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Deaths certified as due to coronary artery disease

SIR,—Professor Denis Pereira Gray and his colleagues repeat what we all seem to hold to be self evident—that ischaemic heart disease is the commonest cause of death in British adults.[1] We also believe that it is commoner here than anywhere else. I think that many of the deaths ascribed to ischaemic heart disease in this country are so ascribed without good evidence.

Whenever a patient dies unexpectedly the case is referred to the coroner, under normal procedure, and a coroner's postmortem examination is performed. My partners and I have been struck by the frequency with which the coronary arteries are examined, atheroma is found, and the cause of death is recorded as myocardial ischaemia due to coronary artery disease, although the brain has not been examined. Does this happen in other areas? What if there had been a stroke or a subarachnoid haemorrhage? The presence of atheroma then would not justify the certified cause of death.

Recently my partner was called urgently to a patient who had had a stroke a few months previously; she had also had a below knee amputation for peripheral vascular disease, and this had broken open and had started to bleed. While he was with her she bled to death. After the postmortem examination the cause of death was given as myocardial ischaemia secondary to coronary artery disease. I suppose that the myocardium was ischaemic, but only because she had exsanguinated. How was this a coronary death?

Is it not likely that we certify far too many deaths as having been due to coronary artery disease, and is it not also likely that countries such as the United States, which have improved their position in the league table for this disease, certify deaths more accurately than we do?

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1 Pereira Gray D, Steele R, Sweeney K, Evans P. Asymptomatic hypercholesterolaemia. *BMJ* 1991;**302**:1022. (27 April.)

Penicillin prophylaxis in children with sickle cell disease

SIR,—Dr David Cummins and colleagues report a study of prophylactic treatment with penicillin in children with sickle cell disease.[1] They found that 31 of the 50 children studied were said to be receiving penicillin every day and that the parents of 37 of the children understood that stopping penicillin could have serious consequences. They conclude that counselling of families of children with the disease needs to be improved if the advantages of neonatal screening for the disease are not to be diminished.

Their paper made me look again at the results of a similar study that I did in 1986.[2] This looked at the care received by young children with sickle cell disease at a teaching hospital in London. The carers of 26 children were interviewed, and the findings were similar to those of Dr Cummins and colleagues. Eighteen children were said to be taking penicillin at least once a day, and the carers of 13 children understood that the aim of penicillin was to prevent infection.

There are, however, problems with presenting results in this way. Firstly, it risks blaming patients inappropriately. In my study eight of the 26 children were taking penicillin less than once a day. Investigation showed, however, that penicillin had not been prescribed for five children and that the carers of another had simply misunderstood the doctor's instructions. In six of the eight cases, therefore, failure to take penicillin daily could not be ascribed to poor compliance.

Secondly, looking only at patients attending a clinic ignores those whose follow up is inadequate—an important group for any screening programme. In my study hospital screening records identified 13 children with sickle cell disease born at the hospital during 1984-5, whom I reviewed in mid-1986. Six had never been followed up; two had been followed up but penicillin had not been prescribed; in one case penicillin had been prescribed but the carer was not interviewed; in one case penicillin had been prescribed but had never been given by the carer; and three children were taking penicillin at least once a day. There was thus good evidence that nine of the 13 children were not taking penicillin; failures of management after screening were more important than parental noncompliance as reasons for inadequate protection.

These findings should be interpreted cautiously. The numbers are small and the study examined care given before evidence from randomised controlled trials of the benefits of prophylactic penicillin in young children with the disease[3,4] was circulated widely. Despite these caveats the central lesson—that the organisation and content of follow up need to be planned as carefully as screening itself—should not be lightly dismissed. Otherwise, neonatal screening is likely to fall short of its aim of reducing the morbidity and mortality associated with sickle cell disease.

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Asymptomatic hypercholesterolaemia

SIR,—In their recent letter[1] Dr Denis Pereira Gray and colleagues state: "Peto estimated that a 1% reduction in serum cholesterol will lead to a 3% reduction in coronary heart disease. On this basis the average reduction of 7% that we are currently achieving through general practitioner advice without drugs is likely to lead to a 21% reduction in coronary disease."

Can they really believe this? Would they conclude that a 33% reduction in cholesterol concentration would be followed by a 100% reduction in coronary disease?

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1 Pereira Gray P, Steele R, Sweeney K, Evans P. Asymptomatic hypercholesterolaemia. *BMJ* 1991;**302**:1022. (27 April.)

AUTHORS' REPLY,—Dr Fogarty's neat reductio ad absurdum illustrates the difficulties in summarising complex statistical models in a single sentence.

Of course we agree with him that it is absurd to predict a 100% reduction in coronary disease, but we still understand that Peto's statistical analyses best represent the relation between the reduction in serum cholesterol concentrations and coronary heart disease. This is a summary of research work that has already been undertaken on the ranges of cholesterol concentrations that are found in Britain. The exact quotation from the Standing Medical Advisory Committee (1990) is:

The Working Party accepted a new analysis of existing data showing that the relationship between blood cholesterol levels and coronary heart disease is stronger than generally realised. This analysis was made available by Mr Peto. It has been presented at scientific meetings and so exposed to scrutiny (Peto, 1989). It is generally accepted that, in middle age, over the range of blood cholesterol concentrations observed in Britain, a 10 per cent reduction in blood cholesterol level is associated with a 20 per cent reduction in coronary heart disease, i.e. a "rule of two" applies. The new analysis suggests the rule of two is an underestimate which fails to allow for the effects of "regression-dilution" bias. It suggests that the true reduction is in fact about 30 per cent, therefore giving a "rule of three". Expressed in more formal terms the cholesterol "elasticity", the percentage change in coronary heart disease events following a one per cent change in blood cholesterol levels, is about three. Elasticity is a proportional rate of change, a unit-free measure of responsiveness.[1]

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1 Standing Medical Advisory Committee. *Blood cholesterol screening: the cost effectiveness of opportunistic cholesterol testing*. London: SMAC, 1990. (Para 2.2.)

Epilepsy and disappearing lesions: adopting a wait and see policy

SIR,—Drs A Kennedy and F Schon recently recommended anticonvulsant treatment alone (that is, adopting a wait and see policy) in the management of epileptic patients in whom computed tomography shows a solitary space occupying intracranial lesions.[1] This policy may be acceptable in the United Kingdom but it is not necessarily suitable in places where computed tomography is not readily available and is expensive.

A 5 year old girl presented to our hospital with a right sided tonic-clonic seizure lasting about 15 minutes. Physical examination was entirely normal. Her full blood count, erythrocyte sedimentation rate, and a chest *x* ray film were all within normal limits. Cerebrospinal fluid contained 6×10^6 red cells/l, no white cells, and protein 0·14 g/l. Computed tomography showed a ring enhancing lesion in the left parietal lobe. Anticonvulsant and antituberculous treatment was started. After four months she had had no further seizures, her weight had increased from 13·2 kg to 15·4 kg, and repeat computed tomography showed near resolution of the lesion.

There is only one computed tomography scanner in Nepal. Few patients can afford the cost of travel to Katmandu and of scanning; fewer still can afford repeat tests. It would seem unjustified in Nepal, where tuberculosis is highly endemic and scanning expensive, to adopt a wait and see policy. Our patient was an exception in that her parents could afford multiple investigations. We think that, in our situation, if computed tomography shows a solitary lesion antituberculous treatment should be started.

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1 Kennedy A, Schon F. Epilepsy: disappearing lesions appearing in the United Kingdom. *BMJ* 1991;**302**:933-5. (20 April.)