Research on sex disparities in patients with coronary heart disease (CHD) largely has focused on events directly related to the coronary arteries themselves—recurrent myocardial infarction, revascularisation and cardiovascular death—rather than the wider consequences of atherosclerotic vascular disease. In contrast, Akyea and colleagues evaluated sex disparities across a broader range of major adverse cardiovascular events (MACE) including not only recurrent CHD, but also stroke, peripheral vascular disease, heart failure and all-cause mortality in a population-based cohort of 143,702 adults (median age 73 years in women and 66 years in men) with no prior cardiovascular events. MACE occurred in 63.8% and recurrent CHD in 46.3% with lower risks of composite MACE (HR 0.68 (95% CI 0.67 to 0.69) or recurrent CHD (HR, 0.60 (0.59 to 0.61) in women compared with men. However, women had a higher risk of stroke (4%, HR, 1.26 (1.19 to 1.33), heart failure (5.5%, HR, 1.09 (1.04 to 1.15) and all-cause mortality (20.5%, HR, 1.05 (1.02 to 1.07), with an older age distribution of events compared with men (figure 1).

In the accompanying editorial, Asleh argues that is ‘time to move from observation to action’ in addressing sex disparities in outcomes after a CHD event. Asleh suggests the following strategies to address these disparities:

► ‘Increase representation of women in preclinical and clinical studies.

► Elucidate further mechanisms response for sex differences in coronary physiology.

► Integrate psychosocial, cultural, race/ethnicity and physiological factors in study designs.

► Improve primary and secondary prevention strategies.

► Educate for seeking early management and improving adherence to medical therapy.

No doubt these strategies would improve outcomes for both women and men with CHD in the long term.

As we perform transcatheter aortic valve implantation (TAVI) for severe AS referred for TAVI for underlying cardiac amyloidosis, AS, aortic stenosis; ATTR, transthyretin cardiac amyloidosis; CA, cardiac amyloidosis; DPD, 3,3-diphosphono-1,2-propanodicarboxylic acid; HMDP, 99mTc-hydroxymethylene diphosphonate; MCF, myocardial contraction fraction; PYP, 99mTc pyrophosphate; TAVI, transcatheter aortic valve implantation.
symptomatic severe aortic stenosis (AS) to older and sicker patients, it has become evident that many patients with AS have concurrent wild-type transthyretin cardiac amyloidosis (ATTR) which may account for persistent symptoms after TAVI. Patel and colleagues compared cardiac remodelling, ventricular function and serum markers in 359 patients with AS alone, 107 with ATTR alone, 35 with both AS and ATTR and 81 matched controls using multimodality imaging, including nuclear scintigraphy. Overall, they found that patients with combined AS and ATTR were similar to those with ATTR alone in terms of carpal tunnel symptoms and diastolic dysfunction, although LV mass was lower in those with AS. Both patients with AS and ATTR (or the combination) had abnormal left ventricular global longitudinal strain and right ventricular annular motion, consistent with early biventricular systolic dysfunction.

Cheng and Griffin point out that the high prevalence of concurrent AS and ATTR is not surprising given that ‘Approximately 25% of people over 85 years of age have ATTR deposition in the myocardium at autopsy, while severe AS affects >3% of individuals over 75 years.’ In addition, ‘the two disease entities share common features, including older age, increased left ventricular (LV) wall thickness, diastolic dysfunction and elevated natriuretic peptides.’ Because treatment of ATTR may improve outcomes after TAVI in patients with combined AS-ATTR, Cheng and Griffin recommend screening in high-risk patients (figure 2).

Another interesting paper in this issue discusses the increased risk of atherosclerotic vascular disease in patients with systemic inflammatory diseases, focusing on the three most common—rheumatoid arthritis, spondylarthritis and inflammatory bowel disease—which affect 5%–7% of the population worldwide. Risk reduction in these patients includes both assessment and treatment of conventional risk factors in conjunction with ensuring optimal anti-inflammatory therapy, with close communication between the cardiology and rheumatology teams in coordination of care.

Another useful article in this issue summarises the use of sodium glucose cotransporter 2 (SGLT2) inhibitor medications in patients with cardiovascular disease. The SGLT2 inhibitors reduce
mortality and hospitalisations in patients with heart failure and in those with Type 2 diabetes complicated by atherosclerotic vascular disease, atrial fibrillation or chronic kidney disease. This article provides practical guidance for cardiologists in identifying patients who might benefit for this newer therapy and avoiding situations in which SGLT2 inhibitor therapy is not appropriate (figure 4).

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