LETTERS TO THE EDITOR

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The role of endomyocardial biopsy in the management of cardiovascular disease: a Scientific Statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology

May we draw your attention to the ESC/AHA/ACC scientific statement published in the December issue of the European Heart Journal.1 Scenario 10 discusses the indication for endomyocardial biopsy in patients with suspected myocardial siderosis (due to hereditary or acquired haemochromatosis). This states that ‘cardiac involvement in haemochromatosis usually can be diagnosed on the basis of history, clinical examination, and echocardiography or cardiac magnetic resonance (CMR) demonstrating dilated cardiomyopathy in the setting of laboratory abnormalities such as elevated serum iron and haemochromatosis gene mutation’. On the contrary, cardiac siderosis often presents late, and ventricular dimensions may be normal until the late stages of disease.2 In addition, conventional markers for iron overload such as serum ferritin and liver iron have been shown to bear no relation to myocardial iron deposition in the commonest form of acquired myocardial siderosis, beta-thalassaemia major.3 However, if access to CMR is indeed available, a robust, simple and quick measurement of myocardial T2* (CMR-measured myocardial T2*) is not available or if aetiologies other than iron overload are being assessed.

References

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The role of endomyocardial biopsy in the management of cardiovascular disease: a Scientific Statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology: reply

We thank Dr Anderson for pointing out that in patients with iron overload in the heart irrespective of the underlying aetiology, e.g. thalassemia, sickle cell anaemia, chronic transfusion therapy, or hereditary haemochromatosis, there is no need for endomyocardial biopsy. Magnetic resonance imaging (MRI) is now an accepted method for the evaluation of myocardial iron overload, and the method has been validated in multiple laboratories.1 It has been shown that serum ferritin measurements are a reasonable surrogate of liver iron, but are poorly correlated with cardiac iron stores. Recently, a new method using the cardiovascular magnetic resonance (CMR) T2* technique has been used for the measurement of tissue iron in the liver, but what has not yet been shown is calibration of myocardial T2* against absolute myocardial iron levels in humans.2,3 In the clinical setting, T2* measurements have been used to assess cardiac iron loading and the studies suggest a useful correlation between myocardial T2* and cardiac function thereby allowing early diagnosis and treatment. Although there is no disputing the clinical merits of CMR-measured myocardial T2* in the evaluation of cardiac function, there may be additional value in knowing the relationship between myocardial T2* and directly measured myocardial iron levels. Validation of myocardial T2* and myocardial tissue iron may not be feasible in humans and may not be required since cardiac iron estimates by MRI in animal models has been validated.4

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