

bances have one common clinical manifestation, namely, premature coronary artery disease. Patients with Down's syndrome have an additional copy of the gene that codes for cystathionine synthase, thus giving them double the amount of this enzyme.³ The result is that they have a much lower than normal homocysteine concentration. Patients with Down's syndrome have been noted as being remarkably free from coronary artery disease.

It has been over two decades since the publication of the original animal work of McCully and Wilson⁵ who proposed the homocysteine theory of arteriosclerosis. After being ignored for many years, homocysteine has finally re-emerged as a risk factor for the development of human atherosclerosis. Although it is "the new player in the field of coronary risk",² homocysteine will become an increasingly important player that can no longer be ignored in the modern-day management of patients with coronary artery disease.

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

SIR,—Professor Cheng indicates that: (1) patients with genetic defects (cystathionine β synthase (CBS) and 5,10-methylenetetrahydrofolate reductase) have high plasma concentrations of homocysteine and premature coronary artery disease (CAD), as confirmed again by Lolin's paper; and (2) patients with Down's syndrome have a high CBS activity (the CBS gene is located on chromosome 21) and subsequently low concentrations of homocysteine resulting in a low incidence of CAD.

The high CBS activity and the low plasma concentration of homocysteine in Down's syndrome patients have been known for a long time¹⁻³ and, in 1977, Murdoch described Down's syndrome as a human model free from atheroma.⁴ Thus, the remarks of Professor Cheng are correct, although not new. The remaining question is the possible causal relationship between the genetic abnormality and the weak incidence of CAD in Down's syndrome patients. The latter is still controversial because of the reduced life expectancy and other interfering factors. Finally, authors have also reported "normal" concentrations of homocysteine in Down's syndrome patients.⁵ I think that we need more data to relate the gene abnormality to a low incidence of CAD in Down's syndrome patients.

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Hypereosinophilic syndrome: endomyocardial fibrosis

SIR,—We were interested to read Pitt and colleagues' description of their patient with endomyocardial fibrosis.¹ At the end of their discussion they mention that future management of their patient will involve assessment for cardiac transplantation. However, they do not mention endocardectomy, which we regard as the operation of choice in suitably symptomatic patients. The operation consists of endocardial decortication, essentially a coring out of the fibrous tissue, with atrioventricular valve replacement or repair. We recently had a 40 year old female Lebanese patient with endomyocardial fibrosis affecting both ventricles who underwent biventricular endocardectomy with tricuspid valve annuloplasty. Although she had a slow recovery requiring inotropic support for five days, and was eventually discharged on the 20th postoperative day, symptomatically she was much improved.

Endocardial decortication for this disease was introduced by Dubost *et al* in 1973² and there are now several operative series in the literature.³ Most series report a hospital mortality rate of 20%, most often secondary to low cardiac output. For those who survive the operation 75% are alive after five years, and in the majority of cases there is good clinical improvement.³ Untreated, patients with endomyocardial fibrosis usually die within three years. Most patients in the reported series were in NYHA class III or IV and it is likely that operative mortality will be less if patients undergo surgery at an earlier stage.

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Endomyocardial fibrosis in Egypt: an illustrated review

SIR,—We read with great interest the paper on endomyocardial fibrosis (EMF) by

Rashwan *et al.*¹ Their association of cases with hepatosplenic schistosomiasis, which was felt to be involved in the production of EMF rather than just the coincidence of two diseases, has been reported previously. We published a case report in 1973 making the suggestion of a pathogenetic relation of schistosomiasis to EMF.² Another case report illustrating the association was published more recently.³

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Deterioration in renal function with enalapril but not losartan in a patient with renal artery stenosis in a solitary kidney

SIR,—Angiotensin-converting enzyme (ACE) inhibitors are widely used in the treatment of hypertension and heart failure. However, when given to patients with bilateral renal artery stenosis or unilateral stenosis in a solitary kidney, they can cause renal impairment that is usually reversible on stopping the drug.¹ Recently, an angiotensin II (AII) receptor blocker (losartan) was introduced for the treatment of hypertension. We report a patient with severe hypertension and apparent heart failure who developed renal impairment with an ACE inhibitor but not with the AII receptor antagonist.

A 73 year old man with a previous history of severe hypertension was admitted to hospital with symptoms and signs of biventricular failure. Blood pressure was 150/90 mmHg and apical heart rate was 148 beats/min on treatment with digoxin 125 μ g daily, frusemide 40 mg daily, enalapril 10 mg twice daily, nifedipine LA 30 mg daily, and warfarin. Serum creatinine was 308 μ mol/l. Renal ultrasonography showed a small left kidney (8.9 cm) and a normal size right kidney. Echocardiography revealed concentric left ventricular (LV) hypertrophy, no valve lesions and good LV function.

Coronary angiography revealed an 80% diffuse disease of the left anterior descending artery, and a 50% obtuse marginal stenosis. Renal angiography performed at the same time revealed total occlusion of the left renal artery and a proximal severe stenosis of a single right renal artery (fig). Enalapril was therefore discontinued and five days later serum creatinine fell to 148 μ mol/l.

Subsequently, blood pressure and symptoms of apparent heart failure were difficult to control despite adequate control of the atrial fibrillation. The patient was then started on losartan 50 mg twice daily in addition to two Frumil (co-amilofruse 5/40)