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Letter to the Editor

Value of cardiac magnetic resonance tagging to detect subclinical contractile dysfunction in Duchenne muscular dystrophy

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In a recent cardiac magnetic resonance (CMR) strain imaging study on 13 Duchenne-muscular-dystrophy (DMD) patients and 9 healthy controls, Ashford *et al.*, (2005) found that subclinical myocardial contractile dysfunction can be detected by CMR-tagging (Ashford Jr, M. W. *et al.*, 2005). We have the following comments and concerns.

According to figure 1 mean normal circumferential strain was reduced in the mid-ventricular and basal segments of the left ventricular (LV) myocardium compared to the apex. On the contrary, mean circumferential strain continuously increased from the apex to the basal segments in DMD-patients. How to interpret the reduction of circumferential strain in controls and the variant behavior in DMD-patients? Why was strain not different between patients and controls in the apex? Differences between the two groups for the mid-wall and basal segments may be simply due to reduced strain values in controls. How to explain the increased variance of strain values in DMD-patients?

Mean heart-rate was significantly increased in the DMD-group, a well known phenomenon in these patients. How can one exclude that CMR findings were simply due to tachycardiomyopathy? Were myocardial net twists or strain dependent on heart-rate in patients and controls? Do strain values change after pharmacological reduction of the heart-rate? Was increased heart-rate induced by stress-immanent CMR?

Since CMR-tagging is a new, user-dependent technique, variant subjective measurements between observers may influence the results. Was inter-observer variability assessed and did it influence the results? It is also not mentioned if the investigators were blinded to the diagnosis during data analysis.

Recently it has been shown that subclinical myocardial affection can be documented by tissue Doppler imaging and backscatter analysis (Giglio, V. *et al.*, 2003). Why were the CMR findings not compared with these methods? Why were no echocardiographic investigations reported?

It remains uncertain how the inclusion criterion "without known heart disease" was defined? It is well known that cardiac involvement starts already in the early stages of the disease and by age 10y nearly all patients present at least with ECG abnormalities (Nigro, G., *et al.*, 1990). Were ECG abnormalities other than sinustachycardia or echocardiographic abnormalities found in DMD-patients? Was reduced strain related to thorax deformity or noncompaction, previously reported in DMD (Finsterer, J. *et al.*, 2005; Sussman, M. 2000)?

It is mentioned that the diagnosis was based on creatine-kinase levels, muscle biopsy and genetic investigations. In how many patients was the diagnosis genetically confirmed and was there a correlation between the type and location of the mutation and CMR-tagging?

Following these objections and the limitations addressed by the authors the conclusion that CMR-tagging may detect subclinical regional myocardial dysfunction is questionable.

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