On systolic murmurs and cardiovascular physiological maneuvers

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Submitted 13 December 2011; accepted in final form 14 August 2012

Salazar SA, Borrero JL, Harris DM. On systolic murmurs and cardiovascular physiological maneuvers. Adv Physiol Educ 36: 251–256, 2012; doi:10.1152/advan.00128.2011.—Physiological principles that directly apply to physical diagnosis provide opportune occasions to bring basic science to the bedside. In this article, we describe the effect of cardiac maneuvers on systolic murmurs and how physiological principles apply to the explanation of the changes noted at the bedside. We discuss the effect of Valsalva, squatting, and hand grip maneuvers on different physiological parameters influencing preload, afterload, chamber dimensions, and pressure gradients. The clinical manifestations noted during the aforementioned maneuvers are described in common cardiac conditions responsible for the production of certain systolic murmurs.

Valsalva; aortic stenosis; squatting; hand grip; hypertrophic obstructive cardiomyopathy

MAKING THE TRANSITION from basic science knowledge to clinical application has always been a challenge in medicine. In particular, physiological principles that have direct clinical pertinence are difficult to demonstrate at the bedside. There have been attempts to use patient simulators to improve cardiovascular physiology understanding, with success (12). The use of information technology to enhance most curriculums is standard and has contributed to improvements in information acquisition and time efficiency.

Although medical information is much more easily accessed than in the past, the clinical skills used for bedside diagnosis have declined (1). There is increased dependence on nonbedside testing to guide the clinician at arriving at a working diagnosis. While the void between technology and “laying of hands” increases, our capacity to apply physiological principles to the clinical assessment deteriorates. I believe that we, as educators, have a responsibility to bridge basic science to the bedside. In this article, we describe the basic principles underlying blood flow that contribute to these sounds. Blood flow is often simplified by Ohm’s law of hydrodynamics (flow = Δpressure/resistance), which applies to all vessels. Since blood vessels are viewed as rigid, cylindrical tubes, the resistance variable can be further characterized according to Jean Poiseuille’s studies on liquid flow in straight, rigid, cylindrical tubes. Ohm’s law of hydrodynamics was modified to give us the Poiseuille-Hagen equation: flow = (Δpressure × πr^4/8 × viscosity × length), where r is the radius. To relate the flow of blood to this equation would require six assumptions:

1. The fluid is incompressible.
2. The tube is straight, rigid, cylindrical, and unbranched and has a constant radius.
3. The velocity of the fluid layer at the wall must be zero.
4. The flow is laminar.
5. The flow is steady, not pulsatile.
6. The viscosity is constant.

While it can be argued that blood flow abides by the first three assumptions, it should be evident that the last three assumptions are not met. For the sake of this article, we will focus on assumptions 4 and 5 and how they relate to murmurs.

**Laminar versus turbulent blood flow.** According to Ohm’s law of hydrodynamics (flow = Δpressure/resistance), flow should increase linearly with the driving pressure (Δpressure) if resistance is constant. This results in laminar blood flow, which can be described as concentric layers of blood moving parallel in the vessel with high velocity in the center of the vessel and low velocities along the walls. Interestingly, at high flow rates, flow rises to a lesser degree and is no longer proportional to driving pressure. This is due to an increase in resistance, which is a consequence of turbulent or nonlaminar blood flow. The point in which laminar blood flow becomes turbulent is called the Reynolds number and is defined by the following equation:

Reynold’s number

\[ = \left(2 \times \text{diameter} \times \text{velocity} \times \text{density}\right) / \text{viscosity} \]

This number lacks units, and blood flow is laminar below 2,000 and highly turbulent above 3,000. The two most likely causes of an increased Reynolds’s number and therefore turbulent blood flow are increased velocity of blood flow and low viscosity (reduced hematocrit). An increase in diameter could
apparently lead to an increased Reynold’s number; however, it is important to recognize that increased diameter is usually associated with a decreased velocity because velocity is proportional to \(1/\text{diameter}^2\).

The differentiation between laminar and turbulent blood flow is clinically significant. Under normal conditions, blood flow is laminar and silent. Turbulent blood flow generates noise due to the vortexes of blood, which can be heard via stethoscope. The most common causes of heart murmurs include mechanisms that affect diameters of vessels or valves, thus affecting the velocity of blood flow through them. Therefore, techniques that can alter velocities through these areas and can change the amount of turbulence are important in the clinical assessment of murmurs.

**Blood flow is pulsatile.** Assumption 5 requires that flow is steady, not pulsatile. This is obviously not evident in our cardiovascular system, in which the ventricles serve as a pressure generator, which contracts and relaxes. This results in the systolic and diastolic blood pressures within the cardiovascular system. More specifically, the pressure increase caused by the isovolumetric contraction of the ventricle is potential energy. The velocity of blood flow during ejection comprises the kinetic energy. According to Bernoulli’s principle, it is the summation of potential and kinetic energy that comprises the total energy. Therefore, it is more accurate to say that blood moves from higher to lower total energies, as opposed to higher to lower pressures. Additionally, the basis of Bernoulli’s principle is that potential and kinetic energy can be interconverted. The best example of this is a blood vessel in which a narrowed portion exists in a central region of a horizontal tube. The pressure in the narrowed portion is less than the accompanying section downstream due to the high resistance, yet flow still persists. Since flow is equal to the product of velocity and cross-sectional area, the narrowing of the tube causes an increase in velocity in that section. Even though the blood in the narrow section has a lower potential energy (pressure) than the downstream section, the kinetic energy (velocity) has been increased. Thus, the combined potential and kinetic energies are greater in the narrow section than the downstream section, and flow is maintained. Comprehension of the relationship between pressure gradients (potential energy) and velocity (kinetic energy) and their roles in the generation of turbulent blood flow are crucial to understand the translation of basic cardiovascular physiology to murmurs.

**Maneuvers**

*The Valsalva maneuver.* The Valsalva maneuver is achieved by closing of the glottis and attempting to forcibly exhale. Since air cannot escape through the closed glottis and the thoracic and abdominal muscles are contracting, an acute increase in intrathoracic and abdominal pressures ensues. Since the venous circulation is a “low-pressure system,” the Valsalva maneuver interferes with venous return to the right side of the heart. Due to the decrease in right ventricular preload, left ventricular end-diastolic volume will diminish. Echocardiographic experiments (11) have documented decreases in left ventricular chamber dimensions and stroke volume during the Valsalva maneuver. In addition, intravascular ultrasound has demonstrated that the increase in intrathoracic pressure results in a decrease in cross-sectional area and flow volume in the superior vena cava, leading to a decrease in preload to the right side of the heart (3). These changes in volume and chamber dimensions have a direct effect on physical exam findings of left-sided systolic murmurs.

A reduction in left ventricular end-diastolic volume induced by the Valsalva maneuver results in a diminished pressure gradient between the ventricle and aorta. Therefore, according to Ohm’s law of hydrodynamics, flow will decrease as well. According to Bernoulli’s principle, a decrease in flow will result in a reduced ejection velocity because the cross-sectional area of the aortic valve has not changed. On the account of decreased ejection velocity, the Reynold’s number and propensity for turbulence will be less. It can therefore be predicted that less turbulence will lead to a decline in the murmur.

*Squatting.* A sudden postural change from standing to squatting results in brisk changes in left ventricular chamber size, cardiac output, heart rate, the blood pressure response, and peripheral vascular resistance. Upon squatting, the compression of the veins in the lower extremities augments venous return to the right atrium. In contrast to the Valsalva maneuver, which leads to a smaller preload, squatting increases end-diastolic volume due to increased venous return. It has been demonstrated that there is also an increase in left ventricular cavity dimension, stroke volume, and arterial pressure and a decrease in heart rate and total peripheral resistance due to baroreceptor reflexes (5, 6). Therefore, it can be predicted that squatting would have the opposite effect of the Valsalva maneuver. The increased preload and ejection velocity would tend to increase the Reynold’s number, the possibility of turbulence, and the intensity of the murmur. It follows that returning to a standing position will decrease venous return, left ventricular chamber size, and stroke volume.

*Hand grip.* Isometric exercise will result in minimal elevation of the heart rate with a significant increase of systolic and diastolic blood pressures leading to an increase in left ventricular afterload (9). The increased afterload attenuates the pressure gradient across the aortic valve, which leads to decreased stroke volume and ejection velocity. Again, decreases in ejection velocity would tend to decrease the Reynold’s number and the potential for turbulence. Therefore, increases in afterload can delay left ventricular emptying and thus have a significant effect on systolic ejection and regurgitant murmurs.

**Systolic Murmurs**

We shall concentrate on the most common and clinically significant encountered murmurs in the adult patient. We will limit our discussion to aortic stenosis (AS), mitral regurgitation (MR), hypertrophic obstructive cardiomyopathy (HOCM), and mitral valve prolapse (MVP). For the sake of discussion regarding the effect of maneuvers on auscultatory findings in systolic murmurs, it is best to concentrate on the anatomic abnormalities that are responsible for the production of the abnormal sounds. It is believed that murmurs are produced by obstruction to laminar flow by abnormal anatomic changes, thus creating vortexes (tiny eddies) (2). The intensity of the disruption of laminar flow over these anatomic changes is partially related to the velocity of flow. The intensity of sound becomes more pronounced as the velocity increases due to an increase in the production of vortexes.

Systolic murmurs are divided into ejection systolic murmurs, AS and HOCM, and regurgitant murmurs, MR and MVP. We shall begin our discussion with the systolic ejection murmurs of AS. We will discuss HOCM and MVP together due to their similarity in behavior with cardiac maneuvers.
AS. AS is characterized by a narrowing in the diameter of the left ventricular outflow tract. As described previously regarding Bernoulli’s principle, the reduction in the cross-sectional area of the valve causes augmented jet velocity, increased Reynold’s number, and a systolic ejection murmur (4). The typical murmur of AS is a crescendo-decrescendo murmur loudest at the base radiating to the carotids. It is helpful to closely listen to the second heart sound (S2), since in severe AS the aortic component is absent and thus a physiologically split S2 is a reliable sign of the absence of severe disease (4). In addition, the timing of the carotid pulse compared with S2 can be helpful in making the differentiation between aortic sclerosis (calcium deposition on the valve cusp without significant stenosis) and AS. With increasing obstruction, the carotid pulse will peak closer to S2 since it will take longer for left ventricular emptying to occur. Also, a weaker pulse would be present due to the decreased peak aortic pressure during systole (Fig. 1A). The finding of both a weak and delayed pulse is termed “pulsus parvus et tardus,” a very specific sign of severe AS (10). When we think of the effect of cardiac maneuvers on systolic ejection murmurs, it is useful to think of changes in stroke volume with regard to flow-velocity relationships and the changes expected in the auscultatory findings.

The effect of the Valsalva maneuver on the intensity of AS is shown in Fig. 1B. When the Valsalva maneuver is performed, as previously explained, there is decreased venous return to the right side of the heart, leading to a decrease in left ventricular end-diastolic volume. Since there is less volume in the left ventricle to be ejected through the narrowed aortic valve (decreased transvalvular flow), there will be less turbulence and thus a decrease in intensity of the murmur.

With squatting, due to the augmentation of venous return from the lower extremities, and the resultant bradycardia, there is an increase in stroke volume (Fig. 1C). The increased volume ejected from the left ventricle will lead to an increase in the intensity of the murmur. Conversely, standing from a squatting position will lead to the pooling of venous blood in the lower extremities with the reverse effect on stroke volume and resultant reduction in the intensity of the murmur.

During hand grip, as discussed above, there is an increase in left ventricular afterload, which causes an increase in the impedance to left ventricular emptying. Since flow is dependent in part on the pressure gradient across the stenosis, it follows that by increasing afterload, the difference in intraventricular pressure to aortic pressure will diminish. The decrease in pressure gradient will cause less blood flow (velocity and quantity) across the valve per unit time, resulting in a decrease of the murmur of AS.

MR. In MR, the defect is in lack of coaptation (approximation) of the mitral valve leaflets during systole, resulting in retrograde flow of part of the stroke volume into the left atrium. Since flow in a vessel is most influenced by the radius of the vessel, one can assume that the decrease in coaptation of the mitral valve leaflets will lead to a greater radius of the incompetent area, facilitating regurgitation. MR is usually noted as a blowing holosystolic murmur loudest at the apex with radiation to the axillary region (Fig. 2A).

If we consider the Valsalva maneuver, high intrathoracic pressures will decrease right ventricular preload, resulting in a smaller left ventricular end-diastolic volume and pressure (Fig. 2B). The reduction in preload will result in diminished ven-
Fig. 2. Effect of procedures on mitral regurgitation. A: mitral regurgitation (baseline). B: mitral regurgitation + Valsalva maneuver. C: mitral regurgitation + squatting.
leaflet. Hand grip will increase left ventricular afterload and decrease left ventricular emptying, thus inducing a relative increase in left ventricular volume during systole. The effect on the murmur of HOCM is that of diminished intensity (10).

MVP. MVP is recognized by auscultation due to its characteristic midsystolic click followed by a late systolic murmur. Unfortunately, these findings can be present intermittently, making the clinical diagnosis at times difficult. It is not unusual to hear a normal cardiac exam, a click only, the murmur only, or a click and murmur in the same individual at different times. The pathology of MVP is due to myxomatous degeneration of the mitral valve leaflets, leading to a redundancy of tissue. This redundancy results in the prolapse of one or both of the leaflets of the mitral valve into the left atrium during systole. The redundant tissue disrupts proper coaptation of the leaflets, facilitating valvular insufficiency, leading to a late mitral regurgitant murmur. It is believed that the click heard before the murmur is due to sudden tensing of the chordae tendineae and/or valve leaflets (7). It is important to realize that the anatomic aberrancy will be exacerbated by decreasing left ventricular end-diastolic volume. The critical volume at which prolapse begins is when the contracting ventricle reaches 75 ml (2). With continued systole, the coaptation of the leaflets then fails, and regurgitation occurs. Decreasing left ventricular end-diastolic volume will facilitate reaching the critical volume earlier during systole, thus precipitating the click and murmur to occur closer to the first heart sound. For this reason, maneuvers that decrease left ventricular volume (e.g., Valsalva or standing) will cause the click and murmur to occur earlier in systole and become more prominent. Likewise, any maneuver that increases left ventricular volume (squatting or leg raise) will result in reaching the critical volume (75 ml) later in systole, leading to a delay in the appearance of the click and murmur.

To summarize, murmurs that decrease in intensity with the Valsalva maneuver, MR and AS, are best distinguished by the use of isometric exercise (hand grip). Murmurs that increase in intensity with the Valsalva maneuver, MVP and HOCM, are best distinguished by the character of the murmur, location, and the presence or absence of a click. The effect of maneuvers on the timing and intensity of the features of MVP are of additional assistance as well as the decrease in intensity of the murmur of HOCM with hand grip.

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS
Author contributions: S.A.S., J.L.B., and D.M.H. analyzed data; S.A.S. drafted manuscript; S.A.S., J.L.B., and D.M.H. edited and revised manuscript; S.A.S., J.L.B., and D.M.H. approved final version of manuscript; J.L.B. prepared figures.

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