Aortic coarctation is the fifth most common congenital heart disease. Surgical correction early in life is associated with a low surgical mortality and excellent short and mid-term outcomes. However, up to 50% of adults with a history of repaired aortic coarctation repair have hypertension at 30 to 40 years of age. The mechanism of hypertension rarely is recurrent aortic narrowing so it has been hypothesized that these patients also have systemic vascular disease, with findings from previous studies suggesting endothelial dysfunction as the culprit. These finding are refuted in a small but elegant study which has been chosen as the Editor’s Choice for this issue of *Heart*. Dr Radke and colleagues (see page 1696) found no significant differences in endothelial function between 20 patients with a repaired aortic coarctation and 22 normal subjects based on a detailed analysis that included peripheral arterial tomographic measurement of flow-mediated vasodilation, plasma levels of inflammatory mediators and endothelial function, and numbers of circulating endothelial progenitor cells (figure 1).

In the accompanying editorial, Dr Mascherbauer (see page 1657) lists other possible mechanisms of hypertension in patients after aortic coarctation repair including decreased vascular compliance and increased stiffness, activation of the renin-angiotensin system, and increased plasma catecholamine levels or some combination of these factors. She recommends frequent blood pressure monitoring with prompt treatment of recurrent coarctation and aggressive medical therapy of hypertension to prevent adverse cardiovascular outcomes in patients with a history of aortic coarctation.

The optimal parameter for evaluating left ventricular systolic performance has long been a clinical challenge. Over the past 50 years, approaches have included measurement of the amount of blood pumped (cardiac output), the ratio of the volume of blood ejected to the end-diastolic volume (ejection fraction, LVEF), the maximum rate of rise in pressure during contraction (dP/dt) and more complex measures of ventricular function, such as end-systolic elastance based on analysis of pressure-volume loops, which tend to be clinically impractical. Recently, global longitudinal strain (GLS) derived from speckle-tracking echocardiography has been proposed as a valid and reproducible measure of ventricular performance that is less load-dependent than EF and thus a potentially more robust measure of ventricular function. In a systematic review and meta-analysis of the published literature, Professor Marwick and colleagues (see page 1673) found that
GLS was a stronger predictor of all-cause mortality than LVEF based on a total of 5721 subjects from 16 published studies (figure 2).

Despite the promise of GLS as a clinical measure of ventricular function, Dr Szymanski and colleagues (see page 1655) question whether LVEF should be replaced by GLS in routine clinical practice today. GLS represents the average longitudinal component of myocardial strain in a single 2D image plane; the ability to measure 3D GLS is not yet a reality. There continues to be significant variability in measurement of GLS; more reproducible measures are likely as this technology is refined. In addition, they note that “LVEF remains a fundamental parameter for the assessment of LV function and prognosis”. On the other hand, GLS is particularly useful in patients with a normal or mildly reduced LVEF because GLS is more sensitive for detection of early myocardial disease. Thus, despite current technological limitations, it likely that GLS will become a standard clinical tool in the near future as imaging experts gain confidence in measurement and as clinicians become familiar with the value of this data.

A quantitative serum marker of endogenous stress, copeptin, has been suggested as having additional value for diagnosis of acute coronary syndrome in patients presenting with chest pain. However, in a prospective cohort study of patients presenting to the Emergency Department with acute chest pain, Dr Muller and colleagues (see page 1708) found elevated copeptin levels in 215 (22%) of the 984 patients with an adjudicated diagnosis of non-cardiac chest pain. Elevated copeptin levels were associated with older age, pre-existing cardiac and noncardiac disorders and increased serum troponin and serum B-type natriuretic peptide levels. Although an elevated copeptin level (over 13 pmol/L) was associated with a higher 2 year mortality (7% versus 2.5%), this difference was primarily related to age and comorbidities.

The Almanac 2014 review article series continues in this issue of Heart with “Almanac 2014: Cardiac Imaging” by Associate Editor, Satoshi Nakatani. Professor Nakatani summarizes the recent original cardiac imaging research published in Heart put into the context of the important advances in the field over this time period. The Almanac articles provide a quick way for readers to update their knowledge on a specific cardiology topic. Look for additional Almanac papers soon on “Global Health and Telemedicine”, “Congenital Heart Disease” and “Valvular Heart Disease”.

This issue’s Education in Heart article (see page 1722) reviews the utility of cardiac magnetic resonance imaging (CMR) for diagnosis of ischemic myocardial dysfunction and nonischemic cardiomyopathies. Clinicians will find useful practical information about when and why to consider CMR imaging in patients with left ventricular systolic dysfunction as well as a glimpse of the exciting future potential of this imaging modality (figure 3).

Check out the Image Challenge (see page 1695) to see if you can identify this echocardiographic finding in the right atrium.
