

## Heartbeat: Highlights from this issue

Catherine M Otto

Cardiologists are seeing increasing numbers of patients with calcific aortic stenosis (AS) as the age distribution of the population shifts toward the elderly in developed countries. Although we now have identified several clinical and genetic factors associated with calcific AS, recognize that about 50% of patients have an underlying bicuspid aortic valve, and have an elementary understanding of the disease process at the tissue level, as yet there is no medical therapy to prevent worsening AS. Instead, the mainstay of clinical management is periodic monitoring for disease progression and symptom onset. However, the optimal monitoring interval is unclear given marked heterogeneity in the rate of anatomic and hemodynamic changes in valve function.

In this issue of *Heart*, Dr Nguyen and colleagues (see page 943) report AS progression in 149 patients enrolled in two prospective studies. The average increase in mean transvalvular gradient was  $+3 \pm 3$  mm Hg/year with a mean increase in CT aortic valve calcification of  $+188 \pm 176$  AU/year. Baseline AS severity was the strongest predictor of disease progression, with more rapid progression in those with more severe disease (figure 1). These data support current guideline recommendations to repeat echocardiographic imaging in adults with Stage B mild AS (aortic velocity 2.0 to 2.9 m/s) every 3 to 5 years, moderate AS (velocity 3.0 to 3.9 m/s) every 1–2 years, and asymptomatic severe AS (Stage C, velocity 4 m/s or higher) every 6 to 12 months.

In trying to understand why severe AS progresses more quickly than mild AS, Drs Dweck and Newby (see page 919) suggest: “One potential explanation is that once established, calcification in the valve proceeds independently from the lipid and inflammatory processes that initiated it. Indeed a self-perpetuating cycle of calcification, altered mechanical stress, valve damage and further calcification may occur in a positive feedback loop” (figure 2).

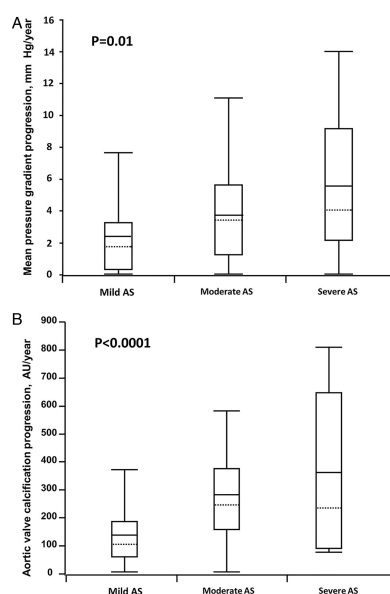
In the absence of medical therapy to prevent or delay disease progression in patients with calcific AS, the only effective

therapy once symptoms supervene is to relieve mechanical left ventricular outflow obstruction by replacing the dysfunctional native valve with a prosthetic valve. In the past the only option was surgical aortic valve replacement (SAVR), a proven effective therapy. Now, transcatheter aortic valve implantation (TAVI) is an attractive alternate therapeutic option. Multiple randomized prospective trials have shown that symptomatic patients with severe AS and a prohibitive surgical risk have improved survival and decreased symptoms with TAVI and that those with a high estimated surgical risk, have an equivalent mortality benefit with TAVI compared to SAVR. The major factor currently limiting the use of TAVI in lower risk patients, is lack of data on longer term clinical outcomes and valve durability.

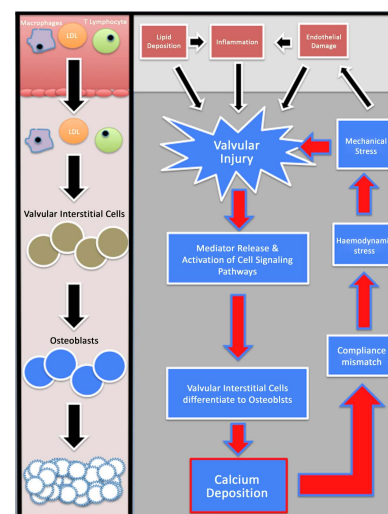
A study in this issue of *Heart* aims to help fill that knowledge gap. In 123 consecutive patients discharged from the Bichat Hospital in Paris after successful TAVI, the overall 6-year survival rate was  $31\% \pm 5\%$  (see page 936). As might be expected given patient age (mean 82

$\pm 8$  years) and the effectiveness of TAVI in relieving outflow obstruction, the majority of deaths were non-cardiac, with predictors of mortality identified as lower limb arteritis, a higher comorbidity score, and moderate to severe aortic regurgitation post-TAVI. Quality of life remained good in survivors at 5 year followup after TAVI (figure 3).

In the accompanying editorial, Drs Newton, Redwood and Prendergast (see page 913) put these findings into the context of other studies on outcomes after SAVR or TAVI for symptomatic patients with severe AS. They conclude that “the reassurance that late deaths after TAVI are not due to cardiovascular causes in the majority of patients does not justify a lowered threshold for TAVI in place of

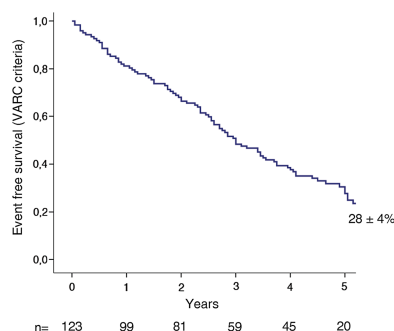


**Figure 1** Yearly haemodynamic (A) and anatomic (B) progression according to the degree of severity of aortic valve stenosis. The box defines the IQR with the mean indicated by the full crossbar and the median indicated by the dotted line. The whiskers indicate the 5th and the 95th percentiles.



**Figure 2** A model for a self-perpetuating cycle of valvular calcification activity in aortic stenosis. Similar to the early stages of atherosclerosis, endothelial injury facilitates the infiltration of oxidised lipids and inflammatory cells into the valve and the release of proinflammatory mediators. These induce valvular interstitial cells to differentiate into osteoblasts, which subsequently coordinate the production of calcium within the valve. Calcification of the valve induces compliance mismatch resulting in increased mechanical stress and injury that triggers further calcification. Hence a self-perpetuating cycle of calcification, valve injury and osteogenic activation is established potentially explaining the exponential increase in disease progression observed as aortic stenosis advances.

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**Figure 3** Event-free survival (events defined according to the Valve Academic Research Consortium-2 (VARC-2) criteria as death, New York Heart Association III–IV, stroke, haemorrhage, hospitalisation for congestive heart failure or aortic prosthesis dysfunction) at 5-year follow-up in patients discharged alive after transcatheter aortic valve implantation.

surgical AVR until adequate data on long-term valve performance become available”.

Our annual Almanac series of review articles summarizes research published in

*Heart* over the previous two years on a specific disease process or research topic. In this issue, the Almanac article on Aortic Valve Disease appears, which provides further background for two research papers mentioned above (see page 929).

Arterial thrombosis after cardiac catheterization is both common and difficult to diagnose in children. Arterial thrombosis occurred in 11.4% of 123 consecutive children undergoing cardiac catheterization via a femoral artery approach at the University Children's Hospital in Zurich over a 12-month period. This complication was more common with younger age (<12 months) and lower body weight. (see page 948) Dr. Krasemann suggests both that guidelines for diagnosis of post-catheterization arterial thrombosis are needed and that “the choice of equipment (smallest possible sheath and catheter, ideally no sheath exchange), heparinisation (ACT>250), type and amount of contrast used, and postprocedural clinical and appa-

allow prospective multicentre studies in larger patient cohorts” (see page 916).

The Education in Heart article in this issue is a beautifully illustrated summary of the “Optimal use of echocardiography in valvular heart disease evaluation” by Dr. Robert J Siegel and colleagues (see page 977). This quick update will be invaluable to echocardiographers and clinical cardiologists who care for patients with valvular heart disease (figure 4).

See the Image Challenge (see page 960) for unusual chest radiograph in a women presenting with acute dyspnea, chest pain and hypotension. Ask yourself both what the chest radiograph shows and why the patient had this finding.



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**Figure 4** Echo-Doppler images of evaluation for aortic stenosis. (A) Parasternal long-axis view demonstrating a heavily calcified (arrows) AV with restrictive opening, also apparent in (B) the short-axis view and on (C) M-mode where cusp separation is measured at 0.8 cm (short arrows). (D) and (E): Corresponding 3D transoesophageal echocardiographic images in the long and short axes, respectively. (F) Doppler measurements show a peak velocity of about 5 m/s with a corresponding peak gradient of 95 mm Hg and a mean gradient of 58 mm Hg. Ao, Aorta; AV, aortic valve; LA, left atrium; PG, pressure gradient.

