heart which, in turn, are related to the progression of the disease.

This is particularly critical for the correct identification of patients to be submitted to cardiac transplantation. The paper by Kell et al. shed some biological light on this important clinical problem; the presentation is very clear and unbiased, highlighting the limits and the potentialities of interleukin-6 in determining the risk stratification of patients with heart failure in NYHA class III.

R. FERRARI
Cattedra di Cardiologia, Università degli Studi di Ferrara, Ferrara e Centro di Fisiopatologia Cardiovascolare, Fondazione Salvatore Maugeri, Gussago (BS), Italy

References


The mitral kissing vegetation

See page 79, doi:10.1053/euhj.2001.2689 for the article to which this Editorial refers

Spread of infection from the infected aortic to the previously healthy unaffected mitral valve is a well known risk in endocarditis. This ‘seeding’ usually involves the chordae or the ventricular surface of the anterior leaflet which may develop a vegetation or a perforation. This is quite distinct from direct spread of infection from the aortic to the mitral valve.

Antibiotics sterilize the blood (but not the vegetations) very quickly after their instigation, but the mitral regurgitation often develops well after blood cultures have become sterile. Patients with aortic regurgitation but no endocarditis do not develop perforations in the anterior leaflet of the mitral valve or anywhere else although little pockets, the so-called Zahn-Schmincke pockets, often develop in the left ventricular walls in patients with chronic aortic regurgitation when it is severe.

Infected vegetations consistently develop at the same sites and the reason for this was elegantly demonstrated by Rodbard[1] who showed that when infected fluid is driven from a high pressure source through a narrow orifice into a low pressure sink a Venturi effect is produced which deposits bacteria in the low pressure sink immediately beyond the orifice. Turbulence and the jet effect cause trauma to the endothelium. A platelet fibrin plug formed at the site may become secondarily infected in the event of bacteraemia. The Venturi effect is attenuated if the orifice is larger as in more severe aortic regurgitation and the jet effect will be lessened. This explains why infective endocarditis rarely occurs in individuals with atrial septal defects, non-restrictive ventricular septal defects or heart failure.

Horstkotte’s group have developed a new theory to explain secondary mitral valve infection in patients...
with primary aortic valve endocarditis and aortic regurgitation\textsuperscript{[2]}. They have shown that secondary infection can be caused by large vegetations which prolapse into the left ventricle and physically hit the anterior mitral leaflet. This is not a common phenomenon and was observed in only 19 out of 192 patients with primary aortic valve endocarditis who were studied by serial transoesophageal echocardiography (TEE). The mean number of TEE examinations per patient was 10.8 and one patient actually tolerated 18. These data did not come easily!

Vegetations were bigger (≥ 6 mm) in patients with mitral kissing vegetations but aortic regurgitation was severe in only nine. Eighteen of the 19 patients underwent surgery, 11 for haemodynamic reasons, five for persisting sepsis and three for emboli.

When recognition of a mitral kissing vegetation was followed by surgery within 4 days all that was needed was careful cleaning and sterilization of the mitral anterior leaflet. No such patient developed either recurrent endocarditis or mitral regurgitation. When patients already had secondary mitral valve involvement the outcome was less favourable. While aortic valve endocarditis is a risk factor for secondary mitral valve infection, the authors confirmed from their 109 patients with mitral valve endocarditis that the reverse is not true (the law according to Rodbard). They concluded that aortic valve endocarditis with large vegetations requires ‘close’ TEE monitoring so that with recognition of mitral kissing vegetations, surgical intervention can be prompt in order to preserve the integrity of the mitral valve.

As our patients may lack the stoicism of Horstkotte’s (and our patience also be less than his) the take home message may be to have a low threshold for aortic valve replacement if TEE shows large vegetations, even if they are not yet prolapsing.

C. OAKLEY
Hammersmith Hospital,
London, U.K.

References
