The Systolic Murmur—Benign or Serious?

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Introduction

Systolic murmurs are commonly detected during cardiac auscultation. As suggested by the title, they are often benign, but can indicate serious or life-threatening cardiac conditions or the need for further evaluation or timely intervention.

All systolic murmurs result from disturbed or “turbulent” flow ultimately resulting from the pumping action of a ventricle, while all diastolic murmurs occur from ventricular inflow during ventricular relaxation. Systolic murmurs can be of outflow origin, either from increased stroke volume (as with anemia, pregnancy, or ventricular overfilling from aortic valve regurgitation) or flow through a narrowed or distorted outflow channel or conduit. Less often, systolic murmurs result from backflow either through an atrioventricular valve (mitral or tricuspid) or through an intracardiac communication, e.g., ventricular septal defect.

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Is the murmur systolic?

An approach to the evaluation of a systolic murmur should begin by ascertaining that the murmur is unequivocally systolic. It is not uncommon to be asked to evaluate a patient purported to have a systolic murmur only to find that the murmur is not systolic. This confusion in timing is prompted by the widespread but erroneous assumption that any readily heard murmur must be systolic. Three techniques can be very helpful in determining that a murmur is systolic, and it is prudent to employ as many these timing aids as possible in order to ensure that a murmur is indeed systolic

A. Correlation with visible or palpable central pulses

B. Cadence

C. Post-PVC augmentation

Correlation with visible or palpable pulses can be challenging if the heart rate is rapid, or if a prominent jugular venous pulsation (JVP) emulates a carotid pulse, so it is important to palpate as well as observe the respiratory effects on any visible pulse in the neck. Carotid pulsations, unlike JVPs are minimally affected by respiration. The later arrival and altered waveform of the radial pulses render them less reliable than the carotid pulse for timing. Palpable precordial pulsations are usually systolic and are therefore reliable timing aids.
The cadence of a murmur can be used to establish its timing within the cardiac cycle. If the heart rate is less than 90, systole will be shorter in duration than diastole, so that the first sound (S1) is closer to the following second sound (S2), than the interval between S2 and the following S1. As a result, systolic murmurs are usually shorter than diastolic murmurs. If the rhythm is irregular, as with atrial fibrillation, the duration of systole varies little, while the duration of diastole and diastolic murmurs is variable.

Post – PVC (Postextrasystolic) augmentation of a murmur is a reliable indicator that a murmur is not only systolic in timing, but probably of outflow tract origin. Detection of this
phenomenon requires comparison of the murmur intensity of the beat following the “dropped beat” with the murmur heard with the subsequent beats. Postextrasystolic augmentation of ventricular contractility, especially if the ventricle is hypertrophic, combined with the fall in arterial pressure caused by increased runoff duration will usually cause more outflow velocity and a louder, longer murmur.

**Continuous murmurs** are frequently confused with murmurs confined to systole. Their true identity can usually be established by moving ("inching") the stethoscope to an area at which the murmur as well as the heart sounds can be heard, and noting the murmur continues through and well beyond S2.

**Location, location, location**

There are three parameters regarding location that are helpful in establishing the possible cause(s) of a systolic murmur:

A. Location on precordium of loudest murmur

B. Location(s) of radiation or distribution

C. Location within systole
The precordial location of the loudest transmission of a cardiac murmur is of help in determining its origin. Radiation of murmurs from their primary location occurs through downstream propagation in outflow murmurs as well as through myocardial transmission.

Outflow murmurs (AKA “ejection murmurs”) are usually loudest over the precordial locations closest to the outflow tract or great artery through which the flow is occurring -- pulmonary outflow murmurs in the second left intercostal space (2 LICS) and aortic in the 2 RICS, and radiate along the course of the great vessels. It should be noted that the pulmonic valve is close to the chest wall in 2LICS while the aortic valve is buried deep within the heart behind the right ventricular outflow tract in the 3-4 LICS. The aorta ascends rightward and ventrally, approaching the chest wall at 2 RICS where aortic outflow murmurs are generally heard best.
Vibrations from murmurs are radiated well through rigid (contracting) myocardium, much as a cast iron pipe conducts sounds over great distances while a rubber hose dampens sonic vibrations. The rigidity of contracting myocardium enables outflow murmurs to be well heard over the ventricle of origin, and may filter out some of the harsher components. A case in point is the “Gallavardin transformation” of the outflow murmur in aortic stenosis, a pure musical murmur radiated toward the apex while a rough or harsh murmur is heard over 2 RICS and the carotid arteries downstream.

**Regurgitant Murmurs** emanating from the tricuspid or mitral valve are usually heard best over the ventricle of origin, probably because of the rigidity of ventricular myocardium and the relative laxity of the atria. The left parasternal location of the right ventricle (RV) makes the murmur of tricuspid regurgitation (TR) well heard along the left sternal border. The murmur of mitral regurgitation (MR) is best heard where the LV is closest to the chest wall, at the apex and left axilla. High velocity jets of MR resulting from a flail posterior leaflet may be directed toward the common atrio-aortic wall and thus emulate an outflow murmur. Flail anterior leaflet MR may be heard over the dorsal spine and occiput.

The murmur caused by a restrictive ventricular septal defect (VSD) is sometimes classified as “regurgitant” in that it results from flow counter to LV outflow, increases LV diastolic volume, and has similar timing. VSD murmurs are best heard over the RV, radiate widely, and often associated with a systolic thrill over 3-4LICS.
Location within Systole is dictated by the hemodynamic relationships between the pumping and recipient chambers. If systole is defined as the interval between the first (S-1) and second (S2) heart sounds, a murmur that occupies all of systole is termed holosystolic. A murmur that occupies the mid portion of the space between S1 and S2 is called mid systolic. An early systolic murmur is initiated by S1 and ends well before S2, while a late systolic murmur begins well after S1 and blends into or continues through S2. The above terminology avoids potentially misleading or inappropriate terminology such as “ejection murmur” and “pansystolic;” the former implies established certainty that the murmur is of outflow origin (which is not always possible by bedside examination) and the latter literally means “all of the systoles” rather than all of systole.

A holosystolic murmur (HSM) results from high velocity blood flow from a ventricle into a recipient chamber (usually an atrium) from the onset of ventricular contraction until ventricular pressure falls as the ventricle relaxes. Only three conditions can cause a HSM: TR, MR, and restrictive (small) VSDs. However, not all TR, MR, or VSD murmurs are holosystolic. Acute MR may cause a shortened murmur if there is marked elevation of left atrial systolic pressure (v-waves) that stifles late systolic backflow. It is erroneously assumed that HSMs will always obscure the first and second sounds, and conversely if heart sounds are heard, the murmur is not holosystolic.
Mid systolic murmurs are generated by disturbed or turbulent outflow, and often termed “ejection murmurs,” but as stated above, the timing and not the presumed cause of the murmur should be applied to these murmurs. Mid systolic murmurs (MSMs) are characteristic of aortic stenosis (AS), pulmonic stenosis (PS), or a high ventricular stroke volume in aortic regurgitation (AR) or atrial septal defect (ASD). MSMs are very common and frequently benign, since they may result from high cardiac output states in normal adolescents or athletes, pregnancy, anemia, or chest wall configuration (pectus excavatum, straight back syndrome, asthenia, etc.). Murmur intensity is directly related to velocity and inversely related to blood viscosity, hence conditions that increase velocity (normal flow through a stenotic orifice or high flow through a normal channel) may produce a murmur.
Mitral regurgitation may produce a crescendo-decrescendo murmur that is heard in the aortic area (2RICS) and can be confused with a murmur of outflow origin, but the holosystolic or late systolic (see below) timing is not compatible with a murmur produced by outflow. It should be remembered that outflow cannot occupy all of systole (holosystolic) because aortic or pulmonic valve closure occurs after cessation of outflow and initiation of backflow, obligating a pause between the end of the murmur and the second sound.

**Figure 5. Mid systolic murmurs** caused by ejection through stenotic semilunar valves have a crescendo-decrescendo configuration and end before the closure sound from the valve. Closure of the aortic or pulmonic valves require retrograde flow in the artery beyond the valve, meaning that the forward flow causing the murmur most terminate and reverse, thus ending the murmur prior to the closure sound. In the depiction of **Pulmonic Stenosis**, the dashed line indicates the closure of the pulmonic valve and P2.
Late systolic timing of a murmur usually implies mitral valve prolapse (MVP) in which the mitral valve is competent in early systole but its leaflets lose apposition and become incompetent in mid and late systole. The acoustic contour of these murmurs may be crescendo-decrescendo, and these murmurs may spill through the time of aortic valve closure and obscure the second sound. Late systolic murmurs in MVP often have a whooping or honking acoustic profile, and may be sufficiently loud to be heard without a stethoscope.

**Figure 6.** This patient with Mitral Regurgitation had a holosystolic murmur well heard in the Aortic area that had mid-peaking amplitude, and was recorded from within the aorta by intracardiac phonocardiography. Pressure recording revealed tall v-waves in the left atrium (LA) and no hemodynamic evidence of aortic stenosis. Atrial fibrillation was present. The mitral regurgitant jet impinged on the left atrial wall adjacent to the aorta, causing the murmur to be transmitted to the aorta.
In Hypertrophic Cardiomyopathy (HCM) Mitral valve dysfunction resembling MVP may result from the dynamic ventricular cavitary obliteration; the mitral valve may be competent in early systole but becomes scrunched and rendered incompetent in mid and late systole. In both of these clinical settings (MVP and HCM) the timing of the murmur may be similarly altered by maneuvers that affect the left ventricular diastolic volume (e.g. standing and
squatting, Valsalva strain and release). Maneuvers that increase LV volume (squatting) will
delay murmur onset, while standing or Valsalva strain will decrease LV volume and increase
the length and intensity of the murmur.

A murmur generated by aortic coarctation, or with increased internal mammary arterial flow in
pregnancy (mammary souffle) or lactation may have late systolic timing because the peak
flow rate occurs later in arteries at a distance from the ventricle.

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**Figure 8. Severe Mitral Regurgitation** with late systolic diminution of the systolic
murmur because the “ventriculoid” left atrial (LA) pressure approaches the left
ventricular (LV) pressure in late systole. The vertical arrows denote the relationship
between S2 and the aortic (Ao) dicrotic notch. There is an S3 associated with the
rapid filling wave (RFW) in the left ventricle.

**Early systolic murmurs** may be generated by TR or MR that is suppressed in late systole.

In a dilated ventricle, the valve leaflets may not be able to meet at the onset of systole but
may appose as the ventricle and annular diameter decrease. As noted above, acute, severe MR may cause the systolic pressure to rise in the left atrium (LA) and rival LV pressure, and thus suppress late systolic regurgitant flow. Small muscular VSDs may close when the septal muscle contracts, terminating the murmur in late systole. The murmur of a muscular ventricular septal defect may disappear or markedly diminish upon standing.

**Sounds Associated with Murmurs**

Although often overlooked (or more appropriately, not even listened for) the sounds that accompany murmurs are often of inestimable value in determining the cause and/or significance of a murmur, especially if the murmur is mid systolic. **Heart sounds** are produced by the abrupt deceleration by impact of a moving column of blood in the cardiovascular system. In a normal heart, **S1** is caused by the abrupt halting of blood pushed toward the atrium at the onset of ventricular contraction when this retrograde surge is abruptly halted by the expanding underbellies of the atroventricular valve leaflets. **S2** results from the abrupt halting of retrograde flow in the aorta or pulmonary artery root when the ventricle relaxes and the semilunar valve closes.
When a holosystolic murmur of tricuspid regurgitation is associated with an exceptionally loud S1 in a patient with pulmonary hypertension, unrecognized mitral stenosis (MS) may be the underlying cause of the pulmonary hypertension. A soft or absent S1 may indicate “preclosure” of the mitral valve or an under-powered ventricular contraction in a failing ventricle. Apparent splitting of S1 caused by an ejection sound (vide infra) can be a clue to significant outflow tract pathology. Ebstein’s anomaly of the tricuspid valve can cause a

Figure 9. Events responsible for the first and second heart sounds and the directions of primary radiation of the sounds are indicated by cartoons with shaded densities representing the pressure in the various chambers. Darkest densities indicate pressures above 80, while the lightest represent less than 10 mmHg.
loud early systolic sound ("sail sound") generated by late closure of the deformed tricuspid valve.

Intensity and patterns of splitting of S2 can also provide diagnostic information. **Loud S2** implies high closing pressure (systemic or pulmonary hypertension), while appreciation of abnormal splitting can narrow the differential diagnostic possibilities raised by systolic murmurs. **Physiological splitting** of S2 occurs when the duration of right ventricular systole is increased by augmented right heart inflow, combined with RV outflow into the expanding lungs that literally vacuum blood out of the right heart with negative pressure, both contributing to delayed pulmonic valve closure. **Persistent splitting** of S2 can result from prolongation right ventricular systolic duration with atrial septal defect (ASD) or pulmonic stenosis (PS). Persistent splitting occurs when late onset (and end) of right ventricular systole with right bundle branch block (RBBB), or early closure of the aortic valve when there is an alternate low impedance outlet such as mitral regurgitation (MR) or ventricular septal defect (VSD). On the other hand, prolongation of, or late onset of, LV systole results in **paradoxical splitting** in which the aortic component of S2, or A2 occurs after the pulmonic component, P2 during expiration so that the normal inspiratory prolongation of right ventricular systole closes the P2-A2 gap. If the onset of left ventricular systole is delayed by left bundle branch block, or the duration of left ventricular systole is prolonged by aortic stenosis or hypertrophic cardiomyopathy, A2 will follow P2 during expiration or apnea, and the split will close as P2 is delayed during inspiration.
**Extra diastolic sounds**, in chronological order of appearance are termed opening snaps (OS), third sounds (S3) and fourth sounds (S4). Stenotic mitral valves usually retain sufficient leaflet flexibility to undergo a partial opening excursion at the base of the valve while parting of the leaflets at the distal end is abruptly halted by commissural fusion, causing the blood moving into the valve sleeve to be prevented from flowing freely into the left ventricle. Blood moving down the mitral sleeve encounters the inability of the funnel-like terminus to open, with the impact generating a sharp early diastolic OS. The timing of the S2-OS interval roughly matches the two syllables in the word “butter.”

An S3 is caused by a ventricle resisting inflow as a result of an absolute or a relative increase in passive ventricular volume. Torrential inflow may occur in high output states or when the atrium has received a large volume of regurgitant flow in the preceding systole. Conversely, a small diastolic input may enter a failing ventricle that has not expelled enough blood during systole to make room for a normal diastolic inflow, abruptly achieving maximal capacity. A caricature of S3 called a **pericardial knock** when the ventricles are incased in a fibrotic or calcified girdle in pericardial constriction.

S4 is generated by a ventricle resisting inflow from augmented atrial contractions, and are typically heard in conditions in which there is hypertrophy of either the right or left ventricle.

**Systolic sounds** causing “splitting” of the first heart sound are called *ejection sounds* (AKA ejection clicks) that are generated at the time of opening of the aortic or pulmonic valve. An ejection sound (ES) may result from abrupt checking of the opening excursion of a
congenitally stenotic aortic or pulmonic valve when the leaflets are mobile but fail to open properly. As blood in the cylindrical outflow tract gains momentum and the semilunar valve initiates an opening excursion, the inability of the stenotic valve to open fully abruptly halts the column of blood, which is then forced through the restricted orifice. Thus an ES can be heard to initiate the mid systolic murmur (MSM) of aortic stenosis (AS) or pulmonic stenosis (PS) when the valve leaflets retain mobility. When fibrosis or calcification render the valve rigid and immobile an ES is not heard. An ES is usually discerned more readily on the upstream (ventricular) side of the diseased semilunar valve. Since both the ES and S1 are louder at the apex than at the base, recognition that the first sound is split can be appreciated better at the apex. An ES can be heard well at the base, where it seems to “jump start” the murmur.

Ejection sounds do not necessarily indicate the presence of valvar stenosis, because they are also heard when the aorta or pulmonary artery are dilated and hypertensive, and/or subjected to high output states when the semilunar valve leaflets are flung open to initiate ejection. The following conditions are frequently associated with an ES: atrial septal defect (ASD), pulmonary or systemic hypertension, aortic regurgitation, anemia, and chronic renal failure with arterio-venous shunt dialysis access.

**Mobile systolic clicks (MSC),** also called mid systolic clicks or non-ejection clicks, are virtually diagnostic of mitral valve prolapse (MVP). The floppy, redundant mitral leaflets inflated by LV systolic pressure billow progressively back into the left atrium, and are eventually checked by chordal restraint. This abrupt halting of LV pressure’s inflation of the
leaves generates a clicking or popping MSC. If the mitral valve is rendered incompetent by loss of leaflet apposition in MVP, the MSC is followed by a late systolic murmur (LSM) that may spill through S2. Not all MVP valves are incompetent, so that there may be no murmur in association with the MSC, and alternatively there may be no MSC in association with the murmur of MVP.

The mobility of a MSC can be demonstrated by postural maneuvers or other perturbations that affect ventriculo-valvar proportionality. The valve is demonstrably “too big” for the LV, and as a result cannot be maintained in a competent sub-annular position when the ventricular cavity size is reduced during systolic ejection. The misfit between the valve and LV can be further perturbed by maneuvers that reduce ventricular size (abruptly standing up from supine or squatting position, Valsalva strain, vasodilators, exercise), or ameliorated by abruptly increasing ventricular size (squatting after standing). As the MSC moves closer to S1 the murmur may become longer and louder, and when it moves closer to S2 the murmur starts later and is often softer.

**Visible or Palpable Pulsations**

Correlation of auscultation with visible or palpable pulsations is an important aspect of cardiac bedside examination. Pulsations not only provide timing signals, but frequently have the added value of providing independent diagnostic information. Although the left ventricular apical impulse is often obscured by obesity, large breast, or barrel chest configuration, when visible and/or palpable it provides precise timing of auscultatory events and a window into LV function. Recordings of the apical pulse, called apexcardiograms
(ACGs) were utilized by clinical investigators in the 1960s to detect exercise induced ischemic changes in ventricular function. The apex impulse, as graphically recorded by an ACG provides an outward thrust during isovolumic left ventricular contraction, the nadir (O Point) at the time of mitral valve opening, the rapid filling wave and “atrial kick” by the ventricle, all with little or no time delay. Pathological apical impulse patterns would include a sustained systolic outward thrust in aortic stenosis or with LV aneurysm, a presystolic impulse in left ventricular hypertrophy, a rapid filling wave in volume overload lesions (MR, AR, dilated cardiomyopathy). A “triple ripple” impulse (atrial kick, early and late systolic outward thrusts) is diagnostic of hypertrophic cardiomyopathy (HCM). A late systolic inward collapse is often found in MVP when the aneurysmal valve pops into the atrium causing a sudden tension release in the LV.

Most left parasternal systolic impulses are caused by the RV, although a rapidly expanding LA from mitral regurgitation (MR) may cause a late systolic heave by pushing the RV from behind. Left parasternal diastolic impulses can arise from RV diastolic overload from tricuspid regurgitation, or when being pushed from behind by a rapidly expanding left ventricle in AR. Hypertensive pulmonary arterial expansion may cause a parasternal impulse in the 2nd or 3rd left intercostal space and a palpable P2.

Figure 10 Carotid arterial pulse contours may be diagnostic of specific causes of systolic murmurs, especially the parvus et tardus pulse associated with aortic stenosis, the bisferiens (double-peaked) pulse of aortic regurgitation, and the spike-and-dome pulse of hypertrophic cardiomyopathy. The systolic ejection period is prolonged in hypertension, aortic stenosis, and hypertrophic cardiomyopathy.
Visible and palpable carotid pulsations provide much better timing of auscultatory events than the radial pulse, particularly at fast heart rates. At rates of 100 or more, the radial systolic pulse will be felt during ventricular diastole. In addition to the value of the carotid pulse for timing of heart sounds, there are three “classic” diagnostic carotid pulse contours: (1) parvus et tardus of AS, bisferiens (double peaked) of AR, and “spike-and-dome” of HCM. It is important to note that all three of these conditions are commonly associated with a similar systolic murmur, but can usually be differentiated from one another by the associated carotid pulse contour. Corrigan’s pulse, bounding and collapsing pulses of the major subcutaneous arteries, and Quincke’s pulse, elicited by gentle pressure on the digital nailbeds to produce systolic blushing and diastolic blanching are also virtually diagnostic of AR.

The jugular venous pulse (JVP) contains valuable diagnostic information that can be gleaned by correlation with the cardiac auscultation. It should be said that the Internal JVP emanates from the same area of the neck as the carotid pulse, and this juxtaposition may lead to considerable confusion when the source of the pulsation is not carefully determined. The JVP will usually vary more with respiration than the carotid and unlike the carotid often has 2 widely spaced pulsations per cardiac cycle when sinus rhythm is present. The JVP pressure declines (x- and y-descents) are more abrupt and therefore more readily seen than the diastolic decline in carotid arterial pressure. The external JVP is several centimeters lateral to carotid arterial pulsations, and will usually faithfully transmit the right atrial pressure contours to the neck, avoiding confusion between arterial and venous pulsations in the
suprasternal notch or emanating from the common carotid/jugular sheath medial to the sternocleidomastoid muscle.

The contour of the JVP carries considerable diagnostic information. Large a-waves, almost coincident with the first heart sound and declining rapidly during systole, indicate RV hypertrophy, while v-waves, reaching a peak coincident with the second sound indicate tricuspid regurgitation (TR).

**Maneuvers and Perturbations**

Some bedside maneuvers are difficult to perform by elderly or infirm patients, but can be very helpful in more agile patients when heart sounds and murmurs raise several diagnostic considerations. However the effects of spontaneous or controlled breathing are almost always possible to observe and can provide important diagnostic information. It is often useful to use your free hand (the one not holding the stethoscope) to “conduct” the patient’s breathing pattern by first rehearsing the maneuver with the patient. Have the patient inspire slowly and quietly by raising your hand palm-up, exhale while lowering your hand palm-down, and suspend respiration after full exhalation by sweeping your pronated hand back and forth for a few seconds. The transient hemodynamic effects of inspiration can only be noted during active inspiration, not by having the patient take and hold a deep breath! If you tell a patient “hold your breath” the person will almost invariably take a deep breath and bear down, involuntarily performing a partial Valsalva strain. When the noises associated with breathing overwhelm the heart sounds, tell the patient to “stop breathing” (as opposed to “hold your breath”), preferably at end expiration. Two or three seconds of apnea can be
tolerated by virtually any patient, regardless of respiratory distress, and will permit clear
listening to several heart beats.

A more dramatic negative intrathoracic pressure pull can be affected by a Mueller maneuver
consisting of having the patient attempt to suck on an imaginary pickle or soda straw with the
mouth closed, causing the cheeks to collapse inward. Alternatively, the patient can be told to
breathe in and out through the nose several times and then compressing the nares for 2-3
seconds while listening to the heart during attempted inspiration. There should be a loud
sucking sound upon release of nasal compression if the patient has successfully maintained
negative intrathoracic pressure. Auscultation during the Mueller maneuver will bring out
subtle murmurs from tricuspid regurgitation (TR) that might otherwise be obscured by the
lung sounds.

**Post-PVC Augmentation** of a systolic murmur is described above, and when present, is
usually indicative of origin in the ventricular outflow tract. Most patients with aortic or
pulmonic stenosis, whether the lesion is valvar, subvalvar, or supravalvar in location, will
have postextrasystolic augmentation of murmur intensity. As previously stated, the
increased inotropic state in the beat following a PVC will generate a more forceful contraction
in the ventricle(s) and will increase the flow velocity causing the murmur. One “special case”
that doesn’t quite fit into this scheme is the murmur of hypertrophic cardiomyopathy
(HCM). Although the murmur is often described as an “outflow murmur” it is very often found
to be holosystolic or late systolic in timing, and thus may be caused by mitral regurgitation.
The augmentation of the murmur is illustrated in Figure 11, and the murmur is seen to begin with the first sound and extend to the second sound, and therefore holosystolic.

Auscultation during Valsalva strain and release is an important bedside maneuver that should be performed correctly to avoid spurious results. The patient should be rehearsed while being told to breathe in deeply, then out, and then to bear down during peak expiration for at least 10-15 seconds, and then relax and resume normal breathing. The examiner’s hand can be placed on the abdomen to counter the outward thrust of the abdomen and determine if the patient is actually bearing down. The Valsalva strain increases intra-

Figure 11. The post-PVC murmur of Hypertrophic Cardiomyopathy (HCM) is often holosystolic, as shown here, and therefore generated by mitral regurgitation. The brisk initial upstroke of the aortic pressure should be contrasted with the delayed aortic upstroke in Aortic Stenosis (Figures 2 and 5). The left ventricular end diastolic pressure (small arrow) is elevated, and there is a fourth heart sound (4).
abdominal and intrathoracic pressure and inhibits venous return to the heart, causing the heart chambers to shrink progressively and the cardiac output to decline. With release, the pent-up systemic venous blood rapidly returns, first to the right heart and followed 3-4 heart beats later by the left heart. It is important to have the stethoscope on the chest during the entire maneuver, especially after release.

Most murmurs will decline in intensity during the Valsalva strain, the heart rate will usually accelerate and a vasopressor response will ensue as the carotid sinus baroreceptors sense a decline in pressure. Upon release of the strain there will be a return right heart murmurs first, followed by left heart murmurs as the chambers progressively and sequentially fill. An early return suggests that the murmur is from the right heart, and if the murmur intensity returns after 3-4 heart beats, the murmur is probably of left heart origin. The vasopressor response during strain may continue into the recovery period, and in turn lead to transient hypertension and bradycardia.

Figure 12. A patient with HCM performs a Valsalva strain during left ventricular and aortic pressure and apical phonocardiographic recording. The systolic murmur intensity increases but does not consistently track with the magnitude of the pressure outflow tract gradient (shaded area). The murmur had musical harmonics that can be seen best in the mid portion of the record.
There are two conditions in which murmur intensity may actually increase during Valsalva strain: Mitral valve prolapse (MVP) and hypertrophic cardiomyopathy (HCM), because the shrinking of the LV exaggerates the disproportionality between the mitral valve and ventricle mentioned above. As the ventricular diastolic volume shrinks, systolic contractions distort the valve, causing the systolic murmur to progressively increase in intensity, and often in duration and pitch as well. Amyloid of the heart may emulate HCM, and will often cause an increase in murmur intensity with Valsalva strain.

**Postural maneuvers** can alter the filling and emptying of the ventricles and triggers autonomic reflexes. Abrupt **standing** momentarily decreases venous return and may cause an exaggerated reflex tachycardia in some individuals with MVP. The resulting exaggeration of the disproportional fit of the mitral valve within the left ventricle will usually cause the systolic murmur to become louder, longer, and higher in pitch in both MVP and HCM. A mobile systolic click (MSC) will occur earlier, and then upon **squatting**, move later as the murmur is “squashed” into S2 in MVP. Abrupt **squatting** will pump a bolus of blood into the central circulation and at the same time increase the impedance to left ventricular ejection. This double effect benefits patients with Fallot’s tetralogy by promoting increased flow to the pulmonary arteries, and may transiently increase the intensity of right heart murmurs.

**Passive leg raising** will cause a sudden volume shift of blood from the periphery into the central circulation, but is not easily performed because it requires several people to achieve the maneuver, and the patient may inadvertently defeat the passivity of the maneuver by
straining. This maneuver may increase the intensity of right heart murmurs and decrease the murmur of hypertrophic cardiomyopathy.

**Sustained isometric handgrip**, especially if performed with both hands, increases impedance to left ventricular ejection and may augment left heart regurgitant murmurs and reduce the murmur of HCM.

Amyl nitrite, a rapid acting inhaled **Vasodilator** was traditionally readily available for bedside evaluation of murmurs, and when used had dramatic and profound effects in enhancing murmurs induced by forward flow (e.g., mitral and stenosis) and reducing the intensity of regurgitant murmurs. Although it is no longer available, it served to elucidate the pathophysiology underlying many murmurs prior to the era of echocardiography. Vasodilators are now widely used in the management of patients with regurgitant left heart valves, and although not as rapid-acting as amyl nitrite, will have a similar effect on the murmurs of mitral and aortic regurgitation.
Innocent Murmurs are those that are not associated with any demonstrable radiographic, electrocardiographic, or echocardiographic, abnormality, and are more common in children than in adults. In some instances, a murmur proclaimed to be “innocent” in childhood proves later to have been caused by previously unrecognized bicuspid aortic valve stenosis, ventricular septal defect, or mitral valve prolapse. These murmurs are thought to be of right heart origin, and usually increase in intensity with inspiration as a result, and often have a twanging “vibratory” quality.

Is a murmur benign or serious? It is overly simplistic to imply that a given murmur is always benign or always serious; in real life experience there are infinite shades of gray. In turns of probability of adverse outcome without timely, specific intervention, the least benign
conditions are aortic stenosis, hypertrophic cardiomyopathy, and tricuspid regurgitation (if it results from pulmonary hypertension). Intermediate risk would be asymptomatic mitral regurgitation, pulmonic stenosis and atrial septal defect. Low risk would include ventricular septal defect and most patients with mitral valve prolapse.

It is hoped that this exercise will serve to sharpen the astute physician’s bedside evaluation of patients with systolic murmurs by calling attention to the ancillary visible, palpable, and auscultatory events that accompany the murmur. Time and space limitations do not permit any discussion of appropriate diagnostic procedures or therapy.
Suggested Reading

