Lecturio Medical Knowledge Essentials – Physical Examination of the Cardiovascular System

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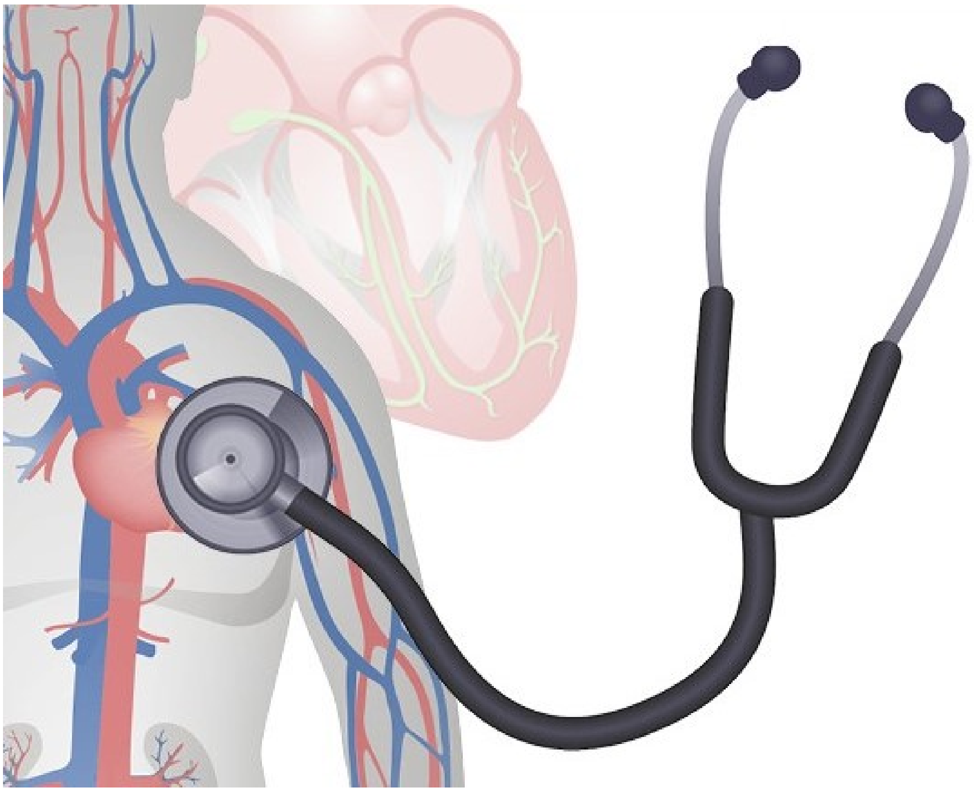

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Cardiovascular examination consists of assessment of the vital signs, jugular venous pulse (JVP), chest inspection, chest palpation, and auscultation of the heart.

For further review of this topic, including links to lectures by specialists in the field, follow this link: https://app.lecturio.com/#/course/c/47869

This article is not intended to be a substitute for professional medical advice and should not be relied on as health or personal advice. **Always seek the guidance of your doctor** or other qualified health professional with any questions you may have regarding your health or a medical condition.

Vital Measurements

You will likely acquire vital measurements for every patient you clinically examine. This will normally **include heart rate (HR)**, **respiratory rate (RR),** and **blood pressure (BP)**. These can be measured with basic equipment (a watch, a sphygmomanometer, and a stethoscope) in most situations and constitute a part of a physician's basic skill set.

It is very important that you learn to perform these examinations as well as the basic rules associated with each measurement. Even if the hospital or clinic provides the BP, most physicians in private practice will repeat all the vital sign measurements (except for temperature).

The patient should be resting comfortably in **supine position**. Access to the chest, arms, and legs is essential. Do not perform the exam through clothing—exposed **skin** is necessary. Having the patient dress in a hospital gown with a draping sheet available is recommended.

Observation

With the anterior chest exposed, observe your patient's thorax and the rest of their body. Look for the following: thorax, eyes, upper and lower extremities, and jugular venous distention.

Thorax

- Scars indicative of cardiac surgery. A vertical scar down the sternum is an indication of prior open-heart surgery.
- Chest deformities including **pectus excavatum** (a sunken sternum and ribs, a symptom of several connective tissue diseases such as Marfan syndrome) and **pectus carinatum** ("pigeon chest," a protrusion of the sternum and ribs).

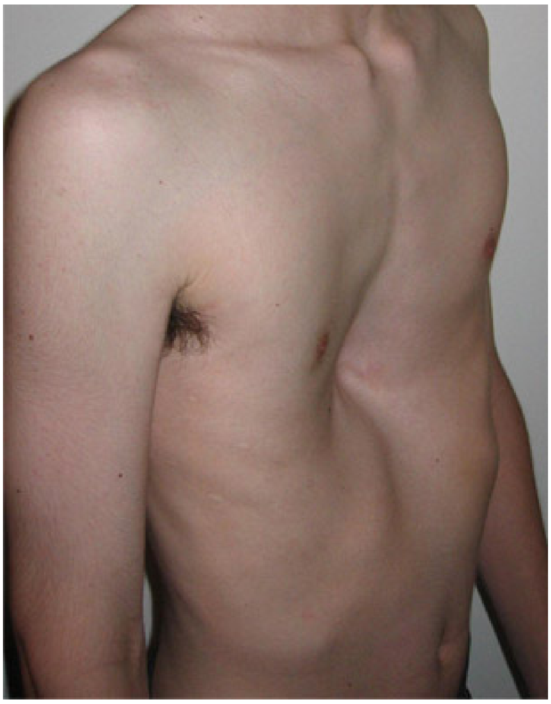


Image: “Pectus excavatum deformity” by The number c. License: CC BY-SA 3.0

Image: “Pectus Carinatum” by The number c. License: CC BY-SA 3.0

Eyes

- Yellow plaques around the eyes and eyelids, called xanthelasma, are a sign of hypercholesterolemia. This sign is a risk factor for cardiovascular disease.
- Roth’s spots are observed on the retina with an ophthalmoscope. They appear as a red ring surrounding a white center. These are indications of infective endocarditis.

Image: “Xanthelasma palpebrarum” by Klaus D. Peter, Gummersbach, Germany. License: CC BY 3.0 DE

Color fundus photograph of the left eye presenting Roth spots in the macula and the inferior temporal branches region

Image: “Figure 2” by Ceglowska, K., et al. License: CC BY 4.0

Upper and Lower Extremities

Clubbing of the fingers or toes. The distal part of the digit flattens and widens. This is a sign of lung disease or chronic hypoxemia, but may occasionally be seen in individuals without these conditions.

Image: “example of clubbing secondary to pulmonary hypertension in a patient with Eisenmenger’s syndrome” by Ann McGrath. License: Public Domain

Cyanosis, blue discoloration of the skin and/or mucous membranes implies poor perfusion The presence of at least 3 g/dL of reduced or deoxygenated hemoglobin (Hb) corresponds to an O2 saturation of < 85% if the patient is not anemic. The lower the Hb level, the lower the O2 saturation needed before cyanosis can be appreciated. At a Hb level of 10 g/dL, cyanosis does not appear until the O2 saturation is ~ 70%. If you see cyanosis in a severely anemic patient, this means that the concentration of Hb is very low and the patient is usually very ill. Cyanosis can be detected in the extremities (peripheral cyanosis) or the lips (central cyanosis, which is more serious).

Infective endocarditis lesions on the hands and feet

Osler’s nodes are raised, painful, red lesions on the hands and feet. They are caused by immune complex depositions. Janeway lesions are small, red, and painless. They are caused by microemboli. **Splinter hemorrhages** manifest as short dark lines beneath the nails. They are also caused by microemboli.

Image: “Splinter hemorrhages” by Splarka. License: Public Domain

Jugular Venous Distention

The cardiovascular exam includes observing the right internal jugular vein (IJV). This test is very useful to **evaluate right heart function and central venous pressure**.

Procedure:

1. Elevate the patient’s head while they are lying supine between 15° and 30°.
2. Identify the right IJV. This may take some practice. It crosses deep to the sternocleidomastoid muscle and anterior to the right ear. Ask the patient to turn their head to the left or to perform a Valsalva maneuver. The hepatojugular reflux maneuver can also help find the internal jugular vein. Apply firm pressure to the right upper quadrant of the liver for a few seconds and the IJV will fill with blood. Finally, a penlight can be very useful while trying to find the IJV.
3. Measure the top of the IJV fluid level in cm above the Angle of Louis (sternal angle). A normal measurement is a vertical height of 3 cm above the sternal angle.

Image: “This photo represents obvious external jugular venous distention in a patient with severe tricuspid regurgitation. Note the ropy vein that courses almost vertical in this patient who is sitting almost upright.” by Ferencga. License: CC BY-SA 3.0

Palpation

The palpation portion of the cardiovascular exam includes **evaluating the extremities and the carotid pulses as well as determining and evaluating the point of maximum impulse** (PMI). A relatively strong vibration is created when the ventricles contract. This vibration is transmitted down the apex of the heart and into the chest wall. In a healthy individual, the PMI is located at the 5th intercostal space along the left midclavicular line (just medial to and below the left nipple).

Evaluation of the Extremities

Temperature

Evaluate the extremities for temperature. Gently touch the hands and feet to determine their temperatures. A well-perfused extremity will be slightly warm or at body temperature. A **cold extremity indicates poor perfusion of the extremity** or that blood is just being shunted away from the skin. A warm extremity indicates a reduction of vascular resistance and may be a sign of septic shock.

Peripheral Pulses

There are a variety of pulse points you should be familiar with. Some are regularly used (radial pulse, carotid pulse) and some are infrequently used (femoral pulse). A thorough cardiac exam requires an **evaluation of all peripheral pulses**. Always **compare the paired pulses** (to detect differences in strength).

- Carotid artery
- Radial artery
- Femoral artery
- Popliteal artery
- Posterior tibial artery
- Dorsalis pedis artery

Peripheral Edema

Palpating the extremities is the preferred method for quantifying peripheral edema. The two types of edema are **pitting and non-pitting edema**.

Pitting edema refers to the depressed or indented area that results from pressure applied over an area of swollen/edematous **subcutaneous tissue**. It is caused by the displacement of thin, watery, protein-poor (transudative) interstitial fluid. Although it can affect any part of the body, **pitting edema** usually occurs in the legs, feet, and ankles when due to venous insufficiency caused by congestive heart failure. Edema associated with decreased plasma oncotic pressure (e.g., low serum albumin associated with liver failure or malnutrition) does not change with dependency. Non-pitting or “brawny” edema is observed when applied pressure does not leave an indentation. It is usually caused by compression or compromise of lymphatic drainage, and can also be seen in myxedema of hypothyroidism. The non-compressible subcutaneous tissue contains proteinaceous and possibly organizing collagenous or myxomatous substances.

Procedure: Press firmly on the affected area—usually the anterior lower leg (pressing down on the underlying tibia). Pitting is measured by the table below:

1+	Barely detectable impression when a finger is pressed into the skin
2+	Slight indentation, 15 seconds to rebound

3+	Deeper indentation, 30 seconds to rebound
4+	> 30 seconds to rebound

Point of Maximal Impulse (PMI)

Procedure:

1. Place the center of your palm at the PMI. The heel of your palm should rest at the left lower sternal border. Your fingers should wrap around the patient laterally.
2. Apply some pressure to the chest wall until you feel the heartbeat in your palm.
3. Identify the point of maximum impulse on the chest wall. It will be a small area, about 1 cm wide.

Obesity will make this part of the exam difficult. **The PMI of a healthy person with a normal and healthy heart will be located near the 5th intercostal space, along the midclavicular line**. The PMI of a dilated ventricle will be displaced laterally.

Thrill

A thrill may be detected if valvular disease is present. This is a **vibration associated with turbulent blood flow** through a damaged or malformed valve. Thrills are located near the valve listening points as discussed.

Auscultation

The detection and recognition of heart sounds play an important role in the diagnosis of various cardiac and valvular conditions. Because familiarity with heart sounds has such profound and practical importance, students undertaking the USMLE are expected to have a good understanding of their pathophysiology and their clinical applications.

Image: "Stethoscope" by Dr. Farouk. License: CC BY 2.0

Types of Heart Sounds

On auscultation, 2 heart sounds are heard from a normal heart, known as the **first and second heart sounds.** They reflect the turbulence created when the heart valves snap shut. Two extra heart sounds may also be heard, called the third and fourth heart sounds, and they may be heard in both normal and abnormal conditions. A murmur consists of a blowing, whooshing, or rasping sound heard during a heartbeat as blood flows through the heart's chambers and valves or through blood vessels near the heart. It can be a sign of a benign/physiologic or pathologic condition.

Murmur

A murmur is a sound that is produced by turbulent blood flow across a heart valve. The turbulent flow can occur for two reasons, the first is when blood flows across an abnormal heart valve and the second is when an increased amount of blood flows across a normal heart valve. Heart murmurs may be classified as physiological or innocent murmurs or pathologic murmurs, based on their etiology.

- A physiological or innocent murmur is heard when there is an increased turbulence of blood flow across a normal valve, as can happen in thyrotoxicosis and anemia, as well as during fever or exercise. The key features of innocent murmurs can be summarized by the "Seven S's":
    - Sensitive (changes with body position or with respiration)
    - Short duration (not holosystolic)
    - Single (no associated clicks or gallops)
    - Small (murmur limited to a small area and not radiating away from this area)
    - Soft (low amplitude)
    - Sweet (not harsh sounding)
    - Systolic (occurs during and is limited to systole)
- A pathologic murmur occurs when there is turbulence of blood flow across an abnormal valve. This can be due to either stenosis or insufficiency.

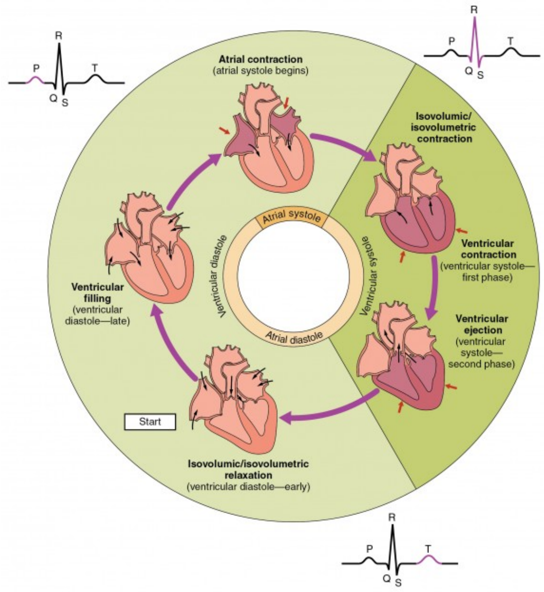
Stenosis

Stenosis refers to the abnormal narrowing of a valve orifice, commonly seen when age-related calcific deposits ("degenerative calcification") occur in the aortic valve or when the mitral valve has been damaged by scar tissue from healed rheumatic heart disease (RHD), mostly seen in developing countries; or by myxomatous disease and fibroelastic deficiency, which is more common in developed countries.

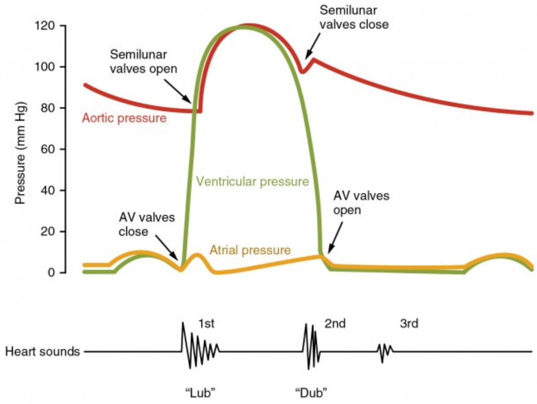
Regurgitation

Regurgitation refers to the abnormal backward flow of blood from a high-pressure chamber to a low-pressure chamber, often due to an incompetent valve (i.e., a valve that cannot close properly). An example is aortic regurgitation (AR), most commonly due to congenital or degenerative abnormalities of the aortic leaflets, aortic root, and ascending aorta in developed countries, while RHD remains the most common cause of severe AR worldwide.

Origins and Timing of the Heart Sounds



[Image](#): Overview of the cardiac cycle. The cardiac cycle begins with atrial systole and progresses to ventricular systole, atrial diastole, and ventricular diastole, when the cycle begins again. Correlations to the ECG are highlighted. "Overview of the Cardiac Cycle" by PhilSchatz. License: [CC BY 4.0](#)



[Image](#): Heart sounds and the cardiac cycle. In this illustration, the x-axis reflects time with a recording of the heart sounds. The y-axis represents pressure. "Heart Sounds and the Cardiac Cycle" by PhilSchatz. License: [CC BY 4.0](#)

First and Second Heart Sounds

The closure of the heart valves produces vibrations that are picked up as the two heart sounds.

The first heart sound, S1, corresponds to the **closure of the atrioventricular valves**—the tricuspid and mitral valves of the heart. S1 represents the **start of ventricular systole**. The closure of the mitral valves precedes the closure of the tricuspid valves, but this is only minimally different so that S1 is usually heard as a single sound. S1 is best heard at the apex of the heart.

The second heart sound, S2, corresponds to the **closure of the semilunar valves**—the aortic and pulmonary valves of the heart. S2 signifies the **end of ventricular systole** and the **beginning of diastole**. Compared to the first heart sound, S2 is shorter, softer, and slightly higher pitched. A reduced or absent S2 indicates pathology due to an abnormal aortic or pulmonic valve.

The pulmonary component of S2 is referred to as P2, and the aortic component is called A2.

It is important to be able to clearly identify S1 and S2 because it helps distinguish systolic from diastolic murmurs and other events in the cardiac cycle. Here are three clues to help distinguish them: the period of time between S1 and S2 (systole) is shorter than the period of time between S2 and the next S1 (diastole); S1 is usually louder than S2 (useful if tachycardia interferes with the interpretation); and S1 is synchronized with the carotid pulse.

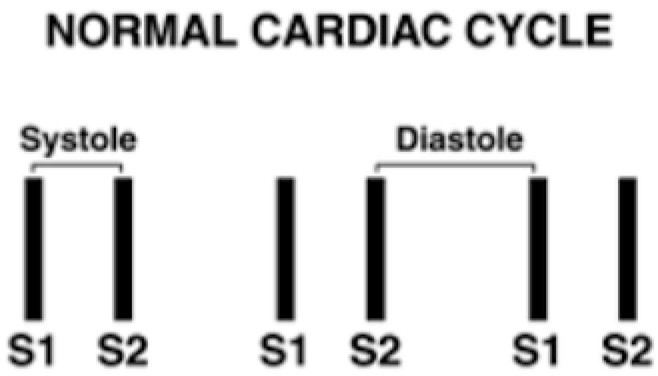
Splitting of the Second Heart Sound

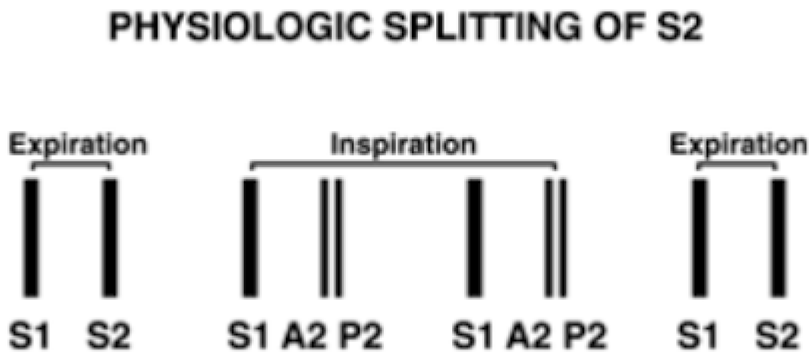
Physiologic Splitting of S2:

Both the aortic valve and the pulmonic valve will close when the pressure above each of them is higher than the pressure in the ventricle below. **The pulmonic valve closes later than the aortic valve because of two main factors.** The first is that the vascular resistance in the pulmonary artery is lower than that in the aorta, so that blood continues flowing into the pulmonary artery after the aortic valve closes. In 70% of normal adults, this difference can be heard as the splitting of the second heart sound. Additionally, during inspiration, more blood fills the right ventricle leading to a slightly longer ejection time of the right ventricle, adding to the delayed pulmonic valve closure and to the length of the S2 split. A2 is heard widely all over the chest. P2 is usually soft and only heard at the pulmonic region (second intercostal space, left sternal border), but even here A2 is louder.

Abnormal (Pathologic) Splitting of S2:

A. Wider-than-normal splitting of S2: It is an exaggerated (persistent) physiological split that is more pronounced during inspiration. Wide splitting is caused by delayed closure of the pulmonic valve, such as in pulmonic stenosis or right bundle branch block, or in early closure of the aortic valve caused by mitral regurgitation.
B. Fixed splitting of S2: wide splitting that does not vary with respiration, often due to prolonged right ventricular systole, seen in atrial septal defect or advanced right ventricular failure.
C. Reversed or paradoxical splitting of S2: Aortic valve closure delayed due to aortic stenosis) or conduction disease (left bundle branch block). Normal inspiratory delay of P2 usually makes the split disappear.





Normal cardiac cycle and splitting of S2. Image by Lecturio.

High-yield fact:

Absent normal/physiologic splitting of S2 can be seen in:

- Severe aortic stenosis (in elderly patients)
- Ventricular septal defect (VSD) with Eisenmenger syndrome (in pediatric patients)

Extra Heart Sounds

Third Heart Sound (S3)

Extra heart sounds include the third and fourth heart sounds. The **third heart sound (S3) is a mid-diastolic, low-pitched sound**. S3 occurs after S2, during the rapid passive filling of the ventricle. With the presence of S3, the heart sounds are described as having a **gallop rhythm**, simply because its addition alongside S1 and S2 creates a cadence of three heart sounds resembling a galloping horse, especially at rapid heart rates, which sounds like "Kentucky." S3 is also called a ventricular gallop.

A physiologic S3 is produced when there is rapid filling during diastole, as can happen in conditions which increase cardiac output such as thyrotoxicosis and pregnancy; it is often a normal finding in children. On the other hand, **a pathologic S3** is produced when there is decreased compliance of the ventricle (dilatation or overload), arising from high left ventricular filling pressures and abrupt deceleration of blood as it flows into the ventricle at the end of the rapid filling phase of diastole. Causes include decreased myocardial contractility, heart failure, and ventricular volume overload from aortic or mitral regurgitation, and left-to-right shunts (e.g., patent ductus arteriosus, ventricular septal defect). Reduced right ventricular compliance can also cause a pathologic S3, including right ventricular failure and **constrictive pericarditis.**

The third heart sound (S3) is an extra heart sound which follows S2 and is caused by blood from the left atrium "colliding" with residual blood in the left ventricle. It is associated with heart failure. Image by Lecturio.

Fourth Heart Sound (S4)

The fourth heart sound (S4) is a late diastolic sound. It is a bit higher pitched than S3. An S4 sound can produce a **gallop rhythm, but with a cadence that matches that of "Tennessee."** It is never heard when there are no atrial contractions (it is absent in atrial fibrillation). S4 is caused by decreased ventricular compliance; the most common causes of a left-sided S4 include hypertensive heart disease, aortic stenosis, and ischemic and hypertrophic cardiomyopathy. Reduced right ventricular compliance, as in pulmonary hypertension and pulmonary stenosis, can also cause a right-sided S4.

The fourth heart sound (S4) precedes S1 and is usually caused by atrial systolic contraction of blood into a poorly-compliant ("stiff") left ventricle, as can be seen in systemic hypertension. Image by Lecturio.

It is possible for the third and fourth heart sounds to co-exist, in which case it is called a **quadruple rhythm**. This indicates significantly impaired ventricular function. If S3 and S4 are superimposed when tachycardia is present, it produces a **summation gallop**.

Murmurs

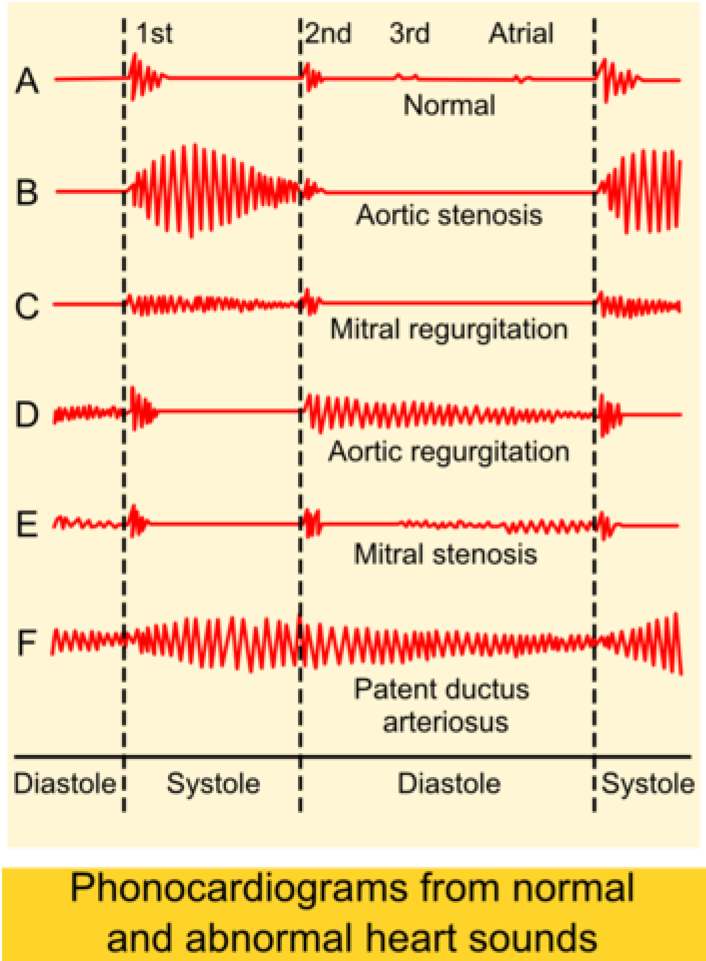


Image: "Phonocardiograms from normal and abnormal heart sounds" by Madhero88. Licence: CC BY 3.0

Systolic Murmurs

Systolic murmurs are murmurs that are **produced during systole** (contraction) of the ventricles, which is the period between S1 and S2. These murmurs can be midsystolic (ejection), late systolic, or pansystolic. Systolic murmurs can be either normal or abnormal.

Midsystolic Ejection Murmurs

Midsystolic ejection murmurs have their highest intensity in the middle of systole. They are often described as having a crescendo-decrescendo quality. This can be a physiological murmur, caused by an increased flow through a normal valve; or it can indicate various pathologic conditions, such as aortic stenosis or pulmonary stenosis. In cases of congenital aortic or pulmonary stenosis, an early high-pitched systolic ejection click may also be heard, representing the sudden opening of these valves, which are still mobile.

Late Systolic Murmurs

Late systolic murmur is when there is a gap between hearing S1 and the murmur. This can be caused by mitral regurgitation, as in the case of papillary muscle dysfunction or mitral valve prolapse.

Pansystolic (Holosystolic) Murmurs

A pansystolic murmur extends from S1 to S2. The pitch and loudness of this murmur stay the same during systole. The murmur is caused by leakage from a high-pressure chamber to a low-pressure chamber. Causes of pansystolic murmurs include mitral or tricuspid regurgitation and ventricular septal defect.

High-yield fact:

A mid-systolic murmur in an asymptomatic individual is most likely physiological, in contrast to diastolic murmurs which are always pathological.

High-yield fact:

It is usually easier to auscultate a systolic murmur than a diastolic one because it tends to be louder, with a harsher sound, and does not usually require a special maneuver to accentuate it.

Diastolic Murmurs

Diastolic murmurs, as their name implies, **occur during diastole of the ventricles**. They are always pathological. Compared to systolic murmurs, they are less common, softer, and more difficult to hear. There are two basic types in adults. **Early decrescendo** (decreasing in intensity) diastolic murmurs is caused by regurgitant flow through an incompetent semilunar valve, usually the aortic. **Rumbling diastolic** murmurs in mid- or late diastole are usually caused by stenosis of an atrioventricular valve, usually the mitral.

Early Diastolic Murmur

Early diastolic murmur starts with S2 and is a decrescendo murmur which is loudest at its commencement. It produces a high-pitched sound. Causes of an early diastolic murmur include aortic regurgitation or pulmonary regurgitation. The decrescendo quality mirrors the peak in aortic and pulmonary pressures at the start of diastole.

Rumbling (Mid- and Late Diastolic) Murmurs

Compared to early diastolic murmurs, these are lower pitched and can be mitral or tricuspid stenosis or an atrial myxoma (rare). In mitral stenosis, the diastolic murmur may be preceded by a high-pitched **opening snap** which represents the abrupt opening of the stenosed mitral valve.

Continuous Murmurs

Continuous murmurs occur during both systole and diastole without a pause. The sound is created by unidirectional flow when there is a communication between a high-pressure and a low-pressure source. The constant pressure gradient results in a continuous flow. The most common causes are nonvalvular and include patent ductus arteriosus, an arteriovenous fistula, and a venous hum (a benign sound, common in children, produced by turbulence of blood in the jugular veins).

Grading of Murmurs

If a murmur is heard, various dynamic maneuver tests are required to characterize it further. These maneuvers alter circulatory hemodynamics and, in doing so, change the emphasis with different murmurs.

- Grade 1: Murmur is very soft, and is initially not heard.
- Grade 2: Murmur is soft, but can be readily heard by a skilled examiner.
- Grade 3: Murmur is easy to hear.

- Grade 4: Murmur is slightly loud and accompanied by a palpable thrill (these murmurs are always pathological).
- Grade 5: Murmur is very loud, and the accompanying thrill is easily palpable.
- Grade 6: Murmur is so loud that it is audible even without direct placement of the stethoscope on the chest.

Note:

The intensity of the murmur doesn't always correlate to the severity of the lesions, as a smaller VSD produces louder murmurs than a larger VSD.

High-yield fact:

- Murmurs of grade III and above are usually pathological.
- Thrills are palpable murmurs, and only can be felt in murmurs of grade IV and above.

Chest Areas, Dynamic Auscultation and Dynamic Maneuvers

There are **four chest areas** that the stethoscope is placed over of to listen to heart sounds and any abnormal findings. **Auscultation** can be carried out in a clockwise manner, starting with the aortic then the pulmonic and mitral areas, followed by the tricuspid area.

To identify the difference between the two heart sounds on auscultation, palpation of the pulse (carotid or radial) while listening to the heart can be helpful. The pulse indicates systole, therefore corresponding to the first heart sound S1. Being aware of when systole and diastole occurs is useful in case an additional heart sound is heard so that it can be timed in the cardiac cycle and accurately described.

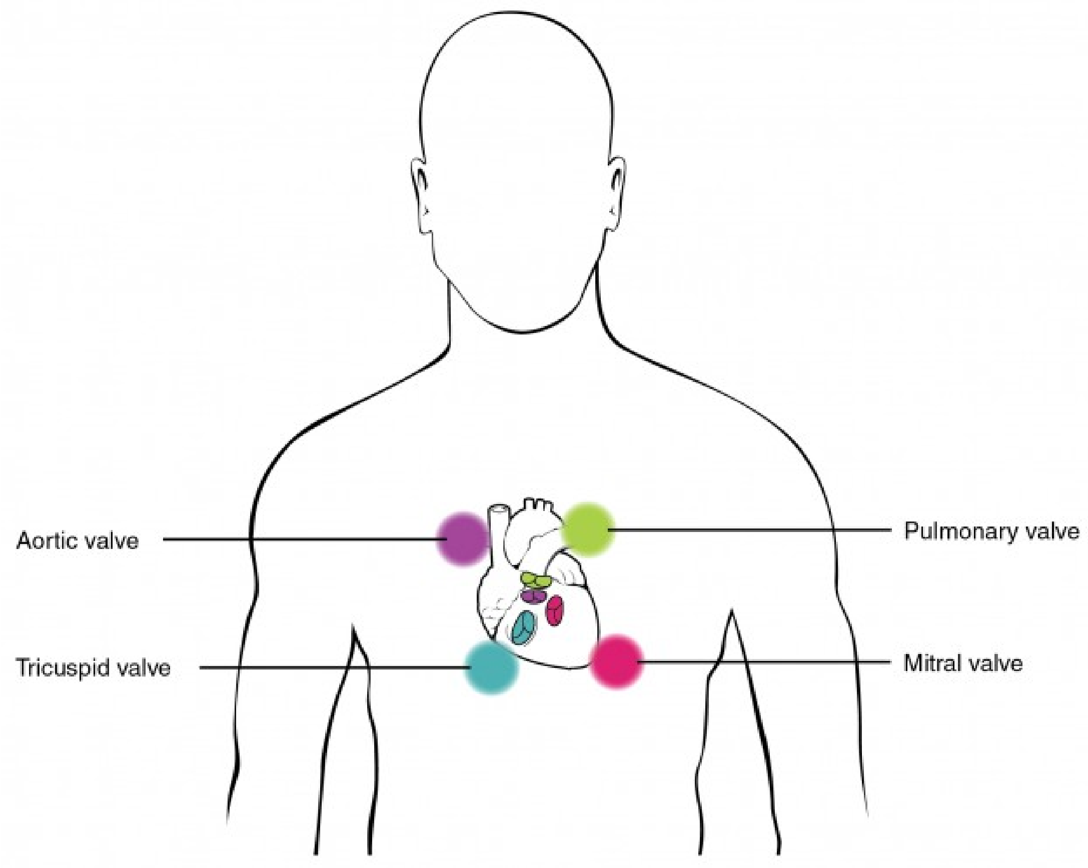


Image: “Stethoscope Placement for Auscultation” by PhilSchatz. License: CC BY 4.0

The **aortic area** is located in the second intercostal space, at the right sternal border. The diaphragm of the stethoscope can be placed at this site to listen for aortic stenosis.

The **pulmonic area** is at the left second intercostal space, at the left sternal border, opposite the aortic area. The diaphragm is placed here to listen for P2 and pulmonary flow murmurs.

The **mitral area** is also referred to as the apex of the heart. It is located in the fifth intercostal space, in the midclavicular line. This area should be auscultated with both the bell and the diaphragm of the stethoscope. Low-pitched sounds, such as the diastolic murmur of mitral stenosis and the third heart sound, can be better appreciated with the bell. The diaphragm can be used to detect high-pitched sounds, such as the fourth heart sound and mitral regurgitation.

The **tricuspid area** is located in the fourth and fifth intercostal spaces, at the left sternal border. The diaphragm is placed at this site to listen for tricuspid regurgitation.

Erb’s point is located at the third intercostal space along the left sternal border, at the midpoint between the base and apex of the heart. Both the S1 and S2 sounds can be heard here, as well as all other heart sounds. **It is often used as a quick assessment of heart rate as well as determining if there is a pulse deficit** (when not all heart beats reach the periphery, e.g., in atrial fibrillation, detected by simultaneously palpating the radial pulse). Aortic regurgitation is also heard best here (if not due to aortic root dilatation).

Know that even when a murmur is heard more clearly at a certain area of the chest, this might not always be helpful in determining its origin because a **murmur can radiate** away its site of origin. For example, a mitral regurgitation murmur is best heard in the mitral area but it may also be heard anywhere else on the chest. This murmur is also characterized by its radiation to the left axilla. The systolic ejection murmur of aortic valve origin characteristically radiates to the carotid arteries.

Dynamic Auscultation

This refers to altering heart sounds by changing the circulatory hemodynamics. This can be used to distinguish the clinical cause of similar auscultatory findings and is frequently tested on board exams. It is important to understand the physiologic alterations produced by certain maneuvers.

Dynamic Maneuvers

If a murmur is heard, various dynamic maneuver tests can be used to characterize it further. These maneuvers alter circulatory hemodynamics and, in doing so, change the emphasis with different murmurs.

Respiration can be used to differentiate between right-sided and left-sided murmurs. **Inspiration** has the effect of **increasing venous return**, and as there is an **increase in blood flow to the right side of the heart, right-sided murmurs are accentuated.** On the other hand, **expiration causes left-sided murmurs to become louder.**

Another respiration maneuver is deep expiration. As the patient leans forward and is in deep expiration, the base of the heart is brought closer to the chest wall. In this maneuver, the murmur of aortic regurgitation, not caused by a dilated aortic root, can be appreciated at Erb’s point, which is the point between the base and the apex of the heart.

1. **The Valsalva Maneuver**

This is a well-known and often-used dynamic maneuver, involving forcible exhalation against a closed glottis after full inspiration for 10-20 seconds, causing increased intrathoracic pressure. The normal systolic blood pressure (SBP) response follows four phases (two phases while the glottis is closed and two after the start of breathing normally):

Phase I (onset of the strain phase): Very short phase at the start of the maneuver. The intrathoracic pressure increases and there is a brief rise in cardiac output and blood pressure due to compression and hence increase in the pressure in all blood vessels within the chest and abdomen including the superior and inferior vena cavae, as well as in the cardiac chambers, associated with a reactive decrease in heart rate due to the baroreceptor reflex.

Phase II (continued strain): SBP decreases to below baseline as the strain is maintained, due to decreased venous return. Most murmurs become softer, but the **systolic murmur of hypertrophic cardiomyopathy increases** and the **mitral valve prolapse murmur is heard**. The heart rate increases due to the baroreceptor response (reflex) to a decreased SBP.

Phase III (“release phase” = normal breathing starts): Very short phase characterized by a rapid drop in SBP and a decrease in left ventricular volume due to decreased intrathoracic pressure, which may cause syncope or pre-syncope/dizziness. Right-sided murmurs are louder for a short interval. The heart rate increases due to the baroreceptor reflex.

Phase IV (recovery or “overshoot” phase): SBP increases, due to reflex sympathetic activation and increased stroke volume. Heart rate decreases due to the baroreceptor reflex.

The Valsalva maneuver accentuates the murmurs of hypertrophic cardiomyopathy and mitral valve prolapse when listening at the left sternal edge.

2. **Squatting**

Squatting is another dynamic maneuver which causes an increase in venous return. In this test, the patient quickly moves from a standing position to a squat. This makes most murmurs louder, including aortic stenosis and mitral regurgitation murmurs, while the murmur of hypertrophic cardiomyopathy and mitral valve prolapse are heard as softer and shorter. When the patient does the opposite, by standing quickly from a squatting position, then the opposite changes in murmurs occur.

3. **Isometric Exercise**

Isometric exercise can also be used for eliciting certain types of murmurs. For this exercise, the patient sustains a **handgrip** for half a minute. This exercise **increases afterload** (or peripheral resistance). The **murmur of mitral regurgitation is accentuated.** The murmur of aortic stenosis and hypertrophic cardiomyopathy becomes softer, while the murmur of mitral valve prolapse becomes shorter.

Summary Table

Heart Sound	Causes
Normal Heart Sounds	
First heart sound (S1)	Closure of the mitral and tricuspid valves
Second heart sound (S2)	Closure of the aortic and pulmonary valves
Extra Heart Sounds	
Third heart sound (S3)	A physiological S3 is caused by rapid diastolic filling (e.g., pregnancy, thyrotoxicosis, and some children). A pathological S3 is caused by reduced compliance of the left ventricle (e.g., left ventricular failure, aortic regurgitation, mitral regurgitation, patent ductus arteriosus, and a ventricular septal defect) or reduced compliance of the right ventricle (right ventricular failure, constrictive pericarditis).
Fourth heart sound (S4)	Decreased ventricular compliance of the left ventricle (aortic stenosis, mitral regurgitation, hypertension, angina, myocardial infarction, and old age) or the right ventricle (pulmonary hypertension, pulmonary stenosis)
Systolic Murmurs	
Midsystolic murmur	Increased flow through a normal valve (physiologic or innocent murmur), aortic stenosis, pulmonary stenosis, hypertrophic cardiomyopathy, atrial septal defect
Late systolic murmur	Mitral regurgitation (MR), due to papillary muscle dysfunction, mitral valve prolapse or infective endocarditis
Pansystolic murmur	Mitral regurgitation, tricuspid regurgitation, ventricular septal defect (VSD), aortopulmonary shunts
Diastolic murmurs	
Early diastolic murmur	Aortic regurgitation, pulmonary regurgitation
Mid-diastolic murmur	Mitral stenosis, tricuspid stenosis, atrial myxoma (rare), acute rheumatic fever murmur (Carey Coombs murmur of mitral valvulitis)
Other	
Presystolic murmur	Mitral stenosis, tricuspid stenosis, atrial myxoma
Continuous murmur	Patent ductus arteriosus, arteriovenous fistula, venous hum

Review Questions

1. **Where is the best site to appreciate the murmur of aortic regurgitation?**
   A. At the left lower sternal border, with the patient in the left lateral decubitus position, after a short period of exercise.

   B. At the aortic area and carotid arteries to assess for radiation.
   C. At Erb's point, at the left lower sternal border in the 3rd ICS, with the patient sitting up, leaning forward, and holding their breath after expiration.
   D. At the left lower sternal border, during phase II of the Valsalva maneuver.
2. **What distinguishes a grade 6 murmur from other grades in the Levine System?**
   A. It is a murmur that is soft and difficult to hear.
   B. It is a murmur that can be heard without direct placement of the stethoscope.
   C. It is a murmur with a palpable thrill accompanying it.
   D. It is a murmur that can only be heard by someone experienced in auscultation.
3. **What is the cause of the physiological splitting of the second heart sound?**
   A. Closure of the mitral and tricuspid valves just before ventricular systole.
   B. Increase in venous return during inspiration, causing the aortic valves to remain open for longer.
   C. Aortic regurgitation with retrograde leakage through the valve during ventricular diastole.
   D. Delayed closure of the pulmonic valve due to lower pressures in the pulmonary circulation and increased venous return during inspiration.

Answers: 1C, 2B, 3D

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