Haemodynamic findings in obstructive hypertrophic cardiomyopathy: pulsus bisferiens and Brockenbrough–Braunwald–Morrow sign
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A 55-year-old gentleman with hypertrophic obstructive cardiomyopathy and heart failure symptoms underwent cardiac catheterization, which confirmed a significant pressure drop (60 mmHg) across the left ventricular outflow tract, a double-peaked pulse (pulsus bisferiens) and an absent postextrasystolic potentiation (Brockenbrough–Braunwald–Morrow sign) in the left ventricular outflow tract and the aorta. He was treated with medical therapy optimization and intracardiac defibrillator implantation. Cardiac catheterization may provide characteristic clues not only to diagnose obstructive hypertrophic cardiomyopathy, but also to understand its pathophysiological correlates.

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A 55-year-old gentleman with a previous diagnosis of hypertrophic obstructive cardiomyopathy was admitted because of worsening shortness of breath and chest pain. At physical examination, he presented a mid-systolic murmur at the base, as well as a pan-systolic murmur at the apex. His ECG showed left bundle branch block, his echocardiography confirmed an asymmetrical septal hypertrophy (40 mm thick) with left ventricular outflow tract (LVOT) obstruction (mean gradient 37 mmHg), systolic anterior motion of the anterior mitral leaflet and moderate mitral regurgitation (Fig. 1a). He underwent cardiac catheterization, which showed a normal (single-peaked) shape and a preserved postextrasystolic potentiation in the left ventricle (Fig. 1b) while confirming a significant pressure drop (60 mmHg) across the LVOT (Fig. 1c). Compared with left ventricular cavity, the LVOT (Fig. 1c) presented no postextrasystolic potentiation (Brockenbrough–Braunwald–Morrow sign) as well as a double-peaked pulse (pulsus bisferiens), which persisted within the aorta (Fig. 1d). After medical therapy optimization with atenolol 100 mg/day and verapamil 240 mg/day, the patient was implanted with an intracardiac defibrillator and remained nearly asymptomatic during a 6-month follow-up.

Around one-third of patients with hypertrophic cardiomyopathy present dynamic obstruction at rest, and another one-third present inducible obstruction during stress.\textsuperscript{1} Apart from the typical pressure drop across the LVOT, cardiac catheterization in patients with advanced dynamic obstruction may also disclose the Brockenbrough–Morrow sign:\textsuperscript{2} the postextrasystolic potentiation, reflected by a transient increase in intraventricular pressure following a premature ventricular contraction, is not propagated through the LVOT because of the concomitant greater dynamic obstruction, so that the LVOT and the aortic postextrasystolic pressures do not increase.

Fig. 1

Asymmetrical septal hypertrophic cardiomyopathy with left ventricular outflow tract (LVOT) obstruction, at echocardiography [(a) four-chamber and three-chamber apical views; the black bar represents maximal septal thickness] and at cardiac catheterization [(b) pressure recording inside the left ventricle; (c) pressure recording across the LVOT; (d) pressure recording across the aortic valve; asterisks indicate postextrasystolic beats; arrows indicate double-peaked pulses].
Moreover, the Venturi effect across the LVOT causes a constant midsystolic pressure drop in the LVOT and the aortic arterial pulses, seen as a double-peaked pulse called pulsus bisferiens, originally described in aortic stenosis. These pathophysiological findings, traditionally seen with cardiac catheterization, should also be searched for during a thorough physical examination and during echocardiography.

References