Radiology

Tracking Myocardial Oxygenation over a Breath Hold with Blood Oxygen Level–Dependent MRI: A Radically Different Approach to Study Ischemia

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In this issue of *Radiology*, van den Boomen et al reported clinical pilot data on the utility of a T2*-sensitive cardiac MRI sequence for tracking changes of myocardial T2 and T2* during a breath hold (1). By using a hybrid sequence, they acquired T2 and T2* maps every heartbeat during a voluntary breath hold in 18 healthy control participants and eight participants with arterial hypertension. During the breath hold, T2 and T2* increased in healthy control participants, whereas there was a net decrease in seven of eight participants with arterial hypertension.

The study is small and has limitations but, despite its preliminary value, the results provide some interesting observations. First, T2 and T2* maps can be generated with short acquisition times, which may help shorten imaging times in participants suspected of having myocardial disease or injury. Myocardial T2 is a water-sensitive parameter and therefore useful for identifying acute myocardial injury. Second, T2 and T2* showed reproducible changes during a breath hold, confirming previous reports in experimental and clinical studies. T2* has been successfully applied for identifying myocardial iron overload. T2*-sensitive cardiac MRI has also been proposed to monitor changes of myocardial oxygenation. Clinical studies have shown a diagnostic value of oxygenation-sensitive cardiac MRI to monitor adenosine-induced vasodilation in coronary artery disease (2,3), valvular heart disease (4), and microvascular dysfunction (5).

Another important observation is that T2 and T2* changed predictably during a voluntary breath hold. It is known that the coronary vasculature shows a strong response to breathing maneuvers. Like the brain, hyperventilation induces vasoconstriction with a subsequent drop of blood flow, whereas apnea (eg, a voluntary breath hold) leads to vasodilatation followed by a strong increase of coronary blood flow (6) (Figure). These responses kick in within seconds, with a plateau reached at about 30 seconds. Abnormal responses have been reported in coronary artery disease, sleep apnea syndrome, and heart failure.

The clinical potential of short breathing maneuvers instead of pharmacologic vasodilation with adenosine or regadenoson is immense. This safer approach does not require intravenous access and it is less costly, more time efficient, and without clinically significant adverse effects.

The study by van den Boomen and colleagues (1) leaves several open questions: First, whereas the apnea-induced T2 and T2* increase is consistent with previous data and with expected physiologic changes (7), it remains unclear why the authors found a net decrease of both T2 and T2* in participants with hypertension. So far, such a paradoxical response has only been reported in segments exposed to severe coronary artery stenosis (8). Second, because maps were acquired during diastole with a readout time of 120 msec, observed changes may have been confounded by the timing of data acquisition during the cardiac cycle. The variation of myocardial blood flow over systole and diastole is known to translate into concordant changes of the signal intensity in T2*-weighted images (9). Another potential confounder is the breathing pattern of the participants before the actual measurement. Relative hyperventilation or hypoventilation may have altered the baseline of the measurements and thus the observed difference and slope. Furthermore, the limit of five data points for curve fitting induces inaccuracies. Finally, because of the small sample size, other uncontrolled confounders such as blood pressure during the examination, lifestyle, and medication may have had an impact on the results.

Despite these limitations, the study by van den Boomen et al (1) confirms the potential for oxygenation-sensitive cardiac MRI to be used as a diagnostic tool on the basis of a relevant marker (myocardial oxygenation itself). Furthermore, it sheds more light on cardiac MRI combined with breathing maneuvers as a safe, simple, and fast diagnostic test for coronary vascular function.

Combined with fast cardiac mapping, a short and noncontrast agent–enhanced cardiac MRI protocol would allow for a rapid assessment of cardiac function, tissue status, and vascular function. Because the majority of cardiac MRI is about cardiomyopathies and coronary artery disease, a

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Conflicts of interest are listed at the end of this article

See also the article by van den Boomen et al in this issue.

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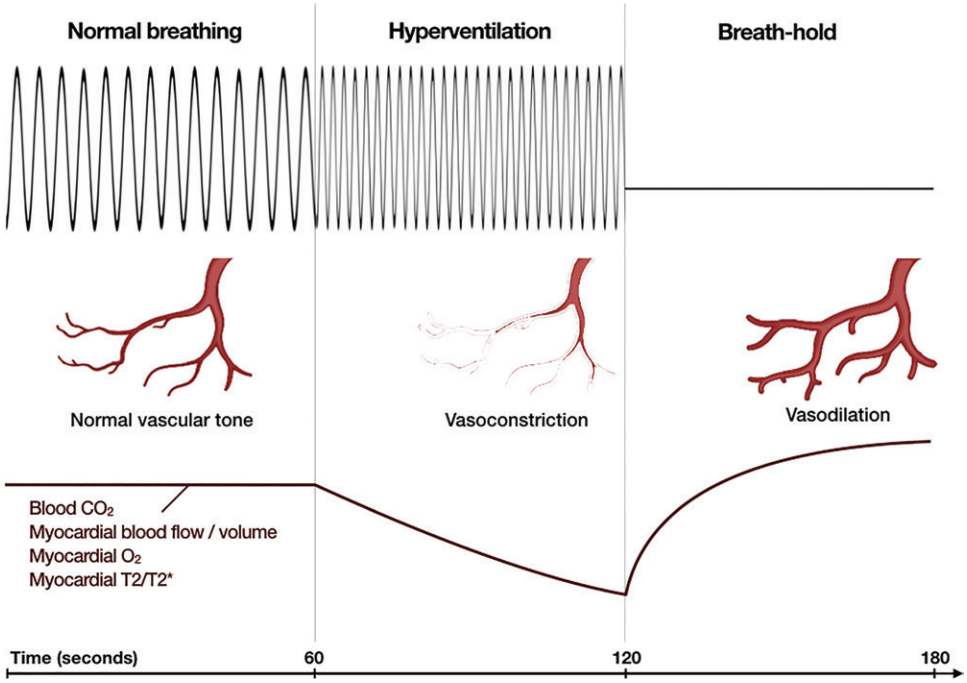

The coronary vascular response to hyperventilation and breath holds. The decrease of CO_2 during hyperventilation leads to vasoconstriction, which results in a reduction of myocardial blood flow and volume and subsequent decrease of myocardial O_2. T2 and especially T2* change accordingly, showing a decrease during hyperventilation and an increase during a breath hold.

short needle-free protocol could have massive implications on workflows in specialized centers.

To explore this potentially disruptive approach, the next steps will have to be answering remaining technical questions (eg, mapping vs T2*-weighted images, hybrid vs other sequences, and temporal resolution vs signal to noise), identifying the most robust breathing protocol for clinical application, and clinically validating the approach in larger studies and various diseases.

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