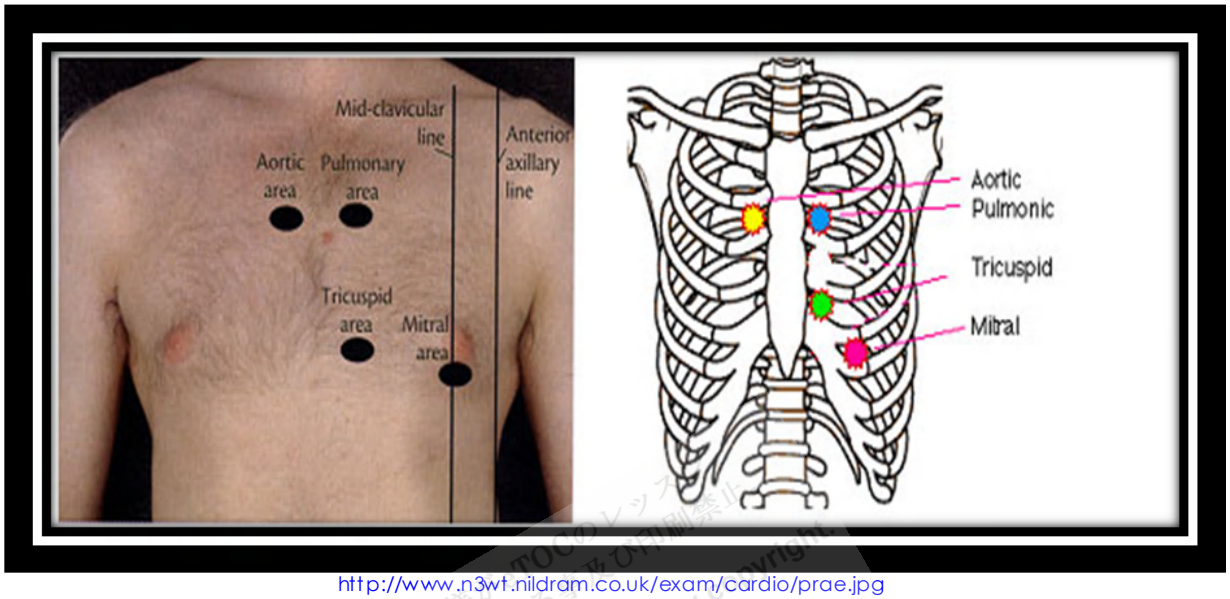
Cardiovascular Examination



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Complete examination of all systems is essential to detect **peripheral** and **systemic effects** of cardiac disorders and evidence of non-cardiac disorders that might affect the heart. Examination includes the following:

- Vital sign measurement
- Pulse palpation and **auscultation**
- Vein observation
- Chest inspection, **percussion**, auscultation, and **palpation**
- Cardiac percussion, palpation, and auscultation
- Lung examination
- Extremity and abdomen examination

Vital Signs

BP is measured in both arms and, for suspected **congenital cardiac disorders** or **peripheral vascular disorders**, in both legs. The **bladder** of an appropriately sized cuff encircles 80% of the limb's circumference, and the bladder's width is 40% of the circumference. The first sound heard as the Hg column falls is **systolic pressure**; disappearance of the sound is **diastolic pressure** (5th-phase Korotkoff sound). Up to a 15 mm Hg pressure differential between the right and left arms is normal; a greater differential suggests a vascular abnormality (eg, **dissecting thoracic aorta**) or a peripheral vascular disorder. Leg pressure is usually 20 mm

Hg higher than arm pressure. **Ankle-brachial index** (ratio of ankle to arm systolic BP) is normally> 1. A **Doppler** probe may be used to measure the ankle BP if the pedal pulses are not easily **palpable**.

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Heart rate and rhythm are assessed by palpating the **carotid** or **radial pulse** or by cardiac auscultation if **arrhythmia** is suspected; some heartbeats during arrhythmias may be audible but do not generate a palpable pulse.

Respiratory rate, if abnormal, may indicate cardiac **decompensation** or a primary lung disorder. The rate increases in patients with heart failure or anxiety and decreases or becomes **intermittent** in the **moribund**. Shallow, rapid respirations may indicate **pleuritic pain.**

Temperature may be elevated by acute **rheumatic fever** or cardiac infection (eg, **endocarditis**). After MI, low grade fever is very common. Other causes are sought only if fever persists > 72 h.

Orthostatic changes: BP and heart rate are measured with the patient **supine**, seated, and standing; a 1-min interval is needed between each change in position. A difference of ≤ 10 mm Hg is normal; the difference tends to be a little greater in the elderly due to loss of **vascular elasticity**.

Pulsus paradoxus: Normally during inspiration, systolic arterial BP can decrease as much as 10 mm Hg, and pulse rate increases to compensate. A greater

decrease in systolic BP or weakening of the pulse during inspiration is considered **pulsus paradoxus**. Pulsus paradoxus occurs in

- **Cardiac tamponade** (commonly)
- **Constrictive pericarditis**, severe asthma, and COPD (occasionally)
- Restrictive **cardiomyopathy**, severe **pulmonary embolism**, and **hypovolemic shock** (rarely)

BP decreases during inspiration because negative **intrathoracic pressure** increases **venous** return and hence right ventricular (RV) filling; as a result, the **interventricular septum bulges** slightly into the left ventricular (LV) outflow tract, decreasing cardiac output and thus BP. This mechanism (and the drop in systolic BP) is exaggerated in disorders that cause high negative **intrathoracic pressure** (eg, asthma) or that restrict RV filling (eg, cardiac tamponade, **cardiomyopathy**) or outflow (eg, pulmonary embolism).

Pulsus paradoxus is quantified by inflating a BP cuff to just above systolic BP and **deflating** it very slowly (eg, ≤ 2 mm Hg/heartbeat). The pressure is noted when Korotkoff sounds are first heard (at first, only during expiration) and when Korotkoff sounds are heard continuously. The difference between the pressures is the "amount" of pulsus paradoxus.

Pulses

Peripheral pulses: Major peripheral pulses in the arms and legs are palpated for **symmetry** and volume (intensity); elasticity of the arterial wall is noted. Absence of pulses may suggest an arterial disorder (eg, **atherosclerosis**) or systemic embolism. Peripheral pulses may be difficult to feel in obese or muscular people. The pulse has a rapid upstroke, then collapses in disorders with a rapid runoff of arterial blood (eg, **arteriovenous** communication, **aortic regurgitation**). The pulse is rapid and bounding in **thyrotoxicosis** and **hypermetabolic** states; it is slow and **sluggish** in **myxedema**. If pulses are **asymmetric**, auscultation over peripheral vessels may detect a **bruit** due to **stenosis**.

Carotid pulses: Observation, palpation, and auscultation of both carotid pulses may suggest a specific disorder. Aging and arteriosclerosis lead to vessel rigidity, which tends to eliminate the characteristic findings. In very young children, the carotid pulse may be normal, even when severe aortic stenosis is present.

Auscultation over the carotid arteries can distinguish **murmurs** from bruits. Murmurs originate in the heart or great vessels and are usually louder over the upper **precordium** and diminish toward the neck. Bruits are higher-pitched, are heard only over the arteries, and seem more superficial. An arterial bruit must be distinguished from a **venous hum**. Unlike an arterial bruit, a venous hum is usually continuous, heard best with the patient sitting or standing, and is eliminated by compression of the **ipsilateral internal jugular vein**.

Table 1

Carotid Pulse Amplitude and Associated Disorders

Carotid Pulse Amplitude	Associated Disorder
Bounding and prominent	Hypertension Hypermetabolic states Disorders with a rapid rise and fall of pressure (eg, patent **ductus arteriosus**)
Jerky, with full expansion followed by sudden collapse (Corrigan's or water-hammer pulse)	Aortic valve regurgitation
Low in **amplitude** and volume with a delayed peak	Aortic stenosis (obstructing left ventricular outflow) Shock
Double-peaked (bifid) with a rapid rise	**Hypertrophic cardiomyopathy**
Bifid with normal or delayed rise	Combined aortic stenosis and regurgitation
Diminished unilaterally or bilaterally, often with a systolic bruit	**Extracranial carotid stenosis** due to atherosclerosis

Veins

Peripheral veins: The peripheral veins are observed for **varicosities**, **arteriovenous malformations** (AVMs) and shunts, and overlying inflammation and tenderness due to **thrombophlebitis**. An AVM or a shunt produces a continuous murmur

(heard on auscultation) and often a palpable thrill (because resistance is always lower in the vein than in the artery during systole and diastole).

Neck veins: The neck veins are examined to estimate venous wave height and waveform. Height is proportional to right atrial pressure, and waveform reflects events in the cardiac cycle; both are best observed in the internal jugular vein.

The jugular veins are usually examined with the patient reclining at 45°. The top of the venous column is normally just below the **clavicles** (upper limit of normal: 4 cm above the sternal notch in a vertical plane). The venous column is elevated in heart failure, volume overload, cardiac tamponade, **constrictive pericarditis**, **tricuspid stenosis**, **superior vena cava obstruction**, or reduced compliance of the RV. If such conditions are severe, the venous column can extend to jaw level, and its top can be detected only when the patient sits upright or stands. The venous column is low in **hypovolemia**.

Normally, the venous column can be briefly elevated by firm hand pressure on the abdomen (**hepatojugular** or **abdominojugular reflux**); the column falls back in a few seconds (maximum 3 respiratory cycles or 15 sec) despite continued abdominal pressure (because a compliant RV increases its stroke volume via the Frank-Starling mechanism). However, the column remains elevated (> 3 cm) during abdominal pressure in disorders that cause a **dilated** and poorly compliant RV or in obstruction of RV filling by tricuspid stenosis or right atrial tumor.

Normally, the venous column falls slightly during inspiration as lowered intrathoracic pressure draws blood from the periphery into the vena cava. A rise in the venous column during inspiration (Kussmaul's sign) occurs typically in chronic constrictive pericarditis, right ventricular MI, and COPD, and usually in heart failure and tricuspid stenosis.

Jugular vein waves can usually be **discerned** clinically but are better seen on the screen during central venous pressure monitoring.

The *a* waves are increased in pulmonary hypertension and tricuspid valve stenosis. **Gianta waves** (**Cannon waves**) are seen in **atrioventricular dissociation** when the atrium contracts while the tricuspid valve is closed. The *a* waves disappear in **atrial fibrillation** and are **accentuated** when RV compliance is poor (eg, in pulmonary hypertension or pulmonic stenosis). The *v* waves are very

prominent in tricuspid regurgitation. The x descent is steep in cardiac tamponade. When RV compliance is poor, the *y* descent is very abrupt because the elevated column of venous blood rushes into the RV when the tricuspid valve opens, only to be stopped abruptly by the rigid RV wall (in **restrictive myopathy**) or the pericardium (in **constrictive pericarditis**).

Chest Inspection and Palpation

Chest contour and any visible cardiac impulses are inspected. The precordium is palpated for pulsations (determining **apical impulse** and thus **cardiac situs**) and thrills.

Inspection: Chest deformities, such as shield chest and **pectus carinatum** (a prominent birdlike **sternum**), may be associated with hereditary disorders involving congenital cardiac defects (eg, **Turner's syndrome**). Rarely, a localized upper chest bulge indicates **aortic aneurysm** due to **syphilis**. **Pectus excavatum** (depressed sternum) with a narrow anteroposterior chest diameter and an abnormally straight thoracic spine may suggest **myxomatous** degeneration of valves or chordae (particularly **mitral**) or **Marfan syndrome**.

Palpation: A central **precordial heave** is a palpable lifting sensation under the sternum and anterior chest wall to the left of the sternum; it suggests severe RV hypertrophy (RVH). Occasionally, in congenital disorders that cause severe RVH, the precordium visibly bulges asymmetrically to the left of the sternum.

A sustained thrust at the apex (easily differentiated from the less focal, somewhat **diffuse precordial heave** of RVH) suggests LV hypertrophy (LVH). Abnormal focal systolic impulses in the precordium can sometimes be felt in patients with a **dyskinetic ventricular aneurysm**. An abnormal diffuse systolic impulse lifts the precordium in patients with severe **mitral regurgitation**. The lift occurs because the left atrium expands, causing anterior cardiac displacement. A diffuse and **inferolaterally displaced apical impulse** is found when the LV is dilated and hypertrophied (eg, in mitral regurgitation).

Location of thrills (palpable buzzing sensation present with particularly loud murmurs) suggests the cause.

Table 2

Location of Thrills and Associated Disorders	
Location of	**Associated Disorder**

Thrill	
Over the base of the heart at the 2nd intercostal space, just to the right of the sternum, during systole	Aortic stenosis
At the apex during systole	Mitral regurgitation
To the left of the sternum at the 2nd intercostal space	Pulmonic stenosis
To the left of the sternum at the 4th intercostal space	Small muscular ventricular septal **defect (Roger's disease)**

A sharp impulse at the 2nd intercostal space to the left of the sternum may result from exaggerated **pulmonic valve closure** in pulmonary hypertension. A similar early systolic impulse at the cardiac apex may represent closure of a stenotic mitral valve; opening of the stenotic valve sometimes can be felt at the beginning of diastole. These findings **coincide** with an **augmented 1st heart sound** and an opening snap of mitral stenosis, heard on auscultation.

Cardiac Auscultation

Auscultation of the heart requires excellent hearing and the ability to distinguish subtle differences in pitch and timing. Hearing-impaired health care practitioners can use **amplified stethoscopes**. High-pitched sounds are best heard with the diaphragm of the stethoscope. Low-pitched sounds are best heard with the bell. Very little pressure should be exerted when using the bell.

Excessive pressure converts the underlying skin into a diaphragm and eliminates very low-pitched sounds.

The entire precordium is examined systematically, typically beginning over the apical impulse with the patient in the left lateral **decubitus position**. The patient rolls supine, and auscultation continues at the lower left sternal border, proceeds **cephalad** with auscultation of each interspace, then **caudad** from the right upper sternal border. The clinician also listens over the left **axilla** and above the clavicles. The patient sits upright for auscultation of the back, then leans forward to aid auscultation of aortic and pulmonic diastolic murmurs or pericardial friction rub.

Major auscultatory findings include

- Heart sounds
- Murmurs
- Rubs

Heart sounds are brief, transient sounds produced by valve opening and closure; they are divided into systolic and diastolic sounds.

Murmurs are produced by blood flow **turbulence** and are more prolonged than heart sounds; they may be systolic, diastolic, or continuous. They are graded by intensity and are described by their location and when they occur within the cardiac cycle.

Table 3

Heart Murmur Intensity

Grade	Description
1	Barely audible
2	Soft but easily heard
3	Loud without a thrill
4	Loud with a thrill
5	Loud with minimal contact between

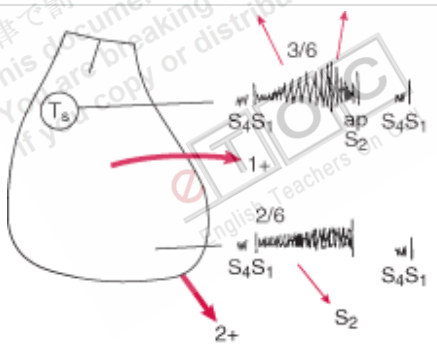
	stethoscope and chest
6	Loud with no contact between stethoscope and chest

Rubs are high-pitched, scratchy sounds often with 2 or 3 separate components; during **tachycardia**, the sound may be almost continuous.

The clinician focuses attention sequentially on each phase of the cardiac cycle, noting each heart sound and murmur. Intensity, pitch, duration, and timing of the sounds and the intervals between them are analyzed, often providing an accurate diagnosis. A diagram of the major auscultatory and palpatory findings of the precordium should be routinely drawn in the patient's chart each time the patient's cardiovascular system is examined. With such diagrams, findings from each examination can be compared.

Fig. 2

Diagram of physical findings in a patient with aortic stenosis and mitral regurgitation.



Murmur, character, intensity, and radiation are depicted. Sound of pulmonic closure exceeds that of aortic closure. Left ventricular (LV) thrust and right ventricular (RV) lift (heavy arrows) are identified. A 4th heart sound (S_4) and systolic thrill (T_S) are present. a = aortic closure sound; p =pulmonic closure sound; S_1 = 1st heart sound; S_2 = 2nd heart sound; 3/6 = grade of **crescendo-diminuendo murmur** (radiates to both sides of neck); 2/6 = grade of **pansystolic apical crescendo murmur**; 1+ =mild precordial lift of RV hypertrophy (arrow shows direction of lift); 2+ = moderate LV thrust (arrow shows direction of thrust).

Systolic heart sounds: Systolic sounds include the following:

- 1st heart sound (S_1)
- Clicks

S_1 and the 2nd heart sound (S_2, a diastolic heart sound) are normal components of the cardiac cycle, the familiar **"lub-dub" sounds.**

S_1 occurs just after the beginning of systole and is predominantly due to mitral closure but may also include tricuspid closure components. It is often split and has a high pitch. S_1 is loud in mitral stenosis. It may be soft or absent in mitral regurgitation due to valve leaflet sclerosis and rigidity but is often distinctly heard in mitral regurgitation due to **myxomatous degeneration** of the mitral apparatus or due to ventricular myocardial abnormality (eg, **papillary muscle dysfunction**, **ventricular dilation**).

Clicks occur only during systole; they are distinguished from S_1 and S_2 by their higher pitch and briefer duration. Some clicks occur at different times during systole as hemodynamics change. Clicks may be single or multiple.

Clicks in congenital aortic or pulmonic stenosis are thought to result from abnormal ventricular wall tension. These clicks occur early in systole (very near S_1) and are not affected by hemodynamic changes. Similar clicks occur in severe pulmonary hypertension. Clicks in mitral or **tricuspid valve prolapse**, typically occurring in mid to late systole, are thought to result from abnormal tension on **redundant** and **elongated chordae tendineae** or **valve leaflets**.

Clicks due to myxomatous degeneration of valves may occur any time during systole but move toward S_1 during maneuvers that transiently decrease ventricular filling volume (eg, standing, Valsalva maneuver). If ventricular filling volume is increased (eg, by lying supine), clicks move toward S_2, particularly in mitral valve prolapse. For unknown reasons, characteristics of the clicks may vary greatly between examinations, and clicks may come and go.

Diastolic heart sounds: Diastolic sounds include the following:

- 2nd, 3rd, and 4th heart sounds (S_2, S_3, and S_4)
- Diastolic knocks
- Mitral valve sounds

Unlike systolic sounds, diastolic sounds are low-pitched; they are softer in intensity and longer in duration. Except for S_2, these sounds are always abnormal in adults.

S_2 occurs at the beginning of diastole, due to aortic and pulmonic valve closure. Aortic valve closure normally precedes pulmonic valve closure unless the former is late or the latter is early. Aortic valve closure is late in left bundle branch block or aortic stenosis; pulmonic valve closure is early in some forms of **preexcitation phenomena**. Delayed pulmonic valve closure may result from increased blood flow through the RV (eg, in **atrial septal defect** of the **common secundum variety**) or complete right bundle branch block. Increased RV flow in atrial septal defect also abolishes the normal respiratory variation in aortic and pulmonic valve closure, producing a fixed split S_2. Left-to-right shunts with normal RV volume flow (eg, in membranous ventricular septal defects) do not cause fixed splitting. A single S_2 may occur when the aortic valve is regurgitant, severely stenotic, or atretic (in truncus arteriosus when there is a common valve).

S_3 occurs in early diastole, when the ventricle is dilated and **noncompliant**. It occurs during passive diastolic ventricular filling and indicates serious ventricular dysfunction in adults; in children, it can be normal. RV S_3 is heard best (sometimes only) during inspiration (because negative intrathoracic pressure augments RV filling volume) with the patient supine. LV S_3 is best heard during expiration (because the heart is nearer the chest wall) with the patient in the left lateral decubitus position.

S_4 is produced by augmented ventricular filling, caused by atrial contraction, near the end of diastole. It is similar to S_3 and heard best or only with the bell of the stethoscope. During inspiration, RV S_4 increases and LV S_4 decreases. S_4 is heard much more often than S_3 and indicates a lesser degree of ventricular dysfunction, usually diastolic. S_4 is absent in **atrial fibrillation** (because the atria do not contract) but is almost always present in active **myocardial ischemia** or soon after MI. S_3, with or without S_4, is usual in significant systolic LV dysfunction; S_4 without S_3 is usual in diastolic LV dysfunction.

A **summation gallop** occurs when S_3 and S_4 are present in a patient with **tachycardia**, which shortens diastole so that the 2 sounds merge. Loud S_3 and S_4 may be palpable at the apex when the patient is in the left lateral decubitus position.

A **diastolic knock** occurs at the same time as S_3, in early diastole. It is not accompanied by S_4 and is a louder, **thudding sound**, which indicates abrupt arrest of ventricular filling by a noncompliant, constricting pericardium.

An **opening snap** may occur in early diastole in mitral stenosis or, rarely, in tricuspid stenosis. Mitral opening snap is very high pitched, brief, and heard best with the diaphragm of the stethoscope. The more severe **mitral stenosis** is (ie, the higher the left atrial pressure), the closer the opening snap is to the pulmonic component of S_2. Intensity is related to the compliance of the valve leaflets: The snap sounds loud when leaflets remain elastic, but it gradually softens and ultimately disappears as sclerosis, fibrosis, and calcification of the valve develop. Mitral opening snap, although sometimes heard at the apex, is often heard best or only at the lower left sternal border.

Approach to murmurs: Timing of the murmur in the cardiac cycle correlates with the cause; auscultatory findings correlate with specific heart valve disorders. Various maneuvers (eg, **inspiration**, **Valsalva**, **handgrip**, **squatting**, **amyl nitrate inhalation**) can modify cardiac physiology slightly, making differentiation of causes of heart murmur possible.

Table 4

Etiology of Murmurs by Timing

Timing	Associated Disorders
Mid systolic (ejection)	Aortic obstruction (**supravalvular stenosis**, **coarctation** of the aorta, aortic stenosis, aortic sclerosis, hypertrophic cardiomyopathy, subvalvular stenosis) Increased blood flow across the aortic valve (**hyperkinetic states**, aortic regurgitation) Dilation of ascending aorta (**atheroma**, **aortitis**, **aneurysm** of aorta) Pulmonic obstruction (**supravalvular pulmonary artery stenosis**, **pulmonic stenosis**, **infundibular stenosis**) Increased blood flow across the pulmonic valve (hyperkinetic states, left-to-right shunt from **atrial septal defect**, **ventricular septal defect**) Dilation of pulmonary artery
Mid-late	Mitral valve prolapse, papillary muscle

systolic	dysfunction
Holosystolic	Mitral regurgitation, tricuspid regurgitation, ventricular septal defect
Early diastolic (regurgitant)	Aortic regurgitation: Acquired or congenital valve abnormality (myxomatous or calcific degeneration, rheumatic fever, endocarditis), dilation of valve ring (aortic dissection, **annuloaortic ectasia**, **cystic medial necrosis**, or hypertension), widening of **commissures** (syphilis); congenital bicuspid valve with or without ventricular septal defect Pulmonic regurgitation: Acquired or congenital valve abnormality, dilation of valve ring (pulmonary hypertension, Marfan syndrome), tetralogy of Fallot, ventricular septal defect
Mid diastolic	Mitral stenosis (rheumatic fever, congenital stenosis, **cor triatriatum**) Increased blood flow across nonstenotic mitral valve (mitral regurgitation, ventricular septal defect, patent ductus arteriosus, high-output states, complete heart block) Tricuspid stenosis Increased blood flow across nonstenotic tricuspid valve (tricuspid regurgitation, atrial septal defect, anomalous pulmonary venous return) Left or right atrial tumors, **atrial ball-valve thrombi**
Continuous	**Patent ductus arteriosus**, coarctation of the pulmonary artery, coronary or **intercostal arteriovenous fistula**, ruptured aneurysm of **sinus of Valsalva**, aortic septal defect, cervical venous hum, anomalous left coronary artery, **proximal coronary artery stenosis**, **mammary souffle** (venous hum from **engorged breast**

vessels during pregnancy), pulmonary artery branch stenosis, bronchial collateral circulation, small (restrictive) atrial septal defect with mitral stenosis, **coronary-cameral fistula**, aortic–right ventricular or atrial fistula

Table 5

Maneuvers That Aid in Diagnosis of Murmurs

Maneuver	Effect on Blood Flow	Effect on Heart Sounds
Inspiration	Simultaneously increases venous flow into the right heart, decreases venous flow into the left heart	Augments right heart sounds (eg, murmurs of tricuspid stenosis and regurgitation, those of pulmonic stenosis* [immediately] and regurgitation [usually]); reduces left heart sounds
Valsalva maneuver	Reduces size of left ventricle (LV); decreases venous return to the right heart and subsequently to the left heart	Augments murmur of hypertrophic obstructive cardiomyopathy and mitral valve prolapse, and diastolic murmur of mitral stenosis; reduces murmurs of aortic stenosis, mitral regurgitation, and tricuspid stenosis
Release of Valsalva maneuver	Increases volume of LV	Augments murmur of aortic stenosis, that of aortic regurgitation (after 4 or 5 beats), and those of pulmonic regurgitation or pulmonic stenosis* (immediately); reduces murmur of tricuspid stenosis
Isometric handgrip	Increases afterload and peripheral arterial resistance	Reduces murmurs of aortic stenosis and hypertrophic obstructive cardiomyopathy; reduces murmur of mitral valve prolapse or papillary muscle dysfunction; augments murmurs of mitral regurgitation and aortic regurgitation and diastolic murmur of mitral stenosis

Squatting	Simultaneously decreases venous return to the right heart and increases afterload and peripheral resistance	Augments murmurs of aortic regurgitation, aortic stenosis, mitral valve prolapse, and mitral regurgitation and diastolic murmur of mitral stenosis; reduces murmur of hypertrophic obstructive cardiomyopathy and mitral valve prolapse or papillary muscle dysfunction
Amyl nitrite	Causes intense venodilation, which reduces venous return to the right heart	Augments murmurs of hypertrophic obstructive cardiomyopathy and mitral valve prolapse; reduces murmur of aortic stenosis
□ *Patient may need to be standing for effect on pulmonic stenosis to be heard.		

All patients with heart murmurs are evaluated by chest x-ray and ECG. Most require **echocardiography** to confirm the diagnosis, determine severity, and track severity over time. Usually, a cardiac consultation is obtained if significant disease is suspected.

Systolic murmurs: Systolic murmurs may be normal or abnormal. They may be early, mid, or late systolic, or **holosystolic** (**pansystolic**). Systolic murmurs may be divided into **ejection**, **regurgitant**, and **shunt** murmurs.

Ejection murmurs are due to **turbulent** forward flow through narrowed or irregular valves or outflow tracts (eg, due to aortic or pulmonic stenosis). They are typically mid systolic and have a **crescendo-diminuendo** character that usually becomes louder and longer as flow becomes more obstructed. The greater the stenosis and turbulence, the longer the crescendo phase and the shorter the diminuendo phase.

Systolic ejection murmurs may occur without hemodynamically significant outflow tract obstruction and thus do not necessarily indicate a disorder. In normal infants and children, flow is often mildly turbulent, producing soft ejection murmurs. The elderly often have ejection murmurs due to valve and vessel sclerosis.

During pregnancy, many women have soft ejection murmurs at the 2nd intercostal space to the left or right of the sternum. The murmurs occur because a physiologic increase in blood volume and cardiac output increases flow velocity through normal structures. The murmurs may be greatly exaggerated if severe anemia complicates the pregnancy.

Regurgitant murmurs represent **retrograde** or abnormal flow (eg, due to mitral regurgitation, tricuspid regurgitation, or ventricular septal defects) into chambers that are at lower resistance. They are typically holosystolic and tend to be louder with high-velocity, low-volume regurgitation or shunts and softer with high-volume regurgitation or shunts. Late systolic murmurs, which may or may not be preceded by a click, are typical of mitral valve prolapse or papillary muscle dysfunction. Various maneuvers are usually required for more accurate diagnosis of timing and type of murmur.

Shunt murmurs may originate at the site of the shunt (eg, **patent ductus arteriosu**s, ventricular septal defects) or result from altered hemodynamics remote from the shunt (eg, pulmonic systolic flow murmur due to an atrial septal defect with left-to-right shunt).

Diastolic murmurs: Diastolic murmurs are always abnormal; most are early or mid diastolic, but they may be late diastolic (**presystolic**). Early diastolic murmurs are typically due to aortic or pulmonic regurgitation. Mid diastolic (or early to mid diastolic) murmurs are typically due to mitral or tricuspid stenosis. A late diastolic murmur may be due to rheumatic mitral stenosis in a patient in sinus rhythm.

A mitral or tricuspid murmur due to an atrial tumor or thrombus may be **evanescent** and may vary with position and from one examination to the next because the position of the intracardiac mass changes.

Continuous murmurs: Continuous murmurs occur throughout the cardiac cycle. They are always abnormal, indicating a constant shunt flow throughout systole and diastole. They may be due to various cardiac defects. Some defects produce a thrill; many are associated with signs of RVH and LVH. As pulmonary artery resistance increases in **shunt lesions**, the diastolic component gradually decreases. When pulmonary and systemic resistance equalize, the murmur may disappear.

Patent ductus arteriosus murmurs are loudest at the 2nd intercostal space just below the medial end of the left clavicle. **Aorticopulmonary** window murmurs are central and heard at the 3rd intercostal space level. Murmurs of systemic arteriovenous fistulas are best heard directly over the lesions; those of pulmonic arteriovenous fistulas and pulmonary artery branch stenosis are more **diffuse** and heard throughout the chest.

During pregnancy, a continuous venous hum from breast vessels (mammary souffle) may be mistaken for a continuous cardiac murmur.

Pericardial friction rub: A pericardial friction rub is caused by movement of inflammatory **adhesions** between visceral and parietal pericardial layers. It is a high-pitched or **squeaking sound**; it may be systolic, diastolic and systolic, or **triphasic** (when atrial contraction accentuates the diastolic component during late diastole). The rub sounds like pieces of leather squeaking as they are rubbed together. Rubs are best heard with the patient leaning forward or on hands and knees with breath held in expiration.

Extremity and Abdominal Examination

The extremities and abdomen are examined for signs of fluid overload, which may occur with heart failure as well as noncardiac disorders (eg, renal, hepatic, lymphatic).

Extremities: In the extremities (primarily the legs), fluid overload is manifest as edema, which is swelling of soft tissues due to increased interstitial fluid. Edema may be visible on inspection, but modest amounts of edema in very obese or muscular people may be difficult to recognize visually. Thus, extremities are palpated for presence and degree of **pitting** (visible and palpable depressions caused by pressure from the examiner's fingers, which displaces the **interstitial fluid**). The area of edema is examined for extent, symmetry (ie, comparing both extremities), warmth, **erythema**, and tenderness. With significant fluid overload, edema may also be present over the **sacrum**, genitals, or both.

Tenderness, erythema, or both, particularly when unilateral, suggests an inflammatory cause (eg, cellulitis or **thrombophlebitis**). **Non-pitting edema** is more suggestive of lymphatic or vascular obstruction than fluid overload.

Abdomen: In the abdomen, significant fluid overload manifests as **ascites**. Marked ascites causes visible abdominal distention, which is tense and non-

tender to palpation, with shifting dullness on abdominal percussion and a fluid wave. The liver may be **distended** and slightly tender, with a hepatojugular reflux present.

Reference: http://www.merckmanuals.com