

dysregulation featuring intramyocardial triglyceride overload and gene expression that are associated with contractile dysfunction (3). In the end, after more than 150 years we are all still walking in Virchow's footsteps.

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REPLY: In the Footsteps of Virchow

We thank Drs. Taegtmeier and Lam for their interest in our recent publication (1) and for the opportunity to discuss their comments.

It is entirely true that fat deposition is part of the normal physiological process of involution. It occurs with aging just as Rudolph Virchow described in 1858 (2). However, the subjects selected for our study were patients with idiopathic dilated cardiomyopathy. We tried to investigate the prevalence of fat deposition in a particular group of subjects and its effects on cardiac functions.

With respect to the intramyocellular or extramyocellular fat, it was reported that intramyocellular lipid could be detected by magnetic resonance spectroscopy (MRS) in the musculoskeletal system (3). Depending on the observed nucleus, MRS provides information on the chemical composition of the tissue. It allows the observation of high-energy phosphates (31P-MRS), glycogen (13C-MRS), or intramyocellular lipids (1H-MRS) based on the nomenclature in muscle spectra recorded in vivo (the CH₂ signal at 1.28 parts per million is usually attributed to intramyocellular lipids and the shifting CH₂ resonances as extramyocellular lipids). However, it is at the cost of a lower spatial resolution by using MRS investigating the chemical composition of the tissue. It is still a challenge to distinguish intramyocellular lipid from extramyocellular lipid with current spatial resolution provided by cardiac magnetic resonance.

Sharma et al. (4) found that intramyocardial triglyceride overload was associated with cardiac contractile dysfunction. Unfortunately, they did not provide medical images or pathological specimen photographs demonstrating fat deposition in the human heart. Maybe such intramyocellular lipid was too small to be seen by the naked eye. Because there was a case of heart transplantation in our study, which was confirmed by pathology, we consider that the fat signal detected by cardiac magnetic resonance in this study is very probably extramyocellular fat instead of intramyocellular fat.

With regard to the "omitted patients' metabolic profiles," we really did not intend to. Because patients in this study had non-ischemic cardiomyopathy, the majority of venous blood lipid results were within normal limits. We did not collect patients' metabolic data and perform further statistical analysis. It may be a limitation of our paper according to the findings by Sharma et al. (4). Anyway, their comments are interesting and point in an important direction to pay attention to the effects of lipid metabolism on cardiac function.

After more than 150 years, we are all still following Dr. Virchow's footsteps. However, we have seen farther by standing on his shoulders.

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