

Cardiac Physical Examination: Clinical “Pearls” and Application

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The cardiac physical examination, which includes both auscultatory and nonauscultatory components, when performed by experienced examiners, is a sensitive, specific and cost-effective method of detecting cardiac disease (1–6). By perfecting and maintaining basic bedside clinical skills, the physician can gain enough confidence in these skills so as to decrease reliance on noninvasive testing and instead utilize additional testing selectively. This review emphasizes selected practical clinical applications of the cardiac physical examination.

Palpation of Precordium

The patient should be examined in both the supine and the left lateral decubitus positions. The normal apical impulse occurs during early systole, with an outward motion imparted to the chest wall. During mid- and late systole, the left ventricle (LV) is diminishing in volume, and the apical impulse moves away from the chest wall (4). Thus, outward precordial apical motion occurring in late systole is abnormal.

The apical impulse of LV enlargement is usually widened or diffuse and displaced leftward. A subtle ventricular rapid filling wave (palpable S_3) may be better visualized than palpated by observing the motion of the stethoscope on the chest wall during simultaneous auscultation. The apical systolic impulse of LV hypertrophy without dilatation is sustained and localized. It may be forceful but should not be displaced. It is often accompanied by a palpable presystolic outward movement, referred to as the *a* wave or *palpable* S_4 . If the apical impulse is not palpable and the patient is hemodynamically unstable, consider cardiac tamponade as a possible diagnosis. In general, pulsations of increased blood flow are dynamic and quick, whereas pulsations related to high-pressure states cause a sustained impulse (1,4).

Precordial motion in the lower sternal area, especially along the left parasternal region, usually reflects right ventricular (RV) activity. RV motion due to systolic overload causes a sustained outward lift. RV diastolic overload (as in atrial septal defect [ASD]) causes a vigorous motion but may not be sustained. When the left-to-right shunt associated with ASD exceeds 2:1, a parasternal lift is usually present. With severe mitral regurgitation, when the left atrium expands in systole but is limited in its posterior motion by the spine, the RV may be displaced forward, and with it, the parasternal region can be lifted indirectly, the *contrecoup* effect; this motion may simulate an RV lift. Significant over-

lap of precordial sites of maximal pulsation occurs in LV and RV overload states.

Jugular Veins

The jugular veins are direct conduits to the right atrium and reflect the hemodynamics of the right side of the heart. Jugular veins that fill with inspiration (Kussmaul's sign) are a clue to constrictive pericarditis, pulmonary embolism, or RV infarction. The hepatojugular and Valsalva's maneuver can be useful for eliciting venous pulsations if they are difficult to visualize. A positive hepatojugular (abdominojugular) reflux sign may be found in LV failure. If the jugular veins are engorged but not pulsatile, consider superior vena caval obstruction.

Arterial Pulse

Carotid pulse

A normal carotid pulse should be easily palpated, whereas the carotid pulse of reduced volume is difficult to feel. Carotid palpation in obese patients with thick necks is an exception to this rule, and reduced carotid pulses in this setting are of less diagnostic value. *Pulsus parvus*, or reduced volume of the carotid arteries, classically occurs in aortic stenosis but can also result from severe stenosis of any of the cardiac valves and can occur with severely low cardiac output states of any cause. Because of the effects of aging on the vasculature, the typical findings of aortic stenosis (*pulsus parvus* and *tardus*) may be absent in the elderly. Inequality of the carotid pulses can be due to carotid atherosclerosis, especially in elderly patients, but in a young patient, consider supravalvular aortic stenosis. (The right side then should have the stronger pulse.) Transmitted murmurs of aortic origin, most often due to aortic stenosis (less often due to coarctation, patent ductus arteriosus, pulmonary stenosis or ventricular septal defect [VSD]), decrease in intensity as they ascend the neck, whereas a carotid bruit is usually louder higher in the neck and decreases in intensity as the stethoscope is inched proximally toward the chest.

Peripheral pulses

Pulsus alternans is associated with severe myocardial failure and is frequently accompanied by a third heart sound, both of which impart an ominous prognosis. In hypertension, simultaneous palpation of radial and femoral pulses may reveal a delay or relative weakening of the latter pulses, suggesting aortic coarctation. Finding a femoral (or carotid) bruit in an older patient suggests atherosclerosis, usually a generalized process, and may therefore suggest a coronary atherosclerotic (ischemic) origin of chest pain when the diagnosis is equivocal.

Heart Sounds

First heart sound (S_1)

S_1 intensity, which usually reflects mitral rather than tricuspid closure, is affected by the P-R interval, mitral valve structure and ventricular contractility. If S_1 is louder at the lower left sternal border than at the apex (implying loud T_1), suspect ASD or tricuspid stenosis. A variable S_1 intensity during a wide complex regular tachycardia suggests atrioventricular dissociation and a ventricular origin of a wide complex arrhythmia.

Systolic ejection sounds (clicks)

The ejection sound occurs in early systole, approximately 40–60 ms after S_1 . The S_1 -ejection sound cadence can be approximated by saying “pa-da” or “pa-ta” quickly (4). Because an aortic ejection sound is not usually heard with uncomplicated coarctation, its presence should suggest associated bicuspid aortic valve. In LV outflow obstruction, the ejection sound localizes the obstruction to the aortic valve level. An ejection sound would be expected to be absent in subvalvular stenosis. The ejection sound is related to mobility of the valve but not the severity. A pulmonary ejection sound can occur in idiopathic dilatation of the pulmonary artery, and the latter may be a masquerader of ASD, especially in young adults. The pulmonary valve ejection sound decreases with inspiration. Almost all other right heart acoustic events augment with inspiration. The aortic ejection sound is transmitted to the aortic area and the apex and does not change appreciably with respiration.

Mid-late nonejection systolic clicks

Although commonly caused by mitral valve prolapse, other causes of nonejection clicks (that can masquerade as mitral prolapse) include ventricular or atrial septal aneurysms, ventricular free wall aneurysms and ventricular and atrial mobile tumors, such as myxoma. Maneuvers that decrease LV volume, such as standing or Valsalva’s maneuver, move the click associated with mitral valve prolapse earlier in the cardiac cycle. Conversely, maneuvers that increase LV volume have a directionally opposite effect. The presence of a typical mid-late systolic click followed by a systolic murmur is diagnostic of mitral valve prolapse.

Second heart sound (S_2)

Physiologic splitting of S_2 is frequently heard in children and young adults, but S_2 is frequently single in older patients. Fixed splitting of S_2 should be verified with the patient in the sitting or standing position. Occasionally, normal subjects appear to have fixed splitting of S_2 in the supine position that becomes single in the upright position. Wide, fixed splitting, although considered typical of ASD, occurs in only 65–70% of patients with ASD (1). Fixed splitting can also be heard at times in patients with pulmonary stenosis or severe RV

failure. Wide splitting of S_2 occurs in partial anomalous pulmonary venous connection, but S_2 usually exhibits normal respiratory variation. Pulmonary hypertension, depending on its cause, may or may not cause wide splitting of S_2 , although the intensity of P_2 is usually increased and widely transmitted throughout the precordium, for example, to the apex. Ordinarily in normal individuals, only A_2 is heard at the apex. When both components of S_2 are heard at the apex in adults, implying an increased pulmonary component of S_2 , suspect ASD or pulmonary hypertension (1).

Paradoxical (reversed) splitting of S_2

Paradoxical splitting of S_2 is commonly caused by left bundle branch block. When paradoxical splitting of S_2 is found in association with aortic stenosis, usually in young adults (assuming left bundle branch block is absent), severe aortic obstruction is suggested. Similarly, paradoxical splitting of S_2 in hypertrophic obstructive cardiomyopathy implies significant resting LV outflow tract gradient (1). Transient paradoxical splitting of S_2 can occur with myocardial ischemia, such as during an episode of angina, either alone or in combination with an apical systolic murmur of mitral regurgitation (related to papillary muscle dysfunction that may also be transient) or a prominent S_4 .

Opening snap

In mitral stenosis, the presence of an opening snap (OS), often accompanied by a loud S_1 , implies a pliable mitral valve that is not heavily calcified. (In such cases, the patient may be a candidate for mitral commissurotomy or balloon valvuloplasty rather than mitral valve replacement.) Significant mitral stenosis may be present in the absence of an OS if the mitral valve leaflets are fixed and immobile. A tumor “plop” due to atrial myxoma has the same early diastolic timing as an OS and can be confused with it.

Third heart sound (S_3)

An S_3 in an older patient (without ventricular volume overload or high output state) implies ventricular systolic failure usually with an increase in filling pressure within the affected ventricle. An S_3 in a patient with valve regurgitation implies severe regurgitation or a failing ventricle or both. An S_3 may be heard in patients with restrictive LV filling who have normal systolic function (diastolic dysfunction). An S_3 is less common in cardiac conditions that cause thick, poorly compliant ventricles (e.g., LV hypertrophy that occurs with pressure overload states). The pericardial knock, which may sound like an early S_3 in timing, is of higher frequency than an S_3 , may vary with respiration and is more widely transmitted.

Fourth heart sound (S_4)

Bedside maneuvers that alter venous return affect the intensity and timing of S_4 but do not influence a split S_1 or S_1 -ejection sound sequence. A loud S_4 can be found in acute mitral regurgitation (e.g., with ruptured chordae) and can be a clue that the regurgitation is of recent onset. An S_4 is rarely audible in normal children and young adults. Although an S_4 can be heard in otherwise normal elderly patients, a palpable presystolic impulse (*a* wave) should not be present unless the LV is abnormal. An S_4 is present in most patients with hypertrophic obstructive cardiomyopathy, as well as in patients with acute myocardial infarction, and is often heard in patients with systemic hypertension and chronic ischemic heart disease. An S_4 may be heard as an early sign of diastolic dysfunction (in the absence of systolic dysfunction).

Cardiac Murmurs

Systolic murmurs

Regardless of the site of LV outflow tract obstruction, the associated systolic murmur usually has a crescendo-decrescendo quality and parallels the instantaneous LV-aortic gradient (1). Stenosis of a semilunar valve causes a delay in the peak intensity of the systolic murmur related to prolongation of ejection. The magnitude of the delay is proportional to the severity of obstruction. For valvular pulmonary stenosis, early timing of the ejection sound, a widely split S_2 and a delayed peak intensity of systolic murmur suggest severe stenosis. The systolic murmur of supravalvular aortic stenosis is maximal in the first or second right intercostal space, and a carotid pulse inequality may be present. The systolic murmur of branch pulmonary artery stenosis is widely transmitted to the axilla and posterior thorax. In organic mitral regurgitation, the murmur intensity is roughly correlated with regurgitation severity (7). Although mitral regurgitation is usually pansystolic, at times it can be late systolic in timing (in this case, suspect mitral prolapse or papillary muscle dysfunction). The absence of an apical systolic murmur, although decreasing the likelihood of significant mitral regurgitation, does not exclude it, especially in the presence of clinical shock states. The murmur of mitral regurgitation that is early systolic in timing may masquerade as an ejection murmur and can be heard in cases of severe regurgitation with markedly increased left atrial pressures, the latter reducing the late systolic LV-left atrial gradient.

In the elderly, the systolic murmur associated with aortic stenosis is often best heard at the apex and may have a musical quality. It may be confused with mitral regurgitation. Variable cardiac cycle lengths (e.g., PVCs or atrial fibrillation) will alter the murmur of aortic stenosis but not mitral regurgitation. The murmur of aortic stenosis is more likely to be transmitted to the right clavicular area and over the right clavicular head, as well as to the right carotid artery,

whereas a functional aortic outflow murmur tends to be more localized to the precordium. The systolic murmur of posterior mitral leaflet syndrome can be transmitted to the aortic area and be confused with aortic stenosis. Palpation of the carotid pulses can help differentiate between the two conditions. The systolic murmur of anterior mitral leaflet syndrome is transmitted posteriorly and can be heard along the thoracic spine and even at the base of the skull.

Although experienced examiners are generally accurate in diagnosing the systolic murmur of severe tricuspid regurgitation, mild and even moderate regurgitation may cause minimal or even no auscultatory signs (8). Although the systolic murmur of severe tricuspid regurgitation may be subtle, other associated bedside findings, including elevated jugular venous pressure with prominent V waves, parasternal lift and pulsatile liver, are important clinical clues. Inspiration may accentuate the murmur of tricuspid regurgitation but not consistently so, and the absence of inspiratory augmentation does not exclude tricuspid regurgitation. When the right ventricle dilates, it may occupy the usual site of the left ventricular apex. Under these circumstances, an apical systolic murmur (due to tricuspid regurgitation) can be confused with or attributed to mitral regurgitation. The systolic murmur of VSD is typically pansystolic and associated with a thrill along the left sternal border, but the murmur can be variable in contour (5). The intensity of the murmur does not correlate with the degree of shunt; that is, a small VSD can cause a very loud murmur. The murmur decreases or even disappears with pulmonary hypertension.

If the maximal intensity of a systolic murmur is in the first and second left intercostal spaces with radiation to the left clavicle, suspect supracristal VSD or patent ductus arteriosus. A loud pansystolic murmur of VSD may mask associated defects, such as patent ductus arteriosus. A wide pulse pressure suggests the latter or associated aortic regurgitation. The combination of VSD and aortic regurgitation may suggest patent ductus arteriosus, but the murmur in the latter condition peaks at S_2 and does not in the combination of VSD and aortic regurgitation.

Innocent systolic murmurs

Innocent or functional systolic murmurs, which occur during early RV or LV ejection, usually are not loud (generally grade 2 or less), are short (virtually never pansystolic) and have no associated abnormal clinical findings (e.g., absence of diastolic murmur, S_2 is normal, and there are no clicks) (6). Thus, innocent murmurs must be found in the setting of an otherwise normal cardiac exam. These murmurs are frequently heard in children, adolescents, and young adults. In the elderly, these murmurs usually emanate from the LV outflow tract and are often best heard at the left sternal border or near the apex. A systolic murmur caused by patent ductus arteriosus or ventricular septal defect can sometimes

masquerade as an innocent murmur. Functional murmurs caused by increased cardiac output can be loud but peak in early to mid systole and end before S_2 .

Diastolic Murmurs

Aortic regurgitation

The increased stroke volume secondary to severe aortic regurgitation may cause a loud systolic ejection murmur, but associated significant aortic stenosis can be excluded by the timing of the murmur (early-to-mid peak) and the rapid upstroke of the carotid pulse. Consider aortic regurgitation when there is a wide arterial pulse pressure, especially in young or middle-aged patients. A low diastolic blood pressure is an important ancillary clue. A short, early diastolic murmur does not exclude significant aortic regurgitation, especially if the patient has evidence of acute heart failure. The murmur of aortic regurgitation, although often heard at the left sternal border, can be primarily transmitted down the right sternal border. If so, one should suspect diseases of the aortic root, such as aortic aneurysm or dissection. The combination of hypertension, chest pain and right sternal border transmission of AR should suggest proximal aortic dissection. When the diastolic murmur has a “honking” or “cooing” quality, consider a perforated, everted or ruptured aortic cusp, such as with infective endocarditis. In the presence of mitral stenosis, an associated early diastolic murmur may be due to aortic regurgitation or pulmonary regurgitation (Graham Steell’s murmur), more often the former.

Pulmonary regurgitation

Pulmonary regurgitation due to pulmonary hypertension begins in early diastole and is long and high pitched. It may mimic the acoustic quality of the murmur of mild aortic regurgitation. In comparison, the murmur of pulmonary regurgitation due to organic pulmonary valve disease is lower pitched, harsher, rumbling, begins slightly later in diastole and often ends in mid-diastole.

Mitral stenosis

The presence of a loud S_1 or an OS should prompt a careful, thorough search for the easily overlooked localized apical diastolic murmur of the mitral stenosis. The duration of the diastolic murmur is related to the severity of mitral stenosis, and it is most prominent during rapid LV filling and persists as long as there is a significant pressure gradient across the mitral valve (1). Thus, a pandiastolic murmur implies severe mitral stenosis. Rarely in mitral stenosis, the diastolic murmur is not heard (so-called *silent* mitral stenosis). The usual reasons for *silent* MS are as follows: improper auscultation (most commonly), very mild mitral stenosis, decrease in flow rates across the mitral valve, such as in severe congestive heart failure or concomitant aortic or tricuspid stenosis, and abnormal chest wall configuration limiting auscultation,

such as in obesity or severe chronic obstructive pulmonary disease, in which case all sounds should be indistinct or distant.

Consider a more intensive auscultatory search for the diastolic murmur of mitral stenosis when atrial fibrillation is found in association with any of the following clinical scenarios: stroke or other systemic or peripheral embolus, unexplained pulmonary hypertension, unexplained congestive heart failure and unexplained recurrent pleural effusions. In some cases, brief exercise (e.g., sit-ups) will help bring out the diastolic murmur of mitral stenosis.

Tricuspid stenosis

A large jugular venous *a* wave (in sinus rhythm) with a slow Y descent should suggest tricuspid stenosis. The clinical findings of concomitant left-sided valve lesions often overshadow the tricuspid valve involvement. The murmur of tricuspid stenosis may be mistaken for aortic or pulmonary regurgitation.

Mid-diastolic flow murmurs

Almost any condition that increases flow across atrioventricular valves (such as mitral regurgitation, patent ductus arteriosus, intracardiac shunts, or complete heart block) can also cause a short mid-diastolic flow rumble (functional diastolic murmur) in the absence of organic atrioventricular valve stenosis.

Continuous murmurs

When a continuous murmur is loudest in the posterior thorax, consider the following: coarctation, pulmonary arteriovenous fistula and peripheral pulmonary stenosis. Continuous murmurs should be differentiated from to-and-fro murmurs, such as combined aortic stenosis and regurgitation. In the latter, the systolic component decreases before S_2 , whereas the continuous murmur of patent ductus arteriosus typically peaks at S_2 .

Bedside Physiologic Maneuvers to Differentiate Different Types of Murmurs

If the maneuver is intended to augment murmur intensity, then one should auscultate at the edge of the murmur’s radiation, where it is barely audible. An increase in intensity would be easier to appreciate in this way. After release of the Valsalva maneuver, with a sudden increase in venous return, right-sided murmurs (that decreased) return immediately (within one or two cardiac cycles), whereas left-sided murmurs gradually return after several cardiac cycles. Systolic ejection murmurs (such as caused by aortic or pulmonary stenosis) increase after a long cycle length, whereas regurgitant murmurs (such as caused by mitral or tricuspid regurgitation) do not. Amyl nitrite administration causes all stenotic murmurs, including hypertrophic obstructive car-

diomyopathy, to become louder. Transient arterial occlusion using blood pressure cuffs is accurate in assessing left-sided regurgitant murmurs, the latter augmenting as afterload is increased.

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