Myocardial bridging with systolic milking is a frequent finding during coronary angiography. Classically, it is considered a benign congenital anomaly because myocardial perfusion occurs in diastole. Milking is limited to systole and should therefore not impair myocardial perfusion. However, this physiology-based evidence is contradicted by numerous cases of coronary thrombus formation and myocardial infarction, in individuals with pathological findings none other than a myocardial bridging. Pressure-derived fractional flow reserve (FFR) measurement during maximum myocardial hyperemia is an established technique to assess the hemodynamic significance of moderate stenoses in atherosclerotic coronary artery disease. Few data are available concerning its use in myocardial

Figure. Left, Midsystolic image of the left coronary artery in anteroposterior view, with 20° of cranial inclination. Just distal to a septal branch, myocardial bridging and “milking” is observed in the left anterior descending artery (encircled in white). Right, End-diastolic view in the same incidence. The segment just distal to the septal branch is smooth and free of atherosclerotic disease. The lower insert of the figure depicts the pressure tracings and their respective values in mm Hg at the level of the aorta (in purple) and in the left anterior descending artery distal to the segment with systolic compression (in green). The ratio of distal pressure to aortic pressure (FFR) is depicted in yellow. Values are mentioned for mean and diastolic pressures (mFFR and dFFR, respectively). The distal measurements were obtained using a 0.014-inch pressure wire, after induction of maximum hyperemia with an intracoronary bolus of adenosine. Left to right: Measurements in baseline condition, during intravenous infusion of dobutamine at 20 μg · kg⁻¹ · min⁻¹, during intravenous infusion of dobutamine at 30 μg · kg⁻¹ · min⁻¹ and administration of 1 mg of atropine, and finally after intravenous administration of 10 mg of metoprolol. The respective heart rates (bpm) are mentioned in blue. At a dose of 20 μg · kg⁻¹ · min⁻¹ of dobutamine, systolic pressure overshoots distally to the dynamic lesion with a diastolic pressure gradient ~0.75. At the highest dose of dobutamine and after administration of atropine, an important diastolic pressure gradient is seen (55/77 ≈ 0.71), indicating impaired myocardial perfusion by the myocardial bridge. This gradient disappeared almost instantly after the intravenous administration of metoprolol, despite a higher heart rate than that during the intravenous infusion of dobutamine at a dose of 20 μg · kg⁻¹ · min⁻¹.
where the evaluation of the hemodynamic significance of frequently encountered mild and moderate cases of myocardial bridging may be a diagnostic dilemma for the clinician. In particular, outcome data to guide therapy based on FFR measurements are unavailable for myocardial bridging as opposed to coronary atherosclerosis.

We performed FFR measurements in a 24-year-old professional cyclist who presented with a prolonged episode of exertional chest pain, and ST-segment inversion in the anterior ECG leads, and left ventricular angiography–confirmed anterior hypokinesia on echocardiography. Coronary angiography revealed myocardial bridging with moderate systolic milking distal to a septal branch of the left anterior descending artery. After intracoronary administration of nitroglycerin, pressures were measured at the level of the guiding catheter and distal to the segment with systolic compression (0.014-inch pressure wire, Brightwire, Volcano Corporation, Rancho Cordova, Calif) in baseline conditions and during infusion of increasing doses of dobutamine (5, 10, 20, and 30 μg · kg⁻¹ · min⁻¹, each in 4-minute steps, administered through the femoral vein). On top of the highest dobutamine dose, 1 mg of atropine was given. As a final step, metoprolol was slowly administered intravenously to a total dose of 10 mg. At the end of each 4-minute step, maximum hyperemia was produced using a 40-μg intracoronary bolus of adenosine.

The myocardial bridge had no significant hemodynamic effect in baseline conditions (reflected by normal mean and diastolic FFR measurements). During infusion of dobutamine, maximal hyperemia induced by adenosine decreased diastolic FFR to <0.75, indicating impaired myocardial perfusion (Figure 1). Interestingly, the gradient disappeared after the intravenous administration of metoprolol, which was mirrored clinically by the disappearance of chest pain, despite a higher heart rate than that during the infusion of 20 μg · kg⁻¹ · min⁻¹ dobutamine.

Disclosures
None.

References

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Hemodynamic Effect of Myocardial Bridging
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