In their article, Surmely et al. describe the differences in atherosclerotic plaque morphology, assessed by Virtual Histology (VH) between patients with chronic stable angina and acute coronary syndromes (ACS). Analysed plaques were considered to be the culprit or target lesions. Interestingly, ACS patients’ plaques were characterized by significantly higher content of fibrous and fibro-fatty tissue than lesions observed in stable angina subjects. This observation is opposite to the previously published data, derived from both histopathology and VH studies, which indicate higher necrotic core content in atherosclerotic plaques derived from ACS patients.

VH is a new, promising method of atherosclerotic plaque evaluation in vivo. However, one of the major limitations of the VH system is the inability to distinguish intraluminal thrombus from tissues within the atherosclerotic lesion. Currently available VH software categorizes thrombus as fibrous or fibro-fatty tissue, which in case of high thrombus content leads to inaccurate lesion morphology analysis. That is why the system manufacturer (Volcano Therapeutics Inc.) advises to exclude thrombus from the analysis at the stage of manual lumen tracing. Exact identification of the border between thrombus and the atherosclerotic lesion itself is very difficult especially in the case of lesion rupture when thrombotic mass fills the emptied plaque cavity. Such a situation is very frequent in ACS patients’ culprit lesions. Therefore, in our opinion, at the current stage of VH system development, lesions with either angiographic or IVUS evidence of thrombus should be excluded from the final morphology analysis.

References


From altus to parvus: cardiac fatigue in athletes

With great interest, I read the article ‘Persistent and reversible cardiac dysfunction among amateur marathon runners’ by Nellon et al. Although the mechanism of such ‘cardiac fatigue’ is unclear, it is likely related to the increased stresses imposed by exercise which affect the functions of right and left ventricles.2,3 This study has demonstrated that volume and cavity depletion may be a trigger, but are not the major factors for persistent cardiac dysfunction after prolonged exercise. That weight and left atrial diameter returned to baseline values during the follow-up period support this conclusion. It can be hypothesized that the mechanism by which this injury could arise involves a vicious cycle of heart and muscle.4 The vicious cycle would occur among relative cardiac deterioration and oxygen kinetics in the muscle. In other words, impaired skeletal muscle aerobic energy provision and work capacity during maximal aerobic exercise in healthy, trained humans are directly related to the inability of the heart to maintain output and oxygen delivery to locomotive skeletal muscle.

Interestingly, after prolonged exercise, changes in diastolic performance are increased velocity of atrial or late diastolic inflow, and result in a decrease in the ratio of early-to-late flow velocities.2,6 In contrast, these changes were rapidly reversible, and the left ventricular filling pattern during the 28 h of recovery is not persistent.2 Although more research is needed to better understand the molecular mechanisms by which exhaust physical activity suppresses diastolic function of left ventricle along period (persistent). I could not find authors’ hypothesis about this interesting result. As a general opinion,