Letter to the Editor: The effect of autonomic nervous system on the impairment of glucose uptake and lipid metabolism in epicardial adipose tissue

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TO THE EDITOR: We read with great interest and excitement the recently published work of Burgeiro et al. (3) about the impairment of glucose uptake and lipid metabolism in epicardial adipose tissue from heart failure patients with or without diabetes. The authors stated the difference between basal and insulin conditions in glucose uptake within epicardial compared with subcutaneous adipocytes. They also found a significant decrease in the isoproterenol-stimulated lipolysis, comparing the two fat depots. In this letter, we aim to emphasize another possible mechanism between fatty tissues and significant changes in glucose and lipid metabolism: the contribution of autonomic nervous system function.

In the literature, Anaruma et al. (1) examined nine subjects with type 1 diabetes mellitus and 10 healthy control subjects about the cardiovascular parameter and plasma biomarker baseline and the heart rate variability and blood glucose in response to a session of aerobic exercise (AE) and during the recovery period. These authors concluded that type 1 diabetes mellitus patients on insulin therapy have poor blood glucose control with greater lipid peroxidation and lower nitrite/nitrate levels accompanied by an imbalance in autonomic function detected by the changes of AE. Fandiño-Vaquero et al. (5) investigated the orosomucoid (ORM) secretion levels from epicardial adipose tissue in patients undergoing cardiac surgery. They stated that EAT-released ORM levels were higher than those in subcutaneous fat tissue but that EAT secretion was lower in patients with type 2 diabetes mellitus than those without type 2 diabetes mellitus, and this difference was enhanced after isoproterenol stimulation. The authors concluded that EAT-released ORM levels in patients with type 2 diabetes mellitus compared with those without type 2 diabetes mellitus or CAD and its regulation by catecholamines might be the mirror of local endothelium dysfunction or inflammatory processes in different cardiovascular disorders.

There are several studies demonstrating a close relationship between EAT and cardiac autonomic function. A previously published study from Zhou et al. (6) revealed that the cardiac ganglionated plexus in the epicardial fat incorporates the autonomic innervation between the extrinsic and intrinsic cardiac autonomic nervous system and affects atrial electrophysiology and pathophysiology. Additionally, Balcioglu et al. (2) stated that sympathovagal imbalance, detected by heart rate variability and turbulence parameters, is associated with epicardial fat thickness. Because sympathovagal imbalance is a predictor of arrhythmic events, epicardial fat may play an important arrhythmogenic role. Carnevali et al. (4) concluded that epicardial fat deficiency in mice leads to an imbalance of the autonomic neural modulation of cardiac function in the sympathetic direction and to a potentially proarrhythmic remodeling of electrical and structural properties of the heart.

As a consequence, we believe that the impairment of cardiac autonomic nervous system function may be helpful in explaining the results of the article by Burgeiro et al (3). We hope that the above-mentioned items will add to the value of the well-written article by Burgeiro et al. (3) regarding the impairment of glucose uptake and lipid metabolism in epicardial adipose tissue from heart failure patients with or without diabetes.

DISCLOSURES
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AUTHOR CONTRIBUTIONS
H.K. conception and design of research; K.O. edited and revised manuscript; A.K. approved final version of manuscript.

REFERENCES

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