Physicians often use a pathophysiologic framework to understand the clinical presentation of cardiac disease and to justify a specific therapeutic intervention. However, our understanding of pathophysiology typically is based on low quality evidence or ‘common sense’ thinking, both of which often turn out to be wrong when rigorous studies are performed. For example, many clinicians erroneously believe that left ventricular (LV) afterload is low in patients with mitral regurgitation (MR) due to a low impedance leak into the left atrium. In this issue of Heart, Gaasch and colleagues\(^1\) provide a mathematical model, with validation based on detailed hemodynamics measurements in a small group of patients, that convincingly demonstrates that retrograde impedance (backflow into the left atrium) exceeds forward impedance (forward flow into the aorta) in patients with severe MR and a normal LV ejection fraction, until regurgitant fraction exceeds 56%. (figure 1) Despite a high retrograde impedance, total impedance (retrograde plus forward) remains low in patients with chronic MR only because LV enlargement allows a normal LV stress-shortening relation.

In an editorial, Martinez-Legazpi, Yotti and Bermejo\(^2\) point out the limitations of using the concept of impedance to characterise valve lesions (figure 2). They conclude: ‘the concept of chronic MR as a disease of pure high-preload and low-afterload is misleading. Afterload is actually normal or above normal in chronic phases of the disease. In addition to increased preload, the abolishment of isovolumic phases is the source of singular biomechanical consequences on the LV. These issues need to be taken into account for a deeper understanding of the ventricular compensatory mechanisms and for making the right clinical decisions in a time of rapidly evolving therapeutic opportunities’.

Dr Blase Carabello provides an even more provocative commentary\(^3\) entitled: ‘A tragedy of modern cardiology: using ejection fraction to gauge LV function in MR’. He puts the current study in context with his comment: ‘The goal in valve disease is to time surgery before LV dysfunction, and its consequences impact outcome. Currently, we are using a 50-year-old load-dependent tool, ejection fraction, to gauge function in the lesion, with the most confusing and least predictable changes in load’. Further, he challenges us to ‘develop biological tools that can peer into the workings of the myocardium to understand and predict when LV contractility is beginning to fall and when LV dysfunction will affect prognosis, tools that can and must replace our ancient, rusty, dull tool, ejection fraction’.

The increased risk of cardiovascular disease in smokers is well established but data is sparse on relative risks in different age groups. With the goal of accounting for population smoking trends, Lloyd and colleagues\(^4\) examined incidence rates and rate ratios for risk of ST elevation MI by age group in South Yorkshire from 2009 to 2012. Smokers under age 50 years had an 8.47 (95% CI 6.80 to 10.54) increase in rate compared with non-smokers of the same age, which was much higher than in older age groups (figure 3).

As so succinctly stated by Dr Arbel in the title of his editorial\(^5\): ‘When will we learn that smoking is bad?’ He goes on to summarise potential mechanisms linking heart disease and smokers including endothelial dysfunction, increased blood viscosity, increased inflammation, platelet activation, metabolic abnormalities, activation of the sympathetic systemic, pro-thrombotic effects and oxidative stress, among others. He recommends that we seek to reduce adverse outcomes related to smoking by focus our efforts on: (1) prevention and education, particularly for young patients, and (2) providing patients with tools to reduce or stop smoking. Simple advice but difficult to implement at the societal level.
The increasing role of advanced imaging modalities for elucidating mechanisms of disease is illustrated by the study of Jenkins and colleagues, which used positron emission tomography (PET), CT imaging and cardiac magnetic resonance imaging (CMR) to study cardiac repair and recovery after recent MI (figure 4). The ability to visualise changes in molecular activity at the tissue level in living patients promises to revolutionise treatment of cardiac disease over the next few decades.

The Education in Heart article in this issue focuses on transoesophageal echocardiography, including indications, risks, pitfalls, and diagnostic value of this imaging modality.

The Image Challenge in this issue requires correct interpretation of an ECG in a young man with exercise induced syncope. These board review style multiple choice questions are a great way to prepare for exams. They are also a fun and easy way to update your knowledge of clinical cardiology with a short discussion related to a clinical image. You can find all the Image Challenges questions on our new website by using the Advanced Search function and searching for the phrase ‘Image Challenge’ in the ‘Full Text or Abstract or Title’ box.

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