Deaths caused 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 and most accepted adult killer. 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 listed 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 likely that countries such as the United States, which have improved their position in the league tables of death from heart disease, certify deaths more accurately than we do?

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Asymptomatic hypercholesterolaemia

Sir,—In their recent letter Dr Denis Pereira Gray and colleagues suggest 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.1

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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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.2 They found that 31 of the 50 children studied who received penicillin every day for 10 years had no attacks of pain. They concluded that prophylaxis is worthwhile and that children with sickle cell disease should continue to receive prophylaxis even if they remain pain-free.3

We have looked at the 13 children under the age of 13 who had penicillin treatment. There were no attacks of pain in any. The parents of each child 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 12 children understood that the aim of penicillin treatment was to prevent infection. There are, however, problems with presenting the results this way. Firstly, it risks blaming patients inappropriately. In my study of the 26 children who 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 three had not followed 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 the fact that the list is adequate for a—important group for any screening programme. In my study hospital screening records identified 13 children with sickle cell disease born at the hospital during 1989, who I reviewed in mid-1986. Six had been reviewed but one had 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 were thus recorded for medical reasons or carers' reasons 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 benefit of penicillin treatment in young children with the disease1 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 fail short of its aim of reducing the morbidity and mortality associated with sickle cell disease.4

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Epilepsy and disappearing lesions: adopting a wait and see policy

Sir,—Dr S 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 lesion.1 This policy may be acceptable in the United Kingdom but it is not necessarily suitable in non-UK 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 x 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 15.0 kg and repeated 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 Kathmandu and of scanning; fewer still can afford repeat tests. We would therefore suggest that, where tuberculosis is endemic, tomography is not invariably used and is expensive.

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