Tuberculous pericarditis

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We question whether children born to diabetic families should ever consume cows' milk. Breast milk and goats' milk are readily available in Britain, and their use should be encouraged for infants, children, and adolescents at risk of developing diabetes mellitus.

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Genetic prediction of adult onset disease

EDITOR,—Genes for an increasing number of adult onset disorders are being defined, and in this era of budgetary restriction and planning by contract we wonder how long doctors can afford to ignore lessons learnt from predictive testing for Huntington's disease. After the localisation of the locus for Huntington's disease in 1983[1] caution was initially exhibited by transatlantic colleagues, who wanted patients and their families legally to exonerate them from the consequences of giving bad news and from the predictable errors inherent in the use of linked genetic markers. In the United States and Britain extensive counselling programmes were developed.[2] People could decide to be tested only after a period of reflection and after receiving accurate information about the limits of the test, the implications for other family members, and the problems of living with good and bad news.

The results of several hundred tests have now been given out in Britain,[2,3] and the counselling programmes are accepted as the minimum for good practice in participating clinical genetics centres.[4] This is in contrast to the situation in other centres in Europe and the United States, where appreciable problems have resulted when inadequate support was provided.

Counselling programmes may be expensive in clinical time, but follow up of the people who have had predictive testing for Huntington's disease in Britain has shown that over half are free of risk and its associated morbidity, while those who receive high risk results cope well with no appreciable changes to their lifestyle.[5] These programmes save the NHS money as well as providing a good service for patients. People at high risk can be followed up so that their problems are dealt with promptly and they may benefit from current research into the disease. Patients find this reassuring, and some visit their general practitioner less frequently than before testing (personal communications from general practitioners).

To give predictive information about other incurable disorders of adult onset to asymptomatic people without such support may well be considered to be negligent. Genetics centres are receiving requests for predictive testing for motor neurone disease, Alzheimer's disease, and some cancers. We believe that clinicians must become aware urgently of the impact of the information that can be produced for these people and that these people should receive the benefit of the experience of the programmes concerned with Huntington's disease.

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Clusters of anophthalmia

EDITOR,—Eduardo E Castilla states that the reported prevalence of anophthalmia and microphthalmia at birth in England and Wales (0·22/10 000) is less than a quarter of the figure for the other populations for which the International Clearinghouse for Birth Defects Monitoring Systems receives data.[1] He also states that anophthalmia is the diagnosis in more than half the cases in England and Wales but in one quarter of those reported elsewhere.[1]

The data for England and Wales come from the nationwide system centred on the Office of Population Censuses and Surveys, which covers only malformations notified soon after birth. In the experience of local English registries, which ascertain malformations from multiple sources, the prevalence of anophthalmia and microphthalmia at birth is much higher (1·3/10 000 in both Birmingham and Liverpool) and at least two thirds of affected subjects have only microphthalmia.[2,3] These findings strongly suggest that the differences reported by Castilla are due (as he hinted that they might be) to incomplete ascertainment, especially of less severe cases, by the nationwide system for England and Wales. This and earlier evidence of the limitations of the nationwide system[4] shows the need for caution when data from this source are used to explore possible clusters of malformations.

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Tuberculous pericarditis

EDITOR,—The patient reported on by C P Clifford and G J Davies, who had tuberculous pericarditis that rapidly progressed to constriction, was Sri Lankan.[1] Tuberculosis is the commonest cause of pericardial effusions in Sri Lanka. We recently prevented constrictive pericarditis in a boy in our unit by using steroids.

A 13 year old boy was admitted with fever and cough of two weeks' duration. He was febrile with a pulse of 96 beats/min and a jugular venous pressure of 5 cm water. He had a remarkable pericardial rub in the left sternal edge. A chest *x* ray film showed severe cardiomegaly, and echocardiography confirmed a large periocardial effusion. A pericardial aspirate contained many red cells and lymphocytes but was repeatedly negative for acid fast bacilli. A Mantoux test yielded a negative result. He was given a course of antituberculosis treatment, and the fever settled completely in five days. Steroid treatment (prednisolone 10 mg three times a day) was started one week after the start of antituberculosis treatment. An echocardiogram at one month showed a mild pericardial effusion with good contractility of ventricles and a normal ejection fraction.

A controlled clinical trial has shown that adding steroids increases the rate of recovery from tuberculous pericarditis compared with that with standard antituberculosis treatment alone.[2] It also reduced the risk of death due to pericarditis and the need for pericardectomy. Furthermore, the highly potent antituberculosis regimen of streptomycin, rifampicin, isoniazid, and pyrazamide daily for 14 weeks followed by rifampicin and isoniazid for six months is believed to be associated with a lower death rate than the less potent regimen of rifampicin, isoniazid, ethambutol, and pyrazinamide for two months followed by rifampicin and isoniazid for four months.[3,4]

It is our practice to start steroids for all patients with tuberculous pericarditis only after the fever has responded to antituberculosis drugs. Thus antituberculosis drugs can be a diagnostic aid in pericardial effusions when the result of a Mantoux test is negative and acid fast bacilli cannot be isolated from pericardial fluid or sputum.

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Imaging methods used in acute aortic dissection

EDITOR,—Our experience as radiologists in a large cardiothoracic centre does not lead us to support the imaging policy put forward by C M Francis and colleagues.[1] Because aortic dissection requires rapid and accurate diagnosis since early surgery may be lifesaving[2] we disagree with the suggestion that transoesophageal echocardiography is the best imaging method. The decision to intervene surgically and the type of operation depend on whether any part of the ascending aorta or vessels of the head and neck are affected. Francis and colleagues themselves state that transoesophageal echocardiography has a restricted view at this site, and it may result in an inaccurate diagnosis. This is supported by results of a study by Nienaber *et al*, which found a specificity of only 68% for transoesophageal echocardiography largely because of false positive findings in the ascending aorta.[3] In addition, none of the useful information on aortic branch vessels, which are easily seen on angiography, can be obtained with transoesophageal echocardiography. We have also found