Postcontractile blood flow as a window to cardiovascular disease?

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A historically important topic in physiology is the control of arterial blood flow (1). Numerous studies have shown that multiple vasodilatory mechanisms exist and that there is enough redundancy and compensatory interaction to make studying the control of blood flow a difficult topic (5). One of the important methods of studying blood flow is to characterize the onset kinetics of oxygen consumption and blood flow (8). These studies have identified a number of different “phases” to the blood flow response, which may be attributed to various dilating mechanisms.

In an article in this issue of the *Journal of Applied Physiology*, Towse and colleagues (12) provide a new approach that uses MRI to evaluate the blood flow responses to a single, brief muscle contraction. As the mechanisms for determining blood flow may well be the same for a single contraction as for the studies on the onset of steady level exercise, this approach may have merit. A single contraction is potentially easier, quicker, and less complicated than performing a longer duration of exercise. Utilizing a combination of methodologies [MRI and near-infrared spectroscopy (NIRS)], Towse et al. (12) found that a short-duration contraction shows two phases. Because of the short contraction duration (1 s) oxygen consumption is not sufficient to cause significant decreases in blood or muscle oxygen consumption; in addition there is an increase in oxygen saturation around 8–10 s after the contraction. This corresponds nicely with an increase in hyperemic blood flow at the same time. A second phase of prolonged hyperemia occurs shortly after, that lasts for 20–30 s. The strength of this study is the use of multiple measuring methodologies and the use of a comprehensive metabolic model that can predict the changes in blood volume, blood flow, and tissue oxygen content after a short-duration exercise. What is especially important to investigators is the demonstration that the NIRS and MRI measurements are in general agreement, and that even MRI flow measurements agree with Doppler ultrasound flow measurements.

As a physiological study, the paper by Towse et al. (12) confirms and extends our understanding of the control of blood flow and muscle metabolism in response to exercise. For example, the paper provides evidence that there is not an “inertial” component to the activation of mitochondrial oxygen consumption. Additionally it provides evidence that the initial phase is consistent with K⁺-induced hyperemia as well as the second phase being more consistent with a tissue oxygen-related dilation. Finally the paper provides support for the mechanisms behind the BOLD effect, which is the change in the relaxation rate of protons attributed to increased oxygen binding to hemoglobin.

A hot topic in physiology and medicine is quantifying the onset and magnitude of cardiovascular disease (4). For example, one of the initial changes in the development of cardiovascular disease is the loss of nitric oxide-related vasodilatation, often measured as flow mediated dilation (FMD) (6, 9). Numerous studies have reported reduced FMD for various disease and predisease states, including diabetes (3, 7, 10). A major advantage of the FMD test is that it is noninvasive and relatively quick to administer. So the question becomes, can the measurements presented by Towse et al. (12) be used to evaluate the onset or magnitude of cardiovascular disease? As shown by Towse et al. (12) and a previous study (13), prior physical activity seems to play a role in the vascular response to the one second contraction. But can vascular pathology be separated out from fitness-related changes? An older study clearly showed that the time course of the hyperemic response to exercise or ischemia is delayed in people with known peripheral arterial disease (11). In addition, the onset kinetics of oxygen consumption at the start of exercise seems delayed in people with Type 2 Diabetes (2). The extent to which the 1-s contraction can be useful may depend on its sensitivity to endothelial cell to smooth muscle cell communication in the vascular system, and future studies will be needed to address this.

The paper by Towse and colleagues (12) certainly represents a classical paper in terms of its comprehensive approach to vascular physiology. The combination of a number of different methodologies, including metabolic modeling, should serve as a bridge paper between different groups of investigators, who may be tempted to focus just on papers using their preferred methodology. It is refreshing to see the agreement between different methodologies and to see a paper that provides mechanistic explanations for their observations. The approach in the article by Towse et al. (12) has the potential to advance our understanding of the physiology of cardiovascular disease.

**DISCLOSURES**

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**REFERENCES**


