smoking is therefore an important one—too important, we believe, to regard as being answered by published studies.

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1 Cook DG, Shaper AG, Pocock SJ, Kussick SJ. Giving up smoking and the risk of heart attacks: a report from the British Regional Heart Study. *Lancet* 1986;ii:1376-80.

Fatal intrathoracic haemorrhage after cardiopulmonary resuscitation and treatment with streptokinase and heparin

SIR,—Haugeberg *et al* (1989;62:157-8) reported the death of a man who was treated with streptokinase after resuscitation by cardiac massage and electrical defibrillation after presumed early myocardial infarction. Previous cardiac massage is a well-known contraindication to treatment with thrombolysis and so his death from massive intrathoracic haemorrhage is no surprise.

My question has to do with the necropsy findings. We are not told whether there was a fresh thrombotic occlusion or any evidence of an acute event in a coronary artery or its muscular territory. The patient had ST segment elevation on the electrocardiogram but no Q waves. The massive enzyme release was to be expected after massage and defibrillation and massage itself may cause posterobasal infarction through mechanical trauma to the limp empty heart.

Had this patient who died from haemorrhage after resuscitation and streptokinase had a coronary artery occlusion? I was surprised that the necropsy report made no mention of the coronary arteries.

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This letter was shown to Dr Haugeberg, who replies as follows:

SIR,—Examination of the coronary arteries showed that the left anterior descending and

right coronary arteries had several atheromatous plaques but no significant stenoses. The left circumflex artery had a narrow lumen with a stenotic area 3 cm distal to its origin. In this area there was intimal discoloration compatible with the presence of a lysed thrombus. There were several other atheromatous plaques distal to this stenosis but no signs of other thrombi.

The myocardium showed signs of recent transmural infarction affecting the posterior wall of the left ventricle, including the posteromedial papillary muscle.

These findings support the diagnosis of posterior myocardial infarction secondary to a thrombotic occlusion of the left circumflex coronary artery.

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BOOK REVIEW

Heart Disease in Women. Ed Pamela S Douglas. (pp 316; £50) (Vol 19/3 of Cardiovascular Clinics. Ed AN Brest). Philadelphia: FA Davis Company, 1989. ISBN 0-8036-2751-3.

On first thought there does not seem to be a need for a book on heart disease in women, because the effects on the heart and circulation of the most important difference between men and women are well covered by chapters on pregnancy in standard cardiological texts—a good example being that by Perloff in Braunwald's *Heart Disease*. There is also Szekely and Snaith's *Heart Disease and Pregnancy*, a classic monograph based on three decades' of clinical experience.

First thoughts on this new volume are vindicated by the second chapter, on the cardiovascular response to exercise, which concludes after 18 fact-filled pages that the overall response of the cardiovascular system to exercise is similar in men and women. However well written, accounts of conditions that have similar manifestations in either sex are gratuitous in such a book, and could equally well appear under the title "Heart Disease in Men".

In the section on pregnancy and heart disease, Oakley advises against the use of tissue valves in women planning a family, and states that there is no justification for using heparin rather than warfarin during the first part of pregnancy. In a later chapter on therapeutics and management during pregnancy, contrary opinions are offered by Rutherford and Hands. One would have to look elsewhere, perhaps to *Textbook of Medical Treatment* edited by Girdwood and Petrie, for a full and balanced account of reproductive prescribing. There are duplicated accounts of the physiological changes of pregnancy and there are other signs of deficient editorial control, my favourite being "bibasilar rales can occasionally be heard bilaterally".

The section on coronary heart disease in women is excellent, with a breadth of view

that is lacking from the symposium proceedings *Coronary Disease in Young Women* edited by Oliver. Information on epidemiology, risk factors, diagnostic testing, and treatment are brought together in a scholarly review. A whole chapter by Osbakken is devoted to exercise testing in women, which poses particular problems, not entirely because the pretest probability of coronary artery disease is less than that in men. Diagnosis of coronary disease by probability analysis combining clinical findings and several test results is discussed and illustrated with a comprehensive nomogram based on the work of Patterson *et al.*

The key role of oestrogens in the protection of pre-menopausal women from coronary artery atherosclerosis, and a possible inter-

action with social factors, is indicated by an account of studies in macaque monkeys. Low status monkeys had depressed ovarian function and more coronary atherosclerosis than their overbearing sisters. There is a timely account of the related topic of oestrogen replacement therapy and coronary disease.

The final section of this collation is a consideration of selected diseases including systemic and pulmonary hypertension, mitral valve prolapse, rheumatic heart disease, and cardiomyopathy. The prevalence of all these conditions is different in women and men. While it may be helpful to have this set out within one set of covers it does not make for interesting reading.

Nowhere is there a mention of syndrome X.
JOHN RAWLES

NOTICES

British Cardiac Society

The Annual General Meeting will take place at the English Riviera Centre, Torquay on 22 to 25 May 1990. The closing date for receipt of abstracts is 19 January 1990.

Receptors of the brain, lung, and heart

A meeting entitled Receptors of the Brain, Lung and Heart: State of the Art will be held on 24 and 25 May 1991 at the Hotel Okura, Amsterdam. Further information from Cader Research BV, PO Box 85, 4854 ZH Breda/Bavel, The Netherlands.