The physiology of cardiac auscultation

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There has been a reduction in the skills and abilities of medical students and residents to diagnose and interpret heart sounds and murmurs. This loss has occurred despite new and sophisticated teaching modalities including graphic instruction, digital recordings, and computer-enhanced simulation [1–3]. Although the reason for reduction in these skill sets is not entirely clear, the increased availability and promotion of newer diagnostic modalities, a reduction in emphasis on auscultation instruction [4], a lack of physician confidence, and increased concern about litigation may be involved in the increased frequency of ordering of additional imaging modalities. Auscultation by a skilled practitioner enables diagnosis in the majority of pediatric cases referred for evaluation of a cardiac murmur.

In childhood the great majority of cardiac murmurs are innocent [5,6]. Although the ECG may rule out significant anatomic structural heart disease, the only definitive way of diagnosing an innocent heart murmur is by auscultation. Thus, it is important that pediatricians be capable of auscultation both as a time- and cost-saving modality and to provide accurate diagnosis, engender patient confidence, provide an explanation of presumed cause, and to provide appropriate instruction concerning lifestyle changes and endocarditis prophylaxis.

Frequently, individuals are inappropriately referred for diagnostic studies alone, most commonly ECG. The procedure is not cost ineffective as a preliminary diagnostic study [7], and parents may not be provided with appropriate feedback. Although they are often informed that no abnormality was detected, parents are often left wondering about the significance of the murmur with re-
gards to exercise, participation in competitive activities, and endocarditis prophylaxis. In addition, patients referred for echocardiographic evaluation of cardiac murmurs are often in the age range in which conscious sedation, with the attendant risks, is necessary to obtain quality diagnostic studies [8]. If sedation is not employed in the uncooperative patient, and the imaging studies are incomplete or technically poor (as can happen in ECG laboratories that infrequently perform pediatric studies), mistaken interpretations may occur, and significant disease may be missed or dismissed.

Primary care physicians are in the best position to provide accurate diagnosis and reassurance. Not all cardiac murmurs warrant referral. Improved physiologic understanding of the physics of sound and murmur creation should provide the foundation for improved skills and confidence in the description and interpretation of pediatric heart sounds and murmurs.

What is sound?

Sound is the sensation of hearing resulting from stimulation of the auditory nerves by vibrations transmitted in a medium. Vibrations are caused by the rhythmic compression and rarefaction of molecules within a medium (Fig. 1). The transmission characteristics, speed, and fidelity of these rhythmic vibrations or waves vary both within and at interfaces between different media, generally air or water. The eardrum receives and responds to these pressure waveforms by converting them to minute kinetic movements conducted by the ossicular chain within the middle ear. The ossicular chain serves to amplify or attenuate the vibratory signal before it is conducted to the oval window of the cochlea of the inner ear. Within the inner ear, movement of lymphatic fluids on either side of Reissner’s membrane stimulates fine hair cells of the organ of Corti, resulting in

![Fig. 1. The dimensions of sound. Sound represents compression and rarefaction of air molecules.](image_url)
ionic fluxes and neurotransmission of signals to the cerebral cortex. Sound waves have three dimensions: intensity, frequency, and timbre or quality (Fig. 2).

**Intensity or loudness**

The crest of a pressure wave has a given height or amplitude (Fig. 2). This amplitude determines the perception of loudness. There is a large range of amplitudes over which sounds can be heard. The loudest sounds cause pain and have amplitudes more than 1 million times greater than the quietest audible sounds. To accommodate this enormous range, sound amplitudes are usually measured on a logarithmic scale. The unit of measurement is the decibel (dB). Sound intensity or the subjective perception of loudness varies with the square of the sound amplitude. Sound intensity is measured by comparing it to some reference value (usually the softest sound intensity that humans can hear). On a decibel scale, sound measured at 10 decibels has 10 times the intensity of the reference value. At 20 decibels the intensity is 100 times greater, and at 30 decibels, it is 1000 times greater than the reference value.
Frequency or pitch

The distance from the crest of one pressure wave to the next is the wavelength (Fig. 2). This wavelength depends on the speed at which sound can travel through a given medium and the temperature. The number of pressure waves per second is the frequency and is measured in Hertz (Hz). With shorter wavelengths there are more pressure waves per second, and the frequencies are higher. Conversely, with longer wavelengths the frequencies are lower. The frequency of a pressure wave determines the perception of pitch; low frequencies are heard as a low pitch and high frequencies as a high pitch. The range of frequencies over which humans can perceive sound is the audible range. It extends from a minimum of 20 to a maximum of 20,000 Hz. The human ear is most sensitive to sounds between 500 and 4000 Hertz. This range contains most frequencies of normal speech. The majority of heart sounds and murmurs range between 5 and 800 Hz. Low frequencies are not heard as well and need larger amplitudes to sound equally loud. Note that many sounds emanating from the heart are below the threshold of audibility and consequently are best perceived by palpation.

Quality or timbre

Besides frequency and intensity, other aspects influence the perception of a sound or murmur. A guitar string, for example, vibrates not only along its whole length but also in segments of halves, thirds, fourths, and so forth. Each segment then vibrates twice, three times, four times, and so forth, as fast as the whole string. The vibration of the whole string produces the fundamental or primary frequency; the weaker amplitudes of the fundamental frequency or secondary frequencies make up the harmonics. The same musical note played on different instruments sounds distinctly different. The timbre or quality of a sound refers to the component parts of a complex waveform. These multiple accessory waveforms or overtones differentiate the character of musical instruments playing a musical note of a similar cycle length. Thus, it is overtones that differentiate a violin or piano playing the same note (eg, middle C at a cycle length of 256 Hz). Although a variety of qualitative terms have been employed to describe heart murmurs (eg, “rumbling,” “machinery,” or “blowing”) there are only two fundamental qualities of sound: music and noise.

Harmony or music

The individual characteristics of the sound depend on the number and relative amplitude of the overtones. When the overtones are multiples of the primary frequency, they create a pleasing sound termed “harmony” or “music.” For example, if the primary note is middle C (256 Hz), music is produced by overtones at cycle lengths of 512, 1024, and 2028 Hz. An awareness of harmonics and overtones will help in understanding the musical quality of some cardiac murmurs.
Dissonance or noise

When a sound wave is composed of many different and unrelated frequencies, a musical quality can no longer be perceived. Instead the sound wave seems dissonant and rough and is described as “noise.” Noise can originate from variable amounts of turbulent flows of gasses and fluids. For example, water rushing through pipes, air escaping from a jet, or blood being ejected from the heart across a stenotic valve causes noise.

What are murmurs?

Cardiac murmurs are audible turbulent sound waves in the range of 20 to 20,000 cycles/second emanating from the heart and vascular system. They are common in pediatric patients and represent the most common reason for referral to pediatric cardiologists [9]. Whereas only 0.8% to 1% of the population has structural congenital cardiac disease, as many as 50% to 85% of the population has an innocent heart murmur during childhood [10], and these murmurs must be distinguished from murmurs associated with significant congenital or acquired heart disease.

The nature of cardiac murmurs is often influenced by the age of the patient at presentation, and a thorough understanding of the sequence of cardiac events and the evolution of the fetal, transitional, and postnatal circulations is required to identify and diagnose murmurs correctly.

The cardiac cycle

Cardiac sounds and murmurs that arise from turbulence or vibrations within the heart and vascular system may be innocent or pathologic. An understanding of the timing of events in the cardiac cycle is a prerequisite to understanding heart murmurs. The relationship between the normal heart cycle and heart sounds is described in Fig. 3.

The cardiac cycle begins with atrial systole, the sequential activation and contraction of the two thin-walled chambers. Atrial systole is followed by the delayed contraction of the more powerful lower chambers, termed ventricular systole. Ventricular systole has three phases:

1. Isovolumic contraction: the short period of early contraction when the pressure builds within the ventricle but has yet to rise sufficiently to permit ejection.
2. Ventricular ejection: the period during which the ventricles eject blood to the body (through the aorta) and to the lungs (through the pulmonary artery).
3. Isovolumic relaxation: the period of ventricular relaxation when ejection ceases and pressure falls within the ventricles.
During ventricular contraction the atria begin to relax (atrial diastole) and receive venous blood from both the body and the lungs. Then in ventricular diastole the lower chambers relax, allowing initial passive filling of the thick-walled ventricles and emptying of the atria. Later, during the terminal period of ventricular relaxation, the atria contract. This atrial systole augments ventricular filling just before the onset of the next ventricular contraction.

The sequence of contractions generates pressure and blood flow through the heart. The relationship of blood volume, pressure, and flow determines the opening and closing of heart valves and generates characteristic heart sounds and murmurs. For example, valve closure at higher pressure creates louder heart sounds.

Fig. 3. Phases of the cardiac cycle. The aortic pressure, left ventricular pressure, left atrial pressure, aortic flow, ECG, and heart sounds are presented. The phases of the cardiac cycle begin with atrial systole (phase 1). Following electrical activation of the ventricle, a rapid rise in ventricular pressure occurs (phase 3, isovolumic contraction) accompanied by closure of the atrioventricular valves (S1) and some bulging of the valves into the atrial chambers. During the period of rapid ejection (phase 4) approximately two thirds of the blood in the ventricle is ejected from the heart in the pulmonary arteries and aorta. During the phase of reduced ejection (phase 5) a lesser volume is pumped out of the ventricles at a decreased rate. During the period of isovolumic relaxation (phase 6), the pressure decreases rapidly in the ventricles. The rapid inflow phase (phase 7) occurs immediately after opening of the atrioventricular valves. The blood that has accumulated in the atria rushes into the ventricles. There is a period of reduced inflow (phase 8) during which some flow continues from the atria but at a reduced rate. (Adapted from Abel FL, McCutcheon EP. Cardiac cycle, heart sounds and cardiology. In: Cardiovascular function, principles and applications. Boston: Little, Brown and Company; 1979; with permission.)

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Changes in the circulation at birth

An understanding of the fetal, transitional, and neonatal adaptations of the circulation is important in the evaluation of the pediatric cardiovascular system, because most organic heart disease is most often evident in association with the circulatory changes occurring at birth. The patient’s age at recognition or referral often indicates the nature of the cardiac anomaly and the urgency with which assessment is necessary.

In the fetus (Fig. 4A), oxygen is derived from the placenta and returns through the umbilical vein and through the ductus venosus to enter the inferior vena cava and right atrium. Preferentially, flow is directed across the foramen ovale to enter the left atrium and subsequently the left ventricle. Deoxygenated blood returning from the superior vena cava and upper body segment is preferentially directed by the flap of the Eustachian valve to enter the right ventricle and then, through the ductus arteriosus, to enter the descending aorta to return through the umbilical arteries to the placenta. The systolic pressures within the ventricles are equal because both chambers pump to the systemic circulation. In utero, however, the right ventricle does the majority of the work, pumping 66% of the combined cardiac output. At transition (Fig. 4B), with the first breath, pulmonary arterial resistance begins to fall as the lungs begin the process of respiration. Pulmonary venous return to the left atrium closes the flap of the foramen ovale. Through mechanical and chemical mechanisms the ductus arteriosus begins to close. In the normal-term infant, this closure is accomplished by 10 to 15 hours of age. Intermittent right-to-left atrial level shunting through the foramen ovale may occur, particularly if pulmonary vascular resistance fails to drop. In addition, structural cardiac abnormalities requiring patency of the ductus arteriosus for maintenance of either pulmonary blood flow (eg, pulmonary atresia) or systemic blood flow (eg, hypoplastic left heart syndrome) most often present within the first few days of life. Thus the timing of presentation of a pediatric patient for evaluation influences the spectra of heart disease to be considered. Ductal-dependent abnormalities (eg, pulmonary atresia, transposition of the great arteries, coarctation of the aorta, hypoplastic left heart syndrome) or significant outflow obstructions (eg, aortic valve stenosis) present in the first few days of life. In the absence of an associated anomaly, hemodynamically significant ventricular septal defects seldom present before 2 and 4 weeks of age. Atrial septal defects are seldom symptomatic in infancy.

Normal intracardiac pressures

In the child, following birth and successful transition, resistance to flow is much lower in the pulmonary circuit than in the systemic circuit. Therefore the pressures are lower in the right-sided chambers than in the left-sided chambers. The values in Fig. 4C reflect pressures during ventricular systole in a normal heart. Pressure in the great vessels during the systole is identical to that in the corresponding ventricle. This equilibrium changes if there is outflow obstruction.
Fig. 4. Fetal, transitional and neonatal circulations. The course of the circulation in the heart and great vessels of the late-gestation fetal lamb (A), within a few hours of delivery (B), and as a newborn (C). The figures in the circles within the chambers and vessels represent percentage of oxygen saturation. The numbers alongside the chambers and vessels are pressures in mm Hg related to amniotic fluid pressure as zero. Ao, aorta; DA, ductus arteriosus; DV, ductus venosus; LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle. (Adapted from Rudolph AM. Congenital disease of the heart. Year Book Medical Publishers, Chicago: Year Book Medical Publishers, 1974; with permission.)
In ventricular diastole the semilunar valves (aortic and pulmonary) close. Resistance to blood flow in the vascular bed determines the diastolic pressures in the great arteries. The thin-walled atria generate much lower pressures than the ventricles, both during the phase of passive atrial filling (v wave) and during atrial contraction (a wave). During ventricular relaxation, the diastolic pressures are lower than those in the atria, enabling filling. An understanding of normal cardiovascular hemodynamics and a knowledge of the cardiac cycle are necessary to understand the more complicated hemodynamics and flow patterns of specific cardiac abnormalities.

**Origins of heart sounds and murmurs**

Normal heart sounds originate from vibrations of heart valves when they close and from heart chambers when they fill or contract rapidly. Like a door that bangs shut, the pressure that forces valve closure has the greatest influence on the intensity of a heart sound. Other mechanical factors such as valve stiffness, thickness, and excursion have less effect on sound intensity.

Cardiac murmurs are the direct result of blood flow turbulence. The amount of turbulence and consequently the intensity of a cardiac murmur depend on (1) the size of the orifice or vessel through which the blood flows, (2) the pressure difference or gradient across the narrowing, and (3) the blood flow or volume across the site. As sound radiates away from its source, sound intensity diminishes with the square of the distance. Consequently, heart sounds should be loudest near the point of origin, but other factors influence this relationship. Sound passage through the body is affected by the transmission characteristics of the tissues. For example, fat has a more pronounced dampening effect on higher frequencies does more dense tissue such as bone. If the difference in tissue density is significant, for example between the heart and lungs, more sound energy is lost. That is, only the loudest sounds may be heard when lung tissue is positioned between the heart and chest wall.

In contrast to intensity, the frequency of a cardiac murmur is proportional only to a pressure difference or gradient across a narrowing. These principles make it easy to understand the basis of heart sounds and murmurs.

**The first heart sound**

The first heart sound (S1) arises from closure of the atrioventricular (mitral and tricuspid) valves in early isovolumic ventricular contraction and consequently is best heard in the tricuspid and mitral areas (Fig. 5). Mitral valve closure occurs slightly in advance of tricuspid valve closure and occasionally, near the lower left sternal edge, two components (splitting) of S1 may be heard. Normally, S1 is single. S1 is most easily heard when the heart rate is slow because the interval between S1 and second heart sound (S2) is clearly shorter than the interval between the second and subsequent S1 (ie, systole is shorter than
diastole). The intensity of S1 is influenced by the position of the atrioventricular valve at the onset of ventricular contraction. If the valves leaflets are far apart, the increased excursion needed to accomplish valve closure increases the intensity of S1.

**The second heart sound**

Shortly after the onset of ventricular contraction, the semilunar valves (aortic and pulmonary) open and permit ventricular ejection. Normally, this opening does not generate any sound. The atroventricular valves remain tightly closed during ventricular ejection. As ventricular ejection nears completion, the pressure begins to fall within the ventricles, and the semilunar valves snap close. This closing prevents regurgitation from the aorta and pulmonary artery back into the heart. The closure of the semilunar valves generates S2. Normally, the second heart sound consists of a louder and earlier aortic valve closure (A2) followed by a later and quieter pulmonary valve closure (P2). One will appreciate normal physiologic splitting or variability most easily in the pulmonary area during or near the end of inspiration. During expiration, the aortic and pulmonary valves

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**Fig. 5.** The four primary areas of auscultation represent the general regions where heart sounds and murmurs of the four cardiac valves are often best heard and defined. The areas overlap considerably, and sounds and murmurs are not limited to these sites, often extending to the lateral chest wall, abdomen, and back. (*From* Pelech AN. The cardiac murmur. Pediatr Clin North Am 1998;45(1):114; with permission.)
close almost synchronously and produce a single or narrowly split S2. Normal splitting of S2 is caused by (1) increased right heart filling during inspiration because of increased blood volume returning through the vena cava, and (2) diminished left heart filling because blood is retained within the small blood vessels of the lungs when the thorax expands. During inspiration, when the right ventricle is filled more than the left, it takes slightly longer to empty. This delayed emptying causes the noticeable inspiratory delay in P2 relative to A2. Splitting of S2 during inspiration, so-called “physiologic splitting,” is a normal finding and should be sought in all patients.

**Classification of cardiac murmurs**

Heart murmurs are the consequence of turbulent blood flow, but not all cardiac murmurs indicate structural or physiologic cardiac problems. One should be able to determine and describe the following characteristics of heart murmurs:

1. **Timing:** the relative position within the cardiac cycle and with relationship to S1 and S2
2. **Intensity or loudness:** murmurs are graded as
   - Grade 1: heard only with intense concentration
   - Grade 2: faint, but heard immediately
   - Grade 3: easily heard, of intermediate intensity
   - Grade 4: easily heard and associated with a thrill (a palpable vibration on the chest wall)
   - Area over which the sound is audible (extent of radiation)
3. **Location on the chest wall with regard to**
   - Area where the sound is loudest (point of maximal intensity)
   - Area over which the sound is audible (extent of radiation)
4. **Duration:** the length of the murmur from beginning to end
5. **Configuration:** the dynamic shape of the murmur
6. **Pitch:** the frequency range of the murmur
7. **Quality:** which relates to the presence of harmonics and overtones

**Systolic murmurs**

Systolic murmurs begin with or follow S1 and end before S2 (Fig. 6). They may be classified as

1. **Holosystolic murmurs,** beginning abruptly with S1 and continuing at the same intensity to terminate at S2, can be shown graphically as a rectangular symbol. These murmurs occur when there is a regurgitant atrioventricular valve (tricuspid or mitral) or in association with most ventricular septal defects.
2. **Ejection murmurs** are crescendo-decrescendo or diamond-shaped murmurs that may arise from narrowing of the semilunar valves or outflow tracts.
The rising and falling nature of the murmur reflects the periods of low flow at the beginning and end of ventricular systole.

3. Early systolic murmurs start abruptly with but taper and disappear before S2 and are exclusively associated with small muscular ventricular septal defects.

4. Mid- to late systolic murmurs begin midway through systole and are often heard in association with the midsystolic clicks and the insufficiency of mitral valve prolapse.

Innocent murmurs are almost exclusively ejection systolic in nature. They are generally soft, are never associated with a palpable thrill, and are subject to considerable variation with changes in the patient’s position.

Fig. 6. Four types of systolic heart murmur. The holosystolic or pansystolic murmur begins abruptly with the first heart sound (S1) and proceeds at the same intensity to the second heart sound (S2). The ejection systolic or crescendo-decrescendo murmur begins with the onset of volume ejection from the heart. As the flow increases, the murmur varies both in intensity and frequency and subsequently tapers as the period of ejection ceases, before S2. The early systolic murmur, like the holosystolic murmur, begins abruptly with S1 but terminates in mid-systole with the cessation of shunt flow. The late systolic murmur begins well after S1, commencing in mid- to late systole in association with the development of valve insufficiency and proceeds at this intensity to S2. (From Pelech AN. The cardiac murmur. Pediatr Clin North Am 1998;45(1):111; with permission.)
**Diastolic murmurs**

Diastole, the period between closure of the semilunar valves and subsequent closure of the atrial ventricular valves, is normally silent because little turbulence is associated with the low-pressure flow through relatively large valve orifices. Regurgitation of the semilunar valves, stenosis of an atrioventricular valve, or increased flow across an atrioventricular valve can all cause turbulence and may produce diastolic heart murmurs (Fig. 7).

1. Early diastolic murmurs are decrescendo in nature and arise from either aortic or pulmonary valve insufficiency (regurgitation).
2. Mid-diastolic murmurs are diamond shaped and occur because of either
   a. Increased flow across a normal tricuspid or mitral valve or
   b. Normal flow across an obstructed or stenotic tricuspid or mitral valve.
3. Late diastolic or crescendo murmurs are created by stenotic or narrowed atrioventricular valves and occur in association with atrial contraction.

**Continuous murmurs**

Flow through vessels, channels, or communications beyond or distal to the semilunar valves is not confined to systole and diastole. Thus there may be

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![Fig. 7. The early diastolic or decrescendo murmur occurs in association with closure of the semilunar valves (S2) and tapers through part or all of diastole. The mid-diastolic murmur rises and falls in intensity with atrial volume entering the ventricle. The late systolic or crescendo diastolic murmur occurs late in diastole with atrial contraction, before systole, and ascends to the first heart sound (S1). (From Pelech AN. The cardiac murmur. Pediatr Clin North Am 1998;45(1):112; with permission.)](image-url)
turbulent flow throughout the cardiac cycle. The resulting murmurs extend up to and beyond S2 (Fig. 8). Continuous murmurs can be heard through part or all of diastole. Continuous murmurs are generally pathologic. The venous hum is a notable exception.

Performance of auscultation

The examining physician should develop a routine of listening systematically to all components of the cardiac cycle and all ausculatory areas with both the bell and diaphragm. Selection of the appropriate stethoscope and maintenance of equipment is important. No one stethoscope is ideal for all practitioners, but a good stethoscope should include both a shallow bell for low frequencies and a thin, stiff diaphragm for high frequencies. The diaphragm is subject to damage, and regular maintenance is often required. The tubing should not so long that sounds are diminished. Perhaps the feature most often neglected is appropriate selection of earpieces. They should be the largest possible soft earpieces that appropriately occlude the external ear canal [11]. When appropriate, patients should routinely be auscultated in the supine, sitting, and standing positions. As will be described for innocent murmurs but equally applicable in organic or pathologic murmurs, the effects of dynamic maneuvers (including respiration, Valsalva, exercise, postural changes, and other maneuvers) provide important diagnostic information [12,13].

When auscultating the patient, the clinician should first direct attention to the normal heart sounds in sequence. The effects of inspiration and expiration on the heart sounds should be appreciated. Then additional heart sounds and murmurs should be addressed. Any variability that occurs with a change of body position should be described.
Thorough auscultation in the cooperative patient may take as long as 5 to 10 minutes and should include listening in the principal areas of the precordial auscultation (tricuspid, pulmonary, mitral, and aortic) with both the bell and diaphragm with the patient in the supine, sitting, and standing positions.

Four areas serve as a guide to auscultation of the heart (see Fig. 5). These areas are the optimal sites for listening to sounds that arise within the chambers and great vessels. The examiner should not feel constrained by these guidelines and should also listen between and beyond these areas.

1. The tricuspid area is represented by the fourth and fifth intercostal spaces along the left sternal edge but often extends to the right of the sternum as well as downward to the subxiphisternal area.
2. The pulmonary area is the second intercostal space along the left sternal border. Murmurs that are best heard in this area may also extend to the left infraclavicular area or often lower, along the left sternal edge to the third intercostal space.
3. The mitral area involves the region of the cardiac apex and generally is at the fifth intercostal space in the midclavicular line. This area may also extend medially to the left sternal edge and also laterally to the region of the axilla.
4. The aortic area, although centered at the second right intercostal space, may extend to the suprasternal area, neck, and inferiorly to the third left intercostal space.

The margins of these areas are ill defined, and auscultation may extend to the axillae, neck, back, or infraclavicular areas.

No matter how experienced the examiner, step-by-step auscultation first for heart sounds, subsequently for systolic murmurs, and then separately for diastolic murmurs is essential. In the cardiac assessment of children, the ability to characterize S2 clearly is perhaps more crucial than characterizing any other sound, and the effects of respiration are important in both the normal and abnormal cardiac assessment. The components of S2 in childhood are normally split with inspiration and become single on expiration. A loud pulmonary closure sound should suggest the possibility of pulmonary artery hypertension. S2 may be widely split or fixed in association with right ventricular volume overload or delayed right ventricular conduction. Normal inspiratory splitting of S2 should be sought and established in all patients but may be difficult to determine in some cases. In the infant with a rapid respiratory rate, the presence of splitting at any time during the respiratory cycle may be accepted as normal.

An ejection click or sharp sound present at the left upper sternal boarder, louder with expiration or heard only on expiration, is characteristic of pulmonary valve stenosis. The ejection click follows the period of isovolumic contraction and occurs as a consequence of restricted semilunar (aortic or pulmonary) valve excursion at the onset of ventricular ejection. When the ejection sound occurs at the right upper sternal border or at the apex, bicuspid or stenotic aortic valve
disease is suggested. In contrast to ejection clicks, right-sided cardiac murmurs are accentuated with inspiration. Left heart auscultatory abnormalities vary little with the respiratory cycle.

**Innocent murmurs of childhood**

By definition, innocent murmurs occur in the absence of structural or physiologic cardiac disease. Innocent or normal murmurs have been called “functional,” “benign,” “innocuous,” or “physiologic,” but are perhaps best termed “normal” to convey accurately to parents the favorable impression and outcome that should accompany the diagnosis. The clinical diagnosis of a normal or innocent murmur should only occur in the setting of an otherwise normal history, physical examination, and appearance.

Normal murmurs of childhood are comprised of six systolic and two continuous types (Box 1) but are never solely diastolic. The intensity or loudness of the murmur is grade 3 or less and consequently is never associated with a palpable thrill. Most murmurs, both innocent and organic, are accentuated by fever, anemia, or increased cardiac output.

**The vibratory Still’s murmur**

The most common innocent murmur in children is the vibratory systolic murmur described by George F. Still in 1909 [14], frequently referred to as “Still’s murmur.” The murmur is most typically audible in children between ages 2 and 6 years but may be present as late as adolescence or as early as infancy. The murmur is low to medium pitched, confined to early systole, generally grade 2 (range, 1–3), and maximal at the lower left sternal edge and extending to the

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**Box 1. Innocent murmurs of childhood**

**Systolic murmurs**

Vibratory Still’s murmur
Pulmonary flow murmur
Peripheral pulmonary arterial stenosis murmur
Supraventricular systolic murmur
Aortic systolic murmur

**Continuous murmurs**

Venous hum
Mammary arterial soufflé
The murmur is generally loudest in the supine position and often changes in character, pitch, and intensity with upright positioning.

The most characteristic feature of the murmur is its vibratory quality initially described by Still as “a twanging sound, very like that made by twanging a piece of tense string” [14]. This vibratory quality is attributable to the presence of harmonic overtones or multiples of the baseline sound frequency (70–150 cycles/second), giving a pleasing or musical character to the murmur. The quality of the murmur can thus never be described as “noisy” or “rough.” Quite characteristically, the intensity of the murmur diminishes and the pitch changes with upright positioning, but the murmur seldom disappears.

The origin of the murmur remains obscure. Its origins have been ascribed to vibration of the pulmonary valves during systolic ejection [15], vibrations arising from the shift in blood mass in the dynamically contracting ventricle [16], physiologic narrowing of the left ventricular outflow tract [17], and the presence of ventricular false tendons [18]. Phonocardiographic recordings have shown the innocent murmur arises from either the right ventricular [19] or left ventricular outflow tracts [17]. The prevalence of false tendons remains high in adults (16.8%) and is more frequently associated with innocent systolic ejection murmurs [20], although the typical vibratory quality of the Still’s murmur is seldom appreciated beyond adolescence.

The pulmonary flow murmur

An innocent pulmonary outflow tract murmur may be heard in children, adolescents, and young adults. The murmur is a crescendo-decrescendo, early- to mid-peak ejection systolic murmur confined to the second and third interspace at the left sternal border. It is of low intensity (grade 2–3) and transmits to the pulmonary area. It is rough and dissonant without the vibratory musical quality of the Still’s murmur. The murmur is best heard with the patient in the supine position and is exaggerated by the presence of a pectus excavatum, a straight back, or kyphoscoliosis, which results in compression or approximation of the right ventricular outflow tract to the chest wall. The murmur is augmented in full exhalation in the supine position, rarely resulting in the perception of a palpable thrill, and is diminished by upright positioning and held inspiration.

The murmur of an atrial septal defect is attributable to increased flow through the pulmonary outflow tract and may be indistinguishable from the innocent pulmonary flow murmur. The hyperdynamic right ventricular impulse, wide splitting of the pulmonary component of S2, and the presence of a mid-diastolic flow rumble should enable distinction.

The murmur of pulmonary valve stenosis may be distinguished from the innocent pulmonary flow murmur by the frequent presence of a systolic thrill, higher pitch, longer duration, or presence of an ejection click. The presence of an ejection click signifies improper opening of a semilunar valve and is always of pathologic origin. In pulmonary stenosis S2 may be widely split, and P2, when audible, is of diminished intensity.
Peripheral pulmonary arterial stenosis murmur

A murmur heard frequently in newborns and infants less than 1 year of age is the audible turbulence of peripheral branch pulmonary arterial stenosis, angulation, or narrowing. These ejection-character murmurs are typically grade 1 or 2, low to moderate pitched beginning in early to mid-systole, and extending up to and occasionally just beyond S2. These murmurs are most often present in normal newborns but may be associated with viral lower respiratory tract infections and reactive airway disease in older infants. In the fetus, the pulmonary trunk is a relatively dilated, domed structure because it receives the majority of the combined cardiac output from the high-pressure right ventricle. Right and left pulmonary artery branches arise from this major trunk as comparatively small lateral branches that receive little intrauterine flow because the lungs are relatively collapsed. When the lungs expand at birth, the relative disparity transiently persists. In addition, the branches arise at comparatively sharp angles from the main pulmonary trunk, accounting for turbulence and a recognized physiologic drop in pressure from the main to the proximal branch pulmonary arteries [21]. In the older infant, regional vascular reactivity and redistribution of pulmonary blood flow associated with a respiratory tract infection may account for the reappearance of the murmur.

Characteristically, the murmurs are often best heard peripherally in the axilla and back with both regional and temporal variability. These murmurs are often missed or overlooked because of the rapid respiratory rate of infants, the similarities in sound frequency of breath sounds and murmurs, and the peripheral location of the murmurs. For reasons that are unclear, these murmurs are often most evident in the recovery phase of a respiratory illness. The murmur of peripheral branch stenosis changes with variations in heart rate, increasing in intensity with heart rate slowing as the stroke volume increases and conversely diminishing with tachycardia and reduction in stroke volume.

The normal peripheral branch stenosis murmur may be indistinguishable from the peripheral murmur of significant stenosis of the branch pulmonary vessels seen in William’s or rubella syndrome or accompanying hypoplasia or narrowing of the pulmonary arteries. In children beyond the first few months of life, murmurs of significant anatomic narrowing may be distinguished by their higher pitch and extension beyond S2. The pulmonary flow murmur of an atrial septal defect may mimic this murmur but characteristically is not seen in this age group. Proximal pulmonary valve or right ventricular outflow obstruction may also closely resemble this murmur but is often louder in intensity, possibly associated with a ejection click, and heard maximally lower along the left sternal border.

The supraclavicular or brachiocephalic systolic murmur

A supraclavicular systolic crescendo-decrescendo murmur may be heard in children and young adults [22]. This systolic murmur is audible maximally above the clavicles and radiates to the neck but may present to a lesser degree on the
superior chest. The murmur is low to medium pitched, of abrupt onset, brief, and maximal in the first half or two thirds of systole. High pitch or extension into diastole is unusual and suggests significant vascular obstruction.

The murmur is present with the patient both supine and sitting but varies with hyperextension of the shoulders [23]. The shoulders can be hyperextended with the elbows brought behind the back until the shoulder girdle is taut. When this maneuver is done rapidly, the murmur diminishes or disappears altogether [24].

Supraclavicular systolic murmurs are thought to arise from the major brachiocephalic vessels as they arise from the aorta [25].

The aortic systolic murmur

Innocent systolic flow murmurs may arise from the outflow tract in older children and adults. The murmurs are ejection in character, confined to systole, and audible maximally in the aortic area. In children, these murmurs may arise secondarily to extreme anxiety, anemia, hyperthyroidism, fever, or any condition of increased systemic cardiac output.

In trained athletes, slower heart rates with increased stroke volume may give rise to short crescendo-decrescendo murmurs of low to medium pitch. Physical examination may suggest a relatively displaced thrusting apex and physiologic S3 [13].

These murmurs must be distinguished from the systolic murmur of hypertrophic cardiomyopathy and additional fixed obstructions of the left ventricular outflow tract. A family history for hypertrophic cardiomyopathy or a family history of unexplained death in a young individual, particularly if associated with activity, is justification for referral. A systolic murmur that becomes louder with performance of the Valsalva maneuver is considered almost diagnostic of hypertrophic cardiomyopathy with systolic anterior motion of the mitral valve. A reduction in venous return results in closer apposition of the septum and mitral valve and dynamic narrowing of the left ventricular outflow tract. By contrast, rapid squatting improves venous return; the left ventricular chamber size is enlarged, the mitral valve and septum are further apart, and the murmur of hypertrophic cardiomyopathy becomes softer [26]. It is often difficult to be certain of the cause of this type of murmur, and referral and further investigations may be indicated.

Normal continuous murmurs

The venous hum

The most common type of continuous murmur heard in children is the innocent cervical venous hum. First described by Potain [27] in 1867, this continuous murmur is most audible on the low anterior part of the neck just lateral to the sternocleidomastoid muscle but often extends to the infraclavicular area.
of the anterior chest wall. The murmur is generally louder on the right than on the left, louder with the patient sitting than supine, and is accentuated in diastole. Intensity varies from faint to grade 6, and occasionally patients are aware of a loud hum [28]. The murmur is quite variable in character and is often described as whining, roaring, or whirring.

The venous hum is best accentuated or elicited with the patient in a sitting position and looking away from the examiner. The murmur often resolves or changes in character when the patient lies down and may be eliminated or diminished by gentle compression of the jugular vein or by turning the patient’s head toward the side of the murmur. The murmur is thought to arise from turbulence at the confluence of flow as the internal jugular and subclavian veins enter the superior vena cava or perhaps from angulation of the internal jugular vein as it courses over the transverse process of the atlas [29].

The mammary arterial soufflé

The mammary soufflé was first described by van den Bergh in 1908 [30]. It occurs most frequently late in pregnancy and in lactating women but may rarely occur in adolescence. The murmur arises in systole but may extend well into diastole, being audible maximally on the anterior chest wall over the breast. There is usually a distinct gap between S1 and the origin of the murmur, thought to relate to the delayed arrival of cardiac stroke volume at the peripheral vasculature. The murmur is generally high pitched and has an unusual superficial character but may vary considerably from day to day. Firm pressure with the stethoscope or digit pressure on the chest wall may occasionally abolish the murmur. The murmur is thought to be arterial in origin, arising from the plethoric vessels of the chest wall [30]. The murmur must be distinguished from the continuous high-pitched murmur of an arteriovenous fistula or a patent ductus arteriosus. The mammary soufflé characteristically varies significantly from day to day, presents in a distinctive patient population, and resolves with termination of lactation.

References


