- 4 **Nielen MM**, van Schaardenburg D, Reesink HW, *et al*. Specific autoantibodies precede the symptoms of rheumatoid arthritis: a study of serial measurements in blood donors. *Arthritis Rheum* 2004;**50**:380–6.
- 5 **del Puente A**, Knowler WC, Pettitt DJ, *et al*. The incidence of rheumatoid arthritis is predicted by rheumatoid factor titer in a longitudinal population study. *Arthritis Rheum* 1988;**31**:1239–44.
- 6 **Mikkelsen WM**, Dodge HJ, Duff IF, *et al*. Estimates of the prevalence of rheumatic diseases in the population of Tecumseh, Michigan, 1959–60. *J Chronic Dis* 1967;**20**:351–69.
- 7 **Goodson NJ**, Symmons DP, Scott DG, *et al*. Baseline levels of C-reactive protein and prediction of death from cardiovascular disease in patients with inflammatory polyarthritis: a ten-year followup study of a primary care-based inception cohort. *Arthritis Rheum* 2005;**52**:2293–9.
- 8 **Sayer AA**, Syddall HE, Gilbody HJ, *et al*. Does sarcopenia originate in early life? Findings from the Hertfordshire Cohort Study. *J Gerontol A Biol Sci Med Sci* 2004;**59**:M930–4.
- 9 **Syddall HE**, Aihie Sayer A, Dennison EM, *et al*. Cohort profile: the Hertfordshire Cohort Study. *Int J Epidemiol* 2005;**34**:1234–42.
- 10 **Prineas RJ**, Crow RS, Blackburn H. *The Minnesota code manual of electrocardiographic findings: standards and procedures for measurement and classification*. Boston: John Wright, 1982.
- 11 **Fall CH**, Osmond C, Barker DJ, *et al*. Fetal and infant growth and cardiovascular risk factors in women. *BMJ* 1995;**310**:428–32.
- 12 **Ramsay LE**, Williams B, Johnston GD, *et al*. British Hypertension Society guidelines for hypertension management 1999: summary. *BMJ* 1999;**319**:630–5.
- 13 **Tighe H**, Carson DA. Rheumatoid factor. In: Ruddy S, Harris ED, Sledge CB, eds. *Kelley's textbook of rheumatology*. 6th ed. Philadelphia: W. B. Saunders, 2001:151–60.
- 14 **Symmons D**, Turner G, Webb R, *et al*. The prevalence of rheumatoid arthritis in the United Kingdom: new estimates for a new century. *Rheumatology (Oxford)* 2002;**41**:793–800.
- 15 **Wick G**, Knoflach M, Xu Q. Autoimmune and inflammatory mechanisms in atherosclerosis. *Annu Rev Immunol* 2004;**22**:361–403.
- 16 **Smeeth L**, Thomas SL, Hall AJ, *et al*. Risk of myocardial infarction and stroke after acute infection or vaccination. *N Engl J Med* 2004;**351**:2611–18.
- 17 **Epstein SE**, Zhu J, Burnett MS, *et al*. Infection and atherosclerosis: potential roles of pathogen burden and molecular mimicry. *Arterioscler Thromb Vasc Biol* 2000;**20**:1417–20.
- 18 **Stolt P**, Bengtsson C, Nordmark B, *et al*. Quantification of the influence of cigarette smoking on rheumatoid arthritis: results from a population based case-control study, using incident cases. *Ann Rheum Dis* 2003;**62**:835–41.

IMAGES IN CARDIOLOGY

doi: 10.1136/hrt.2006.101048

Multiple coronary artery fistulae or Thebesian veins?

A previously healthy 55-year-old patient presented with progressive exertional chest pain. Physical examination showed no abnormalities except for a diastolic cardiac murmur. The ECG showed left ventricular hypertrophy. The laboratory findings and the chest x ray were normal.

An exercise treadmill test was positive. The echocardiogram showed multiple

diastolic jets draining into the left ventricle. A coronary artery angiogram disclosed strongly dilated left and right coronary arteries with multiple microfistulae draining into the left ventricle (panels A and B). In our patient besides the diagnosis of microfistulae, a diagnosis of persistent venae cordis minimae (Thebesian veins) was also considered. However, because there were direct con-

nections between the coronary arteries and visible small vessels draining into the left ventricle and because both coronary arteries showed tortuosity and were strongly dilated (flow-mediated dilatation), we believed that this was a case of multiple fistulae rather than persistent Thebesian veins.

Coronary artery fistulae have an incidence of 0.2% in patients undergoing diagnostic cardiac catheterisation. The normal sites of origin are the right coronary artery (55–60%), the left coronary artery (35%), and both coronary arteries (5%). The most common sites of termination are the right ventricle (40%) and right atrium (23%). Sporadically, coronary artery fistulae drain into the left ventricle. The definitive treatment for coronary artery fistulae is surgery or transcatheter embolisation, but given the anatomy, our patient is currently being evaluated for heart transplantation.

S Bulut, H M M Al Hashimi, H R Gehlmann
s.bulut@cardio.umcn.nl