A lesson in the power of observation

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Disclosures: Author has nothing to disclose with regard to commercial support.

Received for publication May 13, 2018; revisions received May 13, 2018; accepted for publication May 14, 2018; available ahead of print June 28, 2018.

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J Thorac Cardiovasc Surg 2018;156:1616

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2018.05.049>

Teaching the skill of observation is often shortchanged in medical education.¹ As William Osler famously noted, the whole art of medicine is in observation. In this issue of the *Journal*, Cui and colleagues² from the Mayo Clinic provide an example of how the power of a simple observation can have enduring educational value in our approach to diagnosis and management of human disease. The Brockenbrough-Braunwald-Morrow sign was first described in 1961 by the eponymous clinicians³ in patients presenting with dynamic left ventricular outflow tract obstruction and is characterized by a decrease in arterial pulse after a premature ventricular contraction, along with a significant increase in peak left ventricular systolic pressure. Since its first description, numerous examples of this phenomenon has been described in the medical literature, including the report by Cui and colleagues,² attesting to its current relevance. Cui and colleagues² provide clear examples of this phenomenon and demonstrate its absence in the setting of fixed valvular stenosis and setting of membranous and tunnel subaortic stenosis not associated with systolic anterior motion of the mitral valve.

Of interest is why this phenomenon occurs. The explanation for the occurrence of the Brockenbrough-Braunwald-Morrow sign was commonly attributed to a reduction in stroke volume due to an increase in dynamic obstruction. Brockenbrough and associates³ attributed postextrasystolic potentiation to increased diastolic filling as a consequence of the Frank-Starling curve with resulting increased force of contraction. Postextrasystolic potentiation can be

The Brockenbrough-Braunwald-Morrow sign.

Central Message

The Brockenbrough-Braunwald-Morrow sign is an important maneuver in the diagnosis of hypertrophic cardiomyopathy.

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demonstrated, however, in the setting of atrial pacing in the absence of a compensatory pause.⁴ The increase in inotropy is thus more attributable to extracellular calcium ion that enters the cardiac myocyte during a premature beat and is stored in the sarcoplasmic reticulum. The reduction in stroke volume occurs with increasing dynamic obstruction and, in some instances, worsening mitral regurgitation related to systolic anterior motion of the mitral leaflet.

The report by Cui and colleagues² is not only an important lesson in the pathophysiology of a prevalent disease but a lesson in how, as physicians, we need to practice with our eyes wide open.

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