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Physical Examination of the Cardiovascular System

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Abstract

The physical examination, while frequently not performed well, is critical to the diagnosis and management of cardiovascular disorders. This paper describes a basic cardiovascular physical examination and explains findings, with the goal of improving skills in this area.

Keywords

Physical examination, Murmurs, Clicks, Valsalva maneuver

Introduction

Cardiac physical examination skills have waned [1]. While portable ultrasound can aid in the accuracy of the bedside cardiovascular evaluation, the cardiac physical is cheap, of diagnostic value, and establishes rapport between patient and physician [2].

First, it is important to be systematic. Second, form a differential diagnosis before you start, so the physical will help rule in/out the possible diagnoses. In addition, try to correlate all information e.g. if the patient has an Electrocardiogram (ECG) with a right-bundle-branch block, you should hear a wide split second heart sound (S2). Thus, accuracy in the examination is best achieved by evaluating the physiologic variables that characterize cardiac function (pulse amplitude, blood pressure, jugular venous pressure, and makers of neurohumoral activation), and the identification of which cardiac chambers are involved using precordial motion and the electrocardiogram [3]. Finally, innocent murmurs in childhood are not normally found in adults, except for pregnant women whose blood viscosity and velocity resemble children's; when significant murmurs are found in nonpregnant adults, echocardiography is prudent [4].

An important issue that arises during the physical examination is how to distinguish physiological signs from similar pathological signs. For example, in an asymptomatic patient with a 1/6–2/6 short early- or mid-peaking systolic murmur at the left sternal border, this is usually benign and simply represents a physiological flow murmur [5]. In contrast, holosystolic and long systolic murmurs are more significant [6].

In addition, the effect of Valsalva, squatting, and hand grip maneuvers on different physiological parameters influencing preload, afterload, chamber dimensions, and pressure gradients will have specific and predictable effects on true pathological murmurs [5]. Another important aspect of helping to distinguish pathological murmurs is their sound distribution on the chest wall. A recent study noted that when diagnosing systolic murmurs, the most important physical finding may also be the distribution of sound on the chest wall with respect to the 3rd left parasternal space [6]. For instance, aortic valve murmurs radiate symmetrically above and below the 3rd left parasternal space, in an oblique direction to both sides of the sternum, in a pattern sometimes resembling a sash worn over the right shoulder ("broad apical-base" pattern) [6]. In other words, the radiation pattern of the murmur may provide additional clues to its etiology. Learning this will take much time and practice in order to distinguish normal distribution from abnormal radiation suggesting true pathology.

Also, a patient with pericardial effusions, even if small, will result in a diminishing of the frequency of all murmur patterns [6].

Finally, additional classic cardiovascular findings can aid one in refining their differential diagnosis, but these findings are sometimes absent, thus illustrating both the value of the bedside examination and its limits [6].

The reader is recommended to several excellent online training sites including: Heart Sounds - Easy Auscultation (http://www.easyauscultation.com/heart-sounds), Heart Sounds and Murmurs - Practical Clinical Skills (http://www.practicalclinicalskills.com/heart-sounds-murmurs.aspx), The Auscultation Assistant (https://www.med.ucla.edu/wilkes/intro.html), and Blaufuss Multimedia - Heart Sounds and Cardiac Arrhythmias (http://www.blaufuss.org/)

Cardiovascular Physical Examination

After initial inspection, I recommend the following order: hands, head, neck, heart, chest, abdomen, & lower limbs; for auscultation, start supine, then left lateral decubitus position, and finally sit or stand the patient up. The core findings and their associations are listed in Tables 1-8, while a brief explanation follows.

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Table 1: General inspection

Physical Finding	Associated cardiac condition(s)
Peripheral cyanosis	Heart failure, vasoconstriction
Central (circumoral) cyanosis	Congenital heart disease, corpulmonale, right-to-left shunting (cardiac or extracardiac), inadequate pulmonary oxygenation of blood
Differential cyanosis (hands/ fingers pink, feet/toes cyanotic)	Patent ductusarteriosus with pulmonary hypertension & a reversed shunt, transposition of the great arteries, pulmonary hypertension, preductal narrowing of aorta
Orthopnea (unable to lie flat) - patient propped up with >=2 pillows to remain comfortable	Congestive heart failure
Turners Syndrome (dwarfism especially a short stature girl with webbed neck)	Aortic coarctation, bicuspid aortic valve
Marfan's syndrome (arm span>body height)	Aortic regurgitation, mitral valve prolapse
Downs syndrome	Atrial septal defect, ventricular septal defect
Cafe-au-lait spots & mental retardation (Watson's syndrome)	Pulmonary stenosis
Cheyne-Stokes respirations (oscillation of ventilation between apnea and hyperpnea)	Congestive heart failure
Kaposi sarcoma, painful mucocutaneous ulcers, lipodystrophy (HIV patient)	Pericarditis, myocarditis, cardiomyopathy, pulmonary hypertension, valvular disease, coronary artery disease
Low resting oxygen saturation (<93%) on a pulse oximeter on room air	Hypoventilation, ventilation-perfusion mismatch, right-to-left shunt, diffusion limitation, reduced inspired oxygen tension.
Low oxygen saturation corrects with 100% oxygen	Physiological shunting: hypoventilation, pulmonary edema, cardiogenic shock (anatomical shunts don't correct)

Inspection - general

Cyanosis is a bluish discoloration of the skin and mucous membranes resulting from abnormal perfusion by either an increased amount of reduced hemoglobin or abnormal hemoglobin. Peripheral cyanosis is commonly due to cutaneous vasoconstriction secondary to exposure to cold air or water, or from hyperadrenergic states including severe heart failure; the later leads to pallor and coldness of the extremities, and cyanosis of the digits when severe. In contrast, central cyanosis is characterized by decreased arterial oxygenation (in Caucasians arterial saturation <= 85%) [7]. It is the absolute amount of reduced hemoglobin that produces the blue discoloration; hence patients with polycythemia vera become cyanotic at higher levels of arterial saturation, whereas those with anemia may not manifest cyanosis despite marked arterial desaturation until it is severe [7] (Table 1).

Inspection - focused

Enlargement of the aorta may stretch the left recurrent laryngeal nerve as it passes around the ligamentum arteriosum (the embryonic remnant of the ductus arteriosus, located between the pulmonary artery and distal aortic arch), resulting in hoarseness.

Clubbing refers to the swelling of the soft tissue of the terminal phalanx of a digit with subsequent loss of the normal angle between the nail and the nail bed [8] (Table 2).

Palpation - extremities

Inspiration causes the intrathoracic pressure to become more negative, facilitating venous return to the right heart, transiently increasing right ventricular volume; this causes a leftward bulging of the interventricular septum, which slightly limits left ventricular filling, resulting in a small decline in left ventricular output and systolic blood pressure. Many variables influence heart rate [9]. In cardiac tamponade, because the high pressure in the surrounding pericardial fluid limits the total volume shared by the two ventricles, this normal physiological response is exaggerated (pulsus paradoxus), and is a relatively sensitive finding [10]. This biventricular interdependence (series and parallel) plays an important role in the inspiratory decrease in left ventricular stroke volume that results in this physical finding [11]. Further, early recognition of pulsus paradoxus in the emergency room can help to rapidly diagnose and manage cardiac tamponade [11] (Table 3).

Table 2: Focused inspection

Physical Finding	Associated cardiac condition(s)
Head & Neck	
De Musset sign (head bobbing)	Aortic regurgitation
Hoarseness	Pericardial effusion, thoracic aneurysm
Hiccups	Pericardial effusion (phrenic nerve stimulation)
Eyes	
Dislocated lens / ectopialentis (Marfan's syndrome)	Aortic regurgitation, mitral valve prolapse
Blue Sclera, brittle bones (Osteogenesisimperfecta)	Mitral valve prolapse, aortic dilation/ regurgitation and dissection
Bilateral forward displacement of eyeballs (Graves' ophthalmopathy)	Tachycardia, atrial fibrillation, high output heart failure
Systolic pulsation	Severe tricuspid regurgitation
Chest	
Pectusexcavatum, straight/ flat thoracic spine (Straight-back syndrome, ankylosing spondylitis)	Mitral valve prolapse syndrome, aortic regurgitation
Hands & Legs	
Unilateral digital clubbing	Aortic aneurysm (interferes with blood supply to one arm), brachial arteriovenous fistula, arterial graft sepsis, & hemiplegic stroke
Bilateral digital clubbing	Cyanotic congenital heart disease, neoplastic & suppurative intrathoracic disease, diffuse pulmonary disease, infective endocarditis, inflammatory bowel disease, celiac disease, cirrhosis, thyroid acropachy (extreme autoimmune thyroid disease)
Ulnar deviation, "swan neck" (rheumatoid arthritis)	Aortic regurgitation, mitral regurgitation, pericarditis, cardiomyopathy
Arachnodactyly "spider fingers" long and slender digits (Marfan's syndrome)	Aortic regurgitation, mitral valve prolapse
Xanthoma	Hypercholesterolemia
Systolic blush & diastolic blanch when upward traction on the fingernail (Quincke's pulse)	Aortic regurgitation
Thumb with extra phalanx (Holt-Oram syndrome)	Atrial septal defect
Petechiae	Infective endocarditis, trauma
Painless hemorrhagic lesions on palms or soles (Janeway lesions)	Infective endocarditis
Small tender erythematous skin lesions on pads of the fingers, toes, palms or soles (Osler nodes)	Infective endocarditis
Triphasic (white, blue, red) change of finger color in cool temperatures or emotional stress	Raynaud's phenomenon
Bilateral peripheral edema	Excess extracellular fluid (5 liters or more): heart failure, constrictive pericarditis
Unilateral peripheral edema	Venous obstruction/damage/removal, hemiplegia (on paralyzed side)

Jugular venous pulse

The right internal jugular vein is best for examining the jugular venous pulse (JVP) waveform and estimating central venous pressure. A JVP >8cm is elevated; however, many physicians cannot diagnose heart failure by examining the JVP [12] (Table 4).

Kussmaul's sign reflects an inability of the right sided chambers to accept additional volume, typical of constrictive pericarditis. In diastole, as the blood passes from the right atrium to the right ventricle, the right ventricle size expands and quickly reaches the limit imposed by the rigid constricting pericardium. At that point, further filling is suddenly arrested, venous return to the right heart ceases, and systemic venous pressure rises. In addition, the impaired filling of the left ventricle causes a reduction in stroke volume, cardiac output, and blood pressure falls [13].

Table 3: Palpation of extremities

Physical Finding	Associated cardiac condition(s)
Pulse rate	
Heart rate <60, >100	Sinus bradycardia, sinus tachycardia
Irregularly irregular	Atrial fibrillation
Regularly irregular	Bi-/trigeminy, 2nd degree atrioventricular block
Pulse quality	
Radio-femoral delay	Coarctation (associated with bicuspid aortic valve & aortic stenosis)
Hypokinetic (low volume)	Aortic stenosis, congestive heart failure, restrictive pericardial disease, mitral stenosis
Hyperkinetic (large, bounding)	Complete heart block, anxiety, anemia, exercise, fever, patent ductusarteriosus, peripheral atrioventricular fistula, mitral regurgitation, ventricular septal defect, aortic regurgitation
Collapsing / Corrigan's pulse (full hard pulse followed by a sudden collapse)	Aortic regurgitation, hyperdynamic circulation, atherosclerotic aorta, patent ductusarteriosus, arteriovenous aneurysm
Anacrotic (small volume, slow upstroke)	Aortic stenosis
Pulsusbisferiens (two systolic peaks)	Aortic regurgitation, combined aortic regurgitation & aortic stenosis, hypertrophic cardiomyopathy, patent ductusarteriosus
Dicrotic pulse	Dilated cardiomyopathy
Pulsusparvus (small weak pulse)	Aortic stenosis
Pulsustardus (delayed or slow rising systolic peak)	Aortic stenosis
Pulsusparvus et tardus (plateau pulse)	Severe aortic stenosis
Pulses paradoxus (a decrease in systolic blood pressure of >10 mm Hg during inspiration)	Pulmonary embolism, superior vena cava obstruction, cardiac tamponade, constrictive pericarditis, airway obstruction, hypovolemia, pregnancy, morbid obesity
Pulsusbigeminus (groups of two heartbeats close together followed by a longer pause; the second pulse is weaker than the first)	Ventricular bigeminy, hypertrophic obstructive cardiomyopathy
Pulsusalternans (alternating strong & weak pulses)	Left ventricular failure (hypertension, aortic stenosis, coronary artery disease, dilated cardiomyopathies), during or post paroxysmal tachycardia
Blood pressure	
120–139 or 80–89mmHg	Prehypertension
<140/90mmHg	Goal blood pressure in hypertensive patient
<130/80mmHg	Goal blood pressure in diabetes, chronic kidney disease, known coronary artery disease or equivalent (carotid artery disease, abdominal aortic aneurysm & peripheral vascular disease), or 10-year Framingham risk score of >=10%.
< 120/80mmHg	Goal blood pressure in left ventricular dysfunction (ejection fraction <40%)
Wide pulse pressure (e.g. 180/80)	Aortic regurgitation
Narrow pulse pressure (e.g. 120/100)	Aortic stenosis
Differential in 2 arms >10mmHg	Aortic dissection, aortic coarctation, arterial occlusion/stenosis of any cause

It is easier to see the *x, y descents* in the neck than the positive pressure *a, c, v waves*, because the former produce larger excursions [2]. Palpation of the left carotid artery while examining the right JVP helps adjudicate which pulsations are venous & their timing in the cardiac cycle.

The *a wave* just precedes S1 and represents venous distention due to right atrial contraction near the end of diastole.

The *a wave* becomes more prominent in conditions that in which the right atrium is contracting against increased resistance. Amplified or cannon *a waves* are evident when the right atrium contracts against a closed tricuspid valve.

Table 4: Jugular Venous Pulse (JVP)

Physical Finding	Associated cardiac condition(s)
JVP >8 centimeters (or >3 centimeters above sternal angle) with patient's head tilted 45%	Right heart failure, pericardial effusion/ constrictive pericarditis, tricuspid stenosis, tricuspid regurgitation, fluid overload, superior vena cava obstruction, hyperdynamic circulation
Jugular veins fill during inspiration (Kussmaul's sign)	Constrictive pericarditis, corpulmonale, pulmonary embolism, right ventricular infarction, right heart failure, tricuspid stenosis, cardiac tamponade, acute pulmonary hypertension, severe asthma, tension pneumothorax, & exacerbations in chronic obstructive pulmonary disease
Dominant *a wave*	Tricuspid stenosis or atresia, pulmonary stenosis, pulmonary hypertension, right ventricular hypertrophy, right atrial myxoma
Absent *a wave*	Atrial fibrillation
Cannon *a wave*	Complete heart block, paroxysmal atrial tachycardia, ventricular tachycardia, junctional rhythm
Rapid *x descent*	Cardiac tamponade
Dominant *v wave*	Tricuspid regurgitation (as in primary pulmonary hypertension), atrial septal defect (also prominent *y descent*)
Rapid *y descent*	Constrictive pericarditis, severe right heart failure, severe tricuspid regurgitation
Slow *y descent*	Tricuspid stenosis, right atrial myxoma
Blunted *y descent* (or *x descent* more prominent than *y descent*)	Cardiac tamponade

Table 5: Palpation of chest and abdomen

Physical Finding	Associated cardiac condition(s)
Apex beat	
Thrusting / heaving (parasternal)	Left ventricular hypertrophy
Sustained	Hypertrophic cardiomyopathy
Tapping / palpable S1	Mitral stenosis
Double systolic apical impulse	Dyskinetic or aneurysmal left ventricle, hypertrophic cardiomyopathy
Displaced lateral to mid-clavicular line	Dilated cardiomyopathy, aortic regurgitation, mitral regurgitation
Absent apex beat	Pericardial effusion, emphysema, obesity
Left parasternal lift	Severe mitral regurgitation, left atrial enlargement, or ascending thoracic aortic aneurysm
Right parasternal lift	Severe tricuspid regurgitation, right ventricular hypertrophy
Systolic apical thrill	Mitral regurgitation
Diastolic apical thrill	Mitral stenosis
Left parasternal thrill (Intercostal space 3 or 4)	Ventricular septal defect
Systolic basal thrill	Aortic stenosis, pulmonary stenosis
Diastolic basal thrill	Aortic regurgitation, pulmonary regurgitation
Chest wall	
Local costochondral& muscle tenderness (with direct pressure over joint)	Costal chondritis
Pain localized to swollen costochondral&costosternal joints	Tietze's syndrome
Abdomen	
Pulsatile liver	Tricuspid regurgitation
Right upper quadrant tenderness	Acute onset of right heart failure
Hepatomegaly	Right heart failure, tricuspid regurgitation, constrictive pericarditis
Hepatojugular / abdominojugular reflux - Sustained rise in jugular venous pulse on pressing the right upper quadrant or central abdomen for >=10 seconds	Right heart failure, tricuspid regurgitation, constrictive pericarditis

The *c wave* corresponds to tricuspid valve closure & bulging into the right atrium with onset of right ventricular systole.

The *x descent* is from right atrial relaxation & the downward descent of the base of the atrium & tricuspid valve during right

ventricular systole.

The *v wave* is produced by right atrial filling during right ventricular systole when the tricuspid valve is closed, and almost coincides with the S2.

The *y descent* is rapid and deep since opening of the tricuspid valve in early diastolic right ventricular filling is unimpeded. Constrictive pericarditis occasionally has prominent *x descent* in addition to the rapid *y descent* which leads to a "W" shaped JVP. A slow *y descent* suggests an obstruction to tricuspid valve filling.

In cardiac tamponade, the intracardiac diastolic pressures are elevated and equal, ventricular filling is impaired, and cardiac output declines. The pericardial fluid compresses the right ventricle, and prevents its rapid expansion. The right atrium cannot empty quickly, and the *y descent* is blunted or absent [10]. Thus, the *x descent* is most prominent.

Palpation – chest and abdomen

The most inferolaterally palpable beat with the patient supine and in the left lateral position is the apex beat or impulse. It's usually at or medial to the left midclavicular line in the fourth or fifth intercostal space and is a tapping, early systolic outward thrust localized to a point about 2 finger tips in size. It is primarily due to recoil of the heart as blood is ejected (Table 5).

With a left parasternal lift, the pulsation occurs distinctly later than the left ventricular apex beat, is synchronous with the *v wave* in the left atrial pressure curve, and is due to anterior displacement of the right ventricle by an enlarged, expanding left atrium. In right parasternal lift, there is a similar impulse to the left one, it occurs to the right of the sternum in some patients with severe tricuspid regurgitation and a massive right atrium.

Thrills are palpable, low-frequency vibrations felt when your hand touches the chest wall, usually associated with heart murmurs.

Auscultation – heart sounds

Most acoustic stethoscopes have a (1) diaphragm or plastic disc: the underlying sound waves vibrate the diaphragm, creating acoustic pressure waves which travel up the tubing to the listener's ears; best for higher frequency sounds ≥200Hz; & (2) bell or hollow inverted cup: the vibrations of the skin directly produce acoustic pressure waves; best for low frequency sounds (<200 Hz) [14] (Table 6).

Regarding timing, at a resting heart rate of 75beats/minute, ventricular systole lasts 0.30 seconds and diastole 0.50 seconds. Depending on the frequency and amplitude of the sound, the human ear may not distinguish separate sounds that are 0.01-0.02 seconds or less apart [15].

First heart sound "the lub": The first heart sound (S1) consists of a first component of mitral valve closure and a second component from tricuspid valve closure, and is heard the loudest between the left lower sternal border and apex with the stethoscope diaphragm firmly pressed. During systolic contraction, when the left ventricular pressure exceeds that in the left atrium, the mitral valve closes. Physiological splitting of the two high-pitched components of S1 by up to 0.03 seconds exists mainly in early youth according to some published articles is a normal phenomenon, but is not always distinguished [15].

Wide audible splitting of S1 (up to 0.06 seconds) is usually abnormal, and may occur due to delay in the onset of the right ventricular pressure pulse and thus delay in closure of the tricuspid valve, which may occur in in patients with right bundle branch block, Ebstein's anomaly, or right atrial myxoma.

In reversed splitting of S1, the mitral component follows the tricuspid component.

S1 is louder if diastole is shortened (tachycardia), if atrioventricular flow is increased (high cardiac output), prolonged (mitral stenosis), or if atrial contraction precedes ventricular contraction by an unusually short interval (short PR interval).

A soft S1 may be due to poor conduction of the sound through the chest wall, a slow rise of the left ventricular pulse, a long PR interval, or imperfect closure of the mitral valve due to reduced valve substance, as in mitral regurgitation. S1 is also soft when the anterior mitral leaflet is immobile because of rigidity, even in the presence of predominant mitral stenosis.

Second heart sound "the dup": The second heart sound (S2) is split into audibly distinct aortic (A2) and pulmonic (P2) components. Normal physiologic splitting widens with inspiration because the increased right heart volume takes longer to empty, the maximal split being 0.03 seconds. Splitting is heard best at the base of the heart (left/right upper sternal border) with the stethoscope diaphragm firmly pressed.

Splitting that persists with expiration is usually abnormal when the patient is in the upright position. Fixed splitting of S2 occurs in atrial septal defect due to delayed closure of the pulmonic valve.

Table 6: Auscultation – heart sounds

Physical Finding	Associated cardiac condition(s)
First heart sound (S1)	
Loud S1	Mitral stenosis, tricuspid stenosis, Lown-Ganong-Levine syndrome, tachycardia
Soft S1	Mitral regurgitation, severe congestive heart failure, calcified mitral valve, left bundle branch block, long PR interval (1st degree atrioventricular block)
Widely split S1	Right bundle branch block, Ebstein's anomaly, right atrial myxoma
Reversed splitting of S1	Severe mitral stenosis, left atrial myxoma, left bundle branch block
Variable intensity S1	Atrial fibrillation
Second heart sound (S2)	**Aortic valve closure (A2) and Pulmonary closure (P2)**
Soft / absent A2	Severe aortic stenosis
Loud S2 - Loud A2	Systemic hypertension
Loud S2 - Loud P2	Pulmonary hypertension
Reduced splitting of S2	Pulmonary hypertension
Increased splitting of S2 - early A2	Mitral regurgitation
Increased splitting of - late P2: - electrical delay of P2	Right bundle branch block
Increased splitting of - late P2: - mechanical delay of P2	Pulmonary stenosis, ventricular septal defect, obstruction right ventricle, right ventricular failure, mitral regurgitation (with pulmonary hypertension)
Fixed splitting of S2	Atrial septal defect
Paradoxically split S2 - electrical delay of A2	left bundle branch block, right ventricular pacing, right ventricular ectopic beat (delayed excitation of left ventricular systole)
Paradoxically split S2 - mechanical delay of A2	Severe aortic outflow obstruction (aortic stenosis), systolic hypertension, large aorta-to-pulmonary artery shunt, ischemic heart disease, cardiomyopathy, aortic coarctation, patent ductusarteriosus
Single S2 (absence of physiologic splitting)	Tetralogy, truncusarteriosus, tricuspid atresia
Muffled heart sounds	Pericardial effusion
Third heart sound (S3)	
S3 present, 0.14–0.16 seconds after S2	Ventricular septal defect, atrial septal defect, aortic regurgitation, mitral regurgitation, tricuspid regurgitation, patent ductusarteriosus, pregnancy, congestive heart failure, hyperdynamic circulation (fever, anemia, atrioventricular fistula, thiamine deficiency, hyperthyroidism, infection, Paget's disease, pregnancy), physiological < 40 years old
Fourth heart sound (S4)	
S4 present, 0.08-0.12 seconds before S1	Hypertension (systemic or pulmonary), hypertrophic cardiomyopathy, acute myocardial infarction, coronary artery disease, congestive heart failure, aortic stenosis, pulmonary stenosis

Table 7: Auscultation – other sounds

Physical Finding	Associated cardiac condition(s)
Early Systolic Clicks (Ejection Sounds)	
High frequency systolic ejection clicks, 0.09 to 0.14 seconds after first heart sound (S1)	Aortic stenosis (bicuspid aortic valve), pulmonary stenosis, pulmonary hypertension, dilated pulmonary artery, left ventricular outflow obstruction
Midsystolic Clicks (Nonejection sounds)	
Medium-to-high frequency clicks, 0.17 to 0.27 seconds after S1	Mitral valve prolapse (& associated late systolic murmur), tricuspid valve prolapse, nonmyxomatous mitral valve disease, adhesive pericarditis, atrial myxoma, atrial septal aneurysms, left ventricular aneurysm
Early Diastolic Opening Snap (OS)	
High-frequency sound, 0.04 to 0.12 seconds after second heart sound (S2)	Mitral stenosis, tricuspid stenosis
Early-Mid Diastolic Tumor Plops	
Low frequency sound, 0.04 to 0.12 seconds after S2	Atrial myxoma
Early-Mid Diastolic Pericardial Knocks	
Pericardial knock, 0.06 to 0.14 seconds after S2	Constrictive pericarditis

Since the capacitance of the pulmonary bed is greatly increased, right ventricular stroke volume is not appreciably influenced by respiration. Upon inspiration, augmentation of the systemic venous return is counterbalanced by a reciprocal decrease in the volume of the left-to-right shunt, such that right ventricular filling and the timing of P2 relative to A2 does not change, resulting in a fixed split.

Third heart sound: The third heart sound (S3) or ventricular gallop arises from the sudden termination of excessive early rapid diastolic filling & stretching of the left ventricle at the time of the atrioventricular valve opening, with timing like the "-ky" in "Ken-tuc-ky." [2]. An S3 is a dull thud lower in pitch than S1 or S2, and is best heard in the left lateral position with the bell at the apex during expiration (left-sided S3) or at the left sternal border/sub-xiphoid during inspiration (right-sided S3).

The S3 is a barometer of heart failure decompensation: its presence indicates high filling pressures, its absence reflecting improved filling pressures [16].

Fourth heart sound: The fourth heart sound (S4) or atrial gallop is a low-pitched short thud (but higher pitched than S3), presystolic sound produced in sinus rhythm during atrial systole with ejection of a jet of blood against a stiff or non-compliant ventricle, usually having elevated ventricular end-diastolic pressure [17]. It precedes S1 & S2 like "Ten-" in "Ten-nes-see," and is best heard at apex using the bell and with patient in left lateral decubitus position [2]. It is accentuated by mild isotonic or isometric exercise in the supine position.

Auscultation – other sounds

Ejection sounds are sharp, high-pitched click(s) occurring in early systole and closely following S1. They may be aortic or pulmonic in origin, require a mobile valve for their generation, and begin at the time of maximal valve opening. Frequently, the valve is abnormal, and the ejection sound is valvular; this sound is generated by the halting of the doming of the valve. If the valve associated with the ejection sound is normal, it is called a vascular ejection sound. The pulmonic ejection sound, loudest in the 2nd left intercostal space, is the only right-sided sound that is softer during inspiration. With inspiration, increased venous return augments right atrial systole, resulting in partial opening of the pulmonic valve before right ventricular systole commences [18] (Table 7).

Midsystolic clicks may be single or multiple, and probably result from chordae tendineae that are functionally unequal in length on either or both atrioventricular valves and are heard best along the lower left sternal border and at the left ventricular apex [2].

The opening snap (OS) is a brief, crisp, high-frequency, early diastolic sound, due to stenosis of an atrioventricular valve, most

Table 8: Auscultation – murmurs

Physical Finding	Associated cardiac condition(s)
Timing	
Early systolic	Ventricular septal defect, acute mitral regurgitation, acute tricuspid regurgitation
Holosystolic (pansystolic)	Mitral regurgitation, tricuspid regurgitation, ventricular septal defect
Midsystolic (ejection systolic)	Aortic stenosis, pulmonary stenosis, hypertrophic obstructive cardiomyopathy, atrial septal defect, aortic coarctation, pregnancy, mammary soufflé, innocent murmur
Late systolic	Myocardial infarction, ischemia, diffuse myocardial disease, mitral regurgitation from mitral valve prolapse
Early diastolic	Aortic regurgitation, pulmonary regurgitation (± Graham Steell murmur)
Middiastolic	Mitral stenosis, tricuspid stenosis, atrial myxoma (right or left), acute severe aortic regurgitation (Austin-Flint murmur), acute rheumatic fever (Carey Coombs murmur)
Presystolic (Late diastolic)	Tricuspid stenosis, mitral stenosis, atrial myxoma (right or left), acute severe aortic regurgitation (Austin-Flint murmur)
Continuous	Patent ductusarteriosus, cervical venous hum, mammary soufflé, congenital or acquired arteriovenous shunt (e.g. coronary arteriovenous fistula, ruptured aneurysm of aortic sinus of valsalva into a right heart chamber, anomalous left coronary artery, intercostal arteriovenous fistula), small atrial septal defect with a high left atrial pressure, proximal coronary artery stenosis, pulmonary artery branch stenosis, bronchial collateral circulation, aortic coarctation
Modulation (shape)	
Diamond (crescendo-decrescendo)	Aortic stenosis, pulmonary stenosis, hypertrophic obstructive cardiomyopathy
Decrescendo	Aortic regurgitation, pulmonary regurgitation
Plateau	Mitral regurgitation, tricuspid regurgitation
Location	
5th intercostal space mid-clavicular line / apical	Mitral stenosis/regurgitation, hypertrophic obstructive cardiomyopathy
Right 5th interspace	Tricuspid stenosis/regurgitation
Right 2nd interspace / base	Aortic stenosis/regurgitation
Right 1st interspace or higher	Supravalvular aortic stenosis
Right supraclavicular fossa	Cervical venous hum
Left 2nd interspace / upper sternal border	Pulmonic stenosis/regurgitation, patent ductusarteriosus
Left 3rd-4th interspace	Tricuspid regurgitation, hypertrophic obstructive cardiomyopathy
Left & Right of sternum, 4th-6th interspace	Ventricular septal defect
Back/ interscapular	Patent ductusarteriosus, aortic coarctation
Intensity	
1	Faint, must tune in
2	Easily heard
3	Moderately loud
4	Palpable thrill and loud
5	Very loud
6	Heard with stethoscope off chest
Frequency (pitch)	
High	Mitral regurgitation, acquired pulmonary regurgitation, aortic regurgitation
Low	Mitral stenosis (rumble), tricuspid stenosis, congenital pulmonary regurgitation, acute severe aortic regurgitation

Radiation	
Axillary	Mitral regurgitation (anterior or laterally directed jet)
Back / Subscapular	Mitral regurgitation (posteriorly directed jet), patent ductusarteriosus, aortic coarctation
Neck (Carotids)	Aortic stenosis, hypertrophic obstructive cardiomyopathy, supravalvular aortic stenosis (louder in right neck)
Quality	
Blowing	Mitral regurgitation
Varying throughout cycle	Pericarditis (Pericardial friction rub)
Maneuver	**Murmur that becomes louder**
Squatting, raising legs i.e. increase venous return (left ventricular volume)	Aortic stenosis, aortic regurgitation, mitral stenosis, mitral regurgitation, ventricular septal defect, patent ductusarteriosus
Valsalva, inhalation of amyl nitrate, sitting up, standing i.e. decrease left ventricular volume	Mitral valve prolapse (& lengthens murmur), hypertrophic obstructive cardiomyopathy
Handgrip, phenylephrine, or transient arterial occlusion by inflation of bilateral arm cuffs to 20mmHg above systolic blood pressure for 5 seconds (increases systemic arterial resistance)	Mitral regurgitation, aortic regurgitation, ventricular septal defect
Holosystolic louder in inspiration	Tricuspid regurgitation (Carvallo's sign), Pulmonary stenosis, pulmonary regurgitation
Following a premature beat or a long RR interval	Aortic stenosis, pulmonary stenosis
Extra signs	
Other physical findings to support your diagnosis	Blood pressure, jugular venous pulse, thrill, apex beat, peripheral pulse characteristics, changes in first or second heart sound, presence of third or fourth heart sound
Pleural effusion	Left heart failure
Inspiratory rales	Left heart failure
Pulsatile mass below umbilicus	Abdominal aortic aneurysm
Abdominal bruit (lateral)	Renovascular hypertension

often the mitral valve. It is generated when the systolic "bowed" anterior mitral leaflet suddenly changes direction toward the left ventricle during diastole "dome" secondary to the high left atrial pressure. It is heard best with the stethoscope diaphragm at the lower left sternal border and radiates well to the base of the heart. With severe mitral stenosis, greater left atrial pressure causes the mitral valve leaflets to dome sooner, allowing less distance for the leaflets to move in early diastole, and so the opening snap occurs earlier (shorter S2-OS interval); this corresponds with left ventricular isovolumic relaxation time. In general, the longer the diastolic murmur lasts, the more severe the mitral stenosis; this corresponds to a longer duration of the diastolic pressure gradient across the mitral valve [19].

When a large atrial myxoma moves into the region of the mitral or tricuspid valve orifice and obstructs atrioventricular flow during diastole, a tumor plop may be heard in up to 50% of cases [20]. The tumor plop has the same timing as the mitral OS, but differs in that it is a low frequency sound, heard best with the stethoscope bell.

A pericardial knock is a discrete and loud high pitched sound heard in early-mid diastole, occurring slightly earlier than S3 [13]. It is produced when the rapid early diastolic filling of the left ventricle suddenly halts due to the restrictive effect of the rigid pericardium [13].

Auscultation – murmurs

Murmurs are caused by rapid, turbulent blood flow, usually through damaged valves, which causes vibrations which are then acoustically transmitted as sound [21]. During stenosis, blood is forced through a narrow opening, at high speed, causing substantial turbulence and associated murmur. During regurgitation, the valve is prevented from closing fully, which allows blood to spurt backward, and a blowing or hissing sound is heard [21] (Table 8).

Timing

Early systolic murmurs begin with S1 and end in midsystole.

Holosystolic (pansystolic) murmurs begin before aortic ejection (early in contraction) at S1 and end when relaxation is almost complete after S2, and are generated when there is flow between two chambers that have widely different pressures throughout systole, such as the ventricle and either its atrium or the other ventricle (ventricular septal defect, VSD). Carvallo's sign, an increase in the intensity of the pansystolic murmur of tricuspid regurgitation during or at the end of inspiration, in found in most patients with severe tricuspid regurgitation [22]. The murmur of VSD does not vary with respirations and does not radiate to the axilla so can be differentiated from tricuspid regurgitation (Carvallo's sign) and mitral regurgitation, respectively.

Midsystolic (ejection systolic) murmurs starts shortly after S1 and occur when the ventricular pressure becomes high enough to exceed the outflow tract pressure thus forcing the semilunar valve open [23,24]. Most benign (innocent) functional murmurs are midsystolic and originate from the pulmonary outflow tract.

Late systolic murmurs are faint or moderately loud, high-pitched apical murmurs that start well after ejection and do not mask either heart sound, and are probably related to papillary muscle dysfunction caused by ischemia/infarction of these muscles or to their distortion by left ventricular dilation.

Early diastolic murmurs begin with S2. In severe acute aortic regurgitation, the murmur often is lower pitched and shorter in duration than the murmur of chronic aortic regurgitation because the lower pressure difference between the aorta and the left ventricle in diastole. When pulmonic regurgitation develops in the setting of pulmonary hypertension, the murmur begins with a loud P2 and may last throughout diastole (Graham Steell murmur) [25].

Middiastolic murmurs begin at a clear interval after S2 during early ventricular filling, usually arise from the mitral or tricuspid valves, and are due to a mismatch between a decreased valve orifice size and an increased flow rate. The Austin-Flint murmur is a murmur of relative mitral stenosis caused by narrowing of the mitral orifice by the severe aortic regurgitation stream hitting the anterior mitral valve leaflet [26]. The Carey Coombs murmur is a soft blubbering apical middiastolic murmur occurring in the acute stage of rheumatic mitral valvulitis, arising from inflammation of the mitral valve cusps or excessive left atrial blood flow secondary to mitral regurgitation [2].

Presystolic (late diastolic) murmurs begin immediately before S1 during the period of ventricular filling that follows atrial contraction. They are usually due to atrioventricular valve stenosis and have the same quality as the middiastolic filling rumble, but they are usually crescendo, reaching peak intensity at the time of a loud S1. The presystolic murmur corresponds to the atrioventricular valve gradient, which is minimal until the moment of right or left atrial contraction.

Continuous murmurs begin in systole, peak near S2, and continue without interruption through S2 into part or all of diastole. These murmurs result from continuous flow due to a communication between high and low pressure areas that persist through the end of systole and the beginning of diastole. The prototype continuous murmur is patent ductus arteriosus, which is 'machinery-like' & peaks just before or after S2, decreases in late diastole, and may be soft or absent prior to S1.

Modulation (shape)

Modulation refers to the pattern that a murmur makes on a phonocardiogram in or electronic stethoscope display.

Location

The location of the stethoscope on the chest where sounds are loudest can aid in diagnosis.

When the murmur of aortic regurgitation radiates selectively to the right sternal border (3rd & 4th intercostal spaces), it suggests that aortic root dilatation is the cause.

Intensity

Initially, stenotic murmurs get louder as the stenosis gets worse; when the ventricle starts to fail and/or the leaflet motion becomes significantly reduced, the murmur quietens. Paradoxically, with regurgitant murmurs, small high pressure regurgitation may be intense, whereas a wide open regurgitation (less turbulence) may be faint.

Frequency (pitch)

The murmur of acquired pulmonary regurgitation is a high-frequency diastolic blow along the left sternal border. The murmur of congenital pulmonary regurgitation is a low- to medium-pitched, decrescendo murmur heard along the left sternal border, which peaks shortly after P2.

Radiation

The direction of the high velocity jet of blood may transmit sound to certain locations which help diagnosing the sources.

Quality

A Pericardial rub (scratchy) is best appreciated with the patient upright and leaning forward and may be accentuated during inspiration. It has three components related to (1) atrial systole, (2) ventricular systole, and (3) ventricular diastole (with an approximate timing around S3). A friction rub that had been present during the acute phase of pericarditis may disappear if a large effusion separates the inflamed layers from one another.

Maneuvers making murmur louder

I recommend regularly performing and becoming familiar with those maneuvers that increase the intensity of the murmur.

The valsalva maneuver, deep inspiration followed by forced expiration against a closed glottis for 20 seconds, reduces the intensity of most murmurs by diminishing both right and left ventricular filling (ventricular preload). Specifically, during the strain phase, intrathoracic pressure increases leading to decreased venous return to the right heart, which leads to decreased left ventricular filling, resulting in a decreased cardiac output, so most murmurs decrease in intensity [27].

In mitral valve prolapse, when end-diastolic volume is decreased such as with standing or valsalva maneuver, the critical volume is achieved earlier in systole and the click-murmur complex occurs more quickly after the first heart sound.

In hypertrophic obstructive cardiomyopathy, actions that reduce the left ventricular size, such as standing or the Valsalva maneuver, bring the anterior mitral leaflet and the interventricular septum into closer proximity, thus obstructing the left ventricular outflow tract and intensifying the murmur.

Inspiration increases systemic venous return to right heart and increases right sided murmurs.

Extra Signs

These are all the other clues that together lend credence to your diagnosis. For instance, one might expect to see the constellation of pulsatile liver, prominent jugular venous *v waves*, and a pansystolic murmur that increases with inspiration (Carvallo sign) in a patient with severe tricuspid regurgitation [22].

Conclusion

Basic cardiovascular physical examination is a skill that is improved with training and practice. This skill has numerous benefits including establishing a bond between patient and physician, following and managing a patients clinical condition, as well as being very cost effective when done well. Using a systematic manner, and constantly reviewing the possible diagnosis while hunting for clues, the physical examination can become an exciting and elucidating part of clinical medicine.

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