Chapter 16
Blood Pressure, Heart Tones, and Diagnoses

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Abstract The primary purpose of this chapter is to familiarize the reader with the basic concepts of blood pressure, heart tones, and some common diagnoses. It remains the general consensus that even the most sophisticated electronic monitors cannot fully reduce the need for sound clinical skills like proper patient inspection, palpation, percussion, and auscultation. Furthermore, it is important to reinforce the need for a deep understanding of basic physiological principles when interpreting physical examination findings. Invasive and noninvasive methods for assessing blood pressure are discussed in addition to new technologies.

Keywords Blood pressure measurement · Palpation · Doppler effect · Auscultation · Oscillometry · Plethysmography · Arterial tonometry · Arterial cannulation · Heart tones

16.1 Blood Pressure

Fundamental to providing comprehensive care to patients is the ability to obtain an accurate medical history and carefully perform a physical examination. The optimal selection of further tests, treatments, and use of subspecialists depends on well-developed history and physical examination skills. Key elements of a normal physical include obtaining a blood pressure reading and auscultation of the heart, both providing important information about the patient's hemodynamics and aiding in diagnosing anatomical and physiological pathology.

Naive ideas about circulation and blood pressure date as far back as ancient Greece. It took until the 18th century before the first official report describing an attempt to measure blood pressure was written, when Stephen Hales published a monograph on "haemastatics" in 1733. He conducted a series of experiments involving invasive cannulation of arteries in horses for the resultant direct blood pressure measurement. Unfortunately, his methods were not applicable for humans. During the subsequent two centuries, there were many contributions to the science of blood pressure control and its assessment. One of the greatest of these contributions was a publication in Gazetta Medica di Torino in 1896, called "A New Sphygmomanometer" by Dr. Riva-Rocci; this publication is still recognized as the single most important advancement in the field of practical noninvasive methods for blood pressure estimation.

In 1916, French physician Rene Laennec invented the first stethoscope, which was constructed from stacked paper rolled into a solid cylinder. Prior to his invention, physicians around the world would place an ear directly over the patient's chest to hear the heart and/or lung sounds. After Dr. Laennec's initial success, several new models were produced, primarily made of wood; this stethoscope was called a "monaural stethoscope." The "binaural stethoscope" was invented in 1829 by a doctor from Dublin and later gained widespread acceptance. In the 1960s, the Camman binaural stethoscope was considered the standard because of its superior auscultation capabilities.

It is essential for health care professionals and bioengineers to understand how blood pressure and heart tones are obtained, the advantages and disadvantages of the different methods used to obtain them, and how to interpret the gathered information.

16.1.1 Physiology of Blood Pressure

Blood pressure is the force applied on arterial walls as the heart pumps blood through the circulatory system. The rhythmic contractions of the left ventricle result in cyclic

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changes in the arterial blood pressure. During ventricular systole, the heart pumps blood into the circulatory system, and the pressure within the arteries reaches its highest level—a point known as "systolic blood pressure." During diastole, the pressure within the arterial system falls to its lowest level, a point called "diastolic blood pressure."

"Mean blood pressure," often used clinically, represents the time-weighted average of the arterial pressures recorded during one cardiac cycle. The alternating systolic and diastolic pressures create outward and inward movements of the arterial walls, perceived as arterial pulsation or arterial pulse. "Pulse pressure" is another clinical term and represents the difference between the systolic and diastolic blood pressures.

It is accepted convention that blood pressure is measured in millimeters of mercury (mmHg). A normal systolic blood pressure is less than 140 mmHg, and a normal diastolic blood pressure is less than 90 mmHg. Blood pressure higher than normal signifies "hypertension," and one lower than normal is called "hypotension." Normal mean arterial pressure is between 60 and 90 mmHg. The mean arterial pressure has significance in that it will drive tissue perfusion and can be measured directly using an automated blood pressure cuff or calculated using the following formulas:

$$\mathrm{MAP} = \mathrm{DBP} + \mathrm{PP}/3 \text{ or } \mathrm{MAP} = [\mathrm{SBP} + (2 \times \mathrm{DBP})]/3$$

where PP = SBP–DBP, MAP = mean arterial pressure, DBP = diastolic blood pressure, PP = pulse pressure, and SBP = systolic blood pressure.

Blood flow throughout the circulatory system follows pressure gradients. By the time blood reaches the right atrium, representing the end point of the venous system, pressure will decrease to approx 0 mmHg. The two major determinants of the arterial blood pressure are: (1) cardiac output, representing the volume of blood pumped by the heart per minute; and (2) systemic vascular resistance, which is the impediment to blood flow created by the vascular bed. Cardiac output depends on a complexity of numerous factors including preload, contractility, afterload, heart rate, and/or rhythm. Systemic vascular resistance is equally complex and controlled by many additional factors including vasomotor tone of arterioles, terminal arterioles, and/or precapillary sphincters. Blood pressure as a function of the cardiac output and systemic vascular resistance can be expressed with the following abstract formula:

$$\mathrm{BP} = \mathrm{CO} \times \mathrm{SVR}$$

where BP = blood pressure, CO = cardiac output, and SVR = systemic vascular resistance.

Blood pressure decreases by 3–5 mmHg in arteries 3 mm in diameter, and then reaches approximately 85 mmHg when entering arterioles; this accounts for approx 50% of the resistance of the entire systemic circulation. Blood pressure is further reduced to around 30–40 mmHg at the point of entry into capillaries, and then becomes approximately 10 mmHg at the venous end of the capillaries.

The speed of the advancing pressure wave during each cardiac cycle far exceeds the actual blood flow velocity. In the aorta, the pressure wave speed may be 15 times faster than the flow of blood. In an end artery, the pressure wave velocity may be as much as 100 times the speed of the forward blood flow.

As the pressure wave moves peripherally through the arterial tree, wave reflection, refraction, and interference distort the pressure waveform, causing an exaggeration of systolic and pulse pressures. This enhancement of the peripheral pulse pressure causes the systolic blood pressure in the radial artery to be 20–30% higher than the aortic systolic blood pressure and the diastolic blood pressure to be approx 10–15% lower than the aortic diastolic blood pressure. Nevertheless, the mean blood pressure in the radial artery will closely correspond to the mean aortic arterial pressure.

16.1.2 Methods of Measuring Blood Pressure

Arterial blood pressure can be measured both noninvasively and invasively, as described in the following sections.

16.1.2.1 Noninvasive Methods

Palpation

Palpation is a relatively simple and easy method for assessing systolic blood pressure. For example, a blood pressure cuff containing an inflatable bladder is applied to the arm and inflated until the arterial pulse felt distal to the cuff disappears. Pressure in the cuff is then released at a speed of approx 3 mmHg per heartbeat until an arterial pulse is felt again. The pressure at which the arterial pulsation can be detected is the systolic blood pressure; diastolic blood pressure and mean arterial pressure cannot be readily estimated using this method. Furthermore, the systolic blood pressure measured using the palpation method is typically an underestimation of the true arterial systolic blood pressure, i.e., because of insensitivity of the sense of touch and the relative delay between blood flow below the cuff and the appearance of arterial pulsations distal to the cuff.

Doppler Method

The Doppler method for blood pressure measurement is a modification of the palpation method described above. It uses a sensor Doppler probe to determine blood flow distal to the blood pressure cuff. The "Doppler effect" represents a shift in the frequency of a sound wave when a transmitted sound is reflected back from a moving object. Such a sound wave shift (e.g., caused by blood movement in an artery) is detected by a Doppler monitor and presented as a specific swishing sound. The pressure in the cuff, at which a blood flow is detected by the Doppler probe, is the systolic arterial blood pressure. Doppler-assisted blood pressure measurement is more accurate and less subjective in estimating systolic blood pressure, compared to the palpation method. Using Doppler for blood pressure assessment is quite useful for patients in shock and patients in low-flow states, as well as obese and pediatric (very young) patients. Disadvantages of the Doppler method include: (1) an inability to detect the diastolic blood pressure; (2) a necessity for sound-conducting gel between the skin and the probe (air is a poor conductor of ultrasound); (3) the likelihood of a poor signal if the probe is not applied directly over an artery; and/or (4) the potential for motion and electrocautery artifacts.

Auscultation (Riva-Rocci Method)

The auscultation method uses a blood pressure cuff placed around an extremity (usually an upper extremity) and a stethoscope placed above a major artery just distal to the blood pressure cuff (e.g., the brachial artery if using the cuff on the upper extremity). Inflation of the blood pressure cuff above the patient's systolic blood pressure flattens the artery and stops blood flow distal to the cuff. As the pressure in the cuff is released, the artery becomes only partially compressed, which creates conditions for turbulent blood flow within the artery and produces the so-called "Korotkoff" sounds, named after the individual who first described them. Korotkoff sounds are caused by the vibrations created when blood flow in the partially flattened artery transforms from laminar into turbulent, and this state persists as long as there is a turbulent flow within the vessel. Systolic blood pressure is determined as the pressure of the inflated cuff at which Korotkoff sounds are first detected. Diastolic blood pressure is determined as the cuff pressure at which Korotkoff sounds become muffled or disappear.

Sometimes, in patients with chronic hypertension, there can be an auscultatory gap that represents disappearance of the normal Korotkoff sounds in a wide pressure range between the systolic and diastolic blood pressures. This condition will lead to inaccurately low blood pressure assessment. Korotkoff sounds can also be difficult to detect in patients who are in shock or in those with marked peripheral vasoconstriction. The use of microphones and electronic sound amplification can greatly increase the sensitivity of this method. Yet, considerations for systematic errors include motion artifact and electrocautery interference.

Oscillometry

Oscillometry is a noted method for blood pressure measurement which employs an automated blood pressure cuff. Arterial pulsations cause pressure oscillations in a cuff placed over an extremity. These oscillations are at their maximum when the cuff pressure equals the mean arterial pressure, and decrease significantly when the cuff pressure is above the systolic blood pressure or below the diastolic blood pressure. Advantages of this approach include the ease and reliability of use; some of the potential technical problems include motion artifacts, electrocautery interference, and inability to measure accurate blood pressure when patients experience arrhythmias.

When selecting a blood pressure cuff for noninvasive blood pressure measurement, it is important to select the cuff in accordance with the patient's size. Blood pressure cuffs for adult and pediatric patients come in variable sizes. An appropriate size means that the cuff's bladder length is at least 80% and the cuff width is at least 40% of the patient's arm circumference. If the cuff is too small, it will need to be inflated to a greater pressure to completely occlude the arterial blood flow, and the resultant measured pressure will be falsely elevated. On the other hand, if the cuff is too large, the pressure inside the cuff needed for complete occlusion of the arterial blood flow will be less, and the measured pressure will be falsely low.

Blood pressure is most commonly taken while the patient is seated with the arm resting on a table and slightly bent, which will typically position the patient's arm at the level of his/her heart. This same principle should be applied if the patient is in a supine position; the blood pressure cuff should be level with the patient's heart. If the location of the blood pressure cuff during blood pressure measurement is above or below the patient's heart level, measured blood pressure will be either falsely lower or higher than the actual pressure. This difference can be represented as the height of a column of water interposed between the level of the blood pressure cuff and the level of the patient's heart. To convert centimeters of water (cm H_2O) to millimeters of mercury, the measured height of the water column

should be multiplied by a conversion factor of 0.74 (1 cm $H_2O = 0.74$ mmHg).

All of the aforementioned methods for assessing blood pressure do so indirectly by registering blood flow below a blood pressure cuff. Other noninvasive methods include plethysmography and arterial tonometry.

Plethysmography

The plethysmographic method for blood pressure assessment employs the fact that arterial pulsations cause a transient increase in the blood volume of an extremity, and thus in the volume of the whole extremity. A finger plethysmograph determines the minimum pressure needed by a finger cuff to maintain constant finger blood volume. A light-emitting diode and a photoelectric cell are used to detect changes in the relative finger volume; this information is, in turn, used to rapidly adjust the cuff pressure. Data can be displayed as a beat-to-beat tracing. Thus, in healthy patients, the blood pressure measured on a finger will correspond to the aortic blood pressure. Importantly, this relationship does not hold true for patients with low peripheral perfusion, such as those with peripheral artery disease, hypothermia, or patients in low-flow states.

Arterial Tonometry

Tonometry devices can determine beat-to-beat arterial blood pressure by adjusting the pressure required to partially flatten a superficial artery located between a tonometer and a bony surface (e.g., radial artery). These devices commonly consist of an electronic unit and a pressure-sensing head. The system includes an adjustable air chamber and an array of independent pressure sensors that, when placed directly over the artery, assess intraluminal arterial pressure. The resultant pressure record resembles an invasive arterial blood pressure waveform. Yet, limitations to this method include motion artifacts and the need for frequent calibration.

16.1.2.2 Invasive Methods of Blood Pressure Measurement

Indications

Indications for using direct blood pressure monitoring (arterial cannulation) include consistent hemodynamic instability, intraoperative monitoring in selected patients, and use of vasoactive drugs like dopamine, epinephrine, norepinephrine, and the like.

Cannulation Sites

Arteries most often selected for direct cannulation are the radial, ulnar, brachial, femoral, dorsalis pedis, or the axillary. Cannulation of an artery should be avoided if there is: (1) a considered lack of appropriate collateral circulation; (2) a skin infection on or near the site of cannulation; and/or (3) a known preexisting vascular deficiency (e.g., Raynaud's disease). The radial artery is the most often selected artery for invasive blood pressure monitoring because of easy access, superficial location, and good collateral flow to the region it supplies.

Techniques

The two frequently utilized techniques for arterial cannulation are: (1) a catheter over a needle; and (2) the "Seldinger's" technique. When using the catheter over a needle technique, the operator enters a selected artery with a needle over which a catheter has been placed. After free blood flow through the needle, the catheter is advanced over the needle and into the artery, after which the needle is withdrawn. The catheter is then connected to a pressure-transducing system. When using the Seldinger's technique, the operator first enters the artery with a needle. After confirmation of free blood flow through the needle, the operator places a steel wire into the artery and withdraws the needle. A plastic catheter is then advanced into the artery over the wire, then the wire is removed and the catheter is connected to a transducer system. Both methods require sterile techniques and skilled operators.

Considerations

Arterial cannulation provides beat-to-beat numerical information and thus tracings of appropriately monitored waveforms of arterial blood pressure, which is considered a "gold standard" in blood pressure monitoring. Invasive arterial pressure monitoring systems include a catheter (20 gauge catheter for adults), tubing, a transducer, and an electronic monitor for signal amplification, filtering, and analysis. Such pressure transducers are commonly based on the strain gauge principle—stretching a wire of silicone crystal changes its electrical resistance. The catheter, the connective tubing, and the transducer are prefilled with saline, and the use of a pressure bag provides continuous saline flush of the system at a typical rate of 3–5 mL/h. These systems should also allow for intermittent flush boluses.

Quality of information gathered using invasive blood pressure monitoring depends on the dynamic characteristics of the whole system. The complex waveform

obtained from the arterial pulse can be expressed as a summation of simple sine and cosine waves using Fourier analysis. Most invasive blood pressure monitoring systems are designed to have natural frequencies of approximately 16–24 Hz, slightly exceeding the frequency of the arterial pulse waveform in order to reproduce it correctly. This natural frequency is described as that at which the system oscillates when disturbed. Another property of the catheter–tubing–transducer system is its dumping coefficient, characterizing how quickly oscillations in the system will spontaneously decay.

Both the natural frequency and the dumping coefficient are primarily determined by the employed length, size, and compliance of the catheter and tubing, as well as by presence of any air bubbles or blood clots that may be trapped in the tubing. This chapter is not intended to go into details about how to determine and change the system characteristics but, briefly, underdumping the system will exaggerate artifacts. For example, a catheter whip can result in a significant overestimation of the systolic blood pressure. Likewise, overdumping will blunt the response of the catheter–tubing–transducer system and lead to an underestimation of the systolic blood pressure. In addition, systems with low natural frequencies will show amplifications of the pressure curves, thus causing overestimation of the systolic blood pressure. Diastolic blood pressure will also be affected by altering the above-mentioned factors, but to a lesser degree. Note that system response characteristics can be optimized by using short, low-compliance tubing and by avoiding air trapping by using a flushing system.

When an invasive blood pressure system is connected to a patient, it should be zero referenced and calibrated. Zero referencing is performed by placing the transducer at the level of the midaxillary line, which corresponds to the level of the patient's heart when the patient is supine. The system is then opened to air, closed to the patient, and adjusted to a 0 mmHg baseline. Note that for proper zero referencing, it is not necessary for the transducer to be at the level of the patient's heart as long as the stopcock, which is opened to air during zero referencing, is at that level. The system is then directed to record from the patient and thus is ready for use.

System calibration is a separate procedure and involves connecting the invasive blood pressure system to a mercury manometer, closing the system to the patient, and pressurizing it to certain predetermined pressures. The gain of the monitor amplifier is then adjusted until displayed pressure equals the pressure in the mercury manometer. Recommendations are to perform zero referencing at least once on every clinical shift and this type of calibration at least once daily. It should be noted that some of the more contemporary transducer designs rarely require external calibration.

When connected to a patient, today's invasive blood pressure monitoring systems provide digital readings of systolic, diastolic, and mean blood pressures and pressure waveforms. Watching the trend of a waveform and its shape can provide other important information as well. More specifically, the top of the waveform represents the systolic blood pressure, and the bottom is the diastolic blood pressure. The dicrotic notch is caused by the closure of the aortic valve and backsplash of blood against the closed valve. The rate of the upstroke of the arterial blood pressure wave depends on the myocardial contractility, whereas the rate of the downstroke is affected by the systemic vascular resistance. Exaggerated variation in the size of the waves with respiration suggests hypovolemia. Thus, the trained eye can gain insight about a patient's cardiovascular status by evaluating aspects of this signal. Integrating the area under the waveform can be used for calculating the mean arterial pressure.

Complications

Potential complications associated with arterial cannulation include bleeding, hematoma, infection, thrombosis, ischemia distal to the cannulation site, vasospasm, embolization with air bubbles or blood clots, nerve damage, pseudoaneurysm formation, atheroma, and/or inadvertent intraarterial drug injection.

16.1.3 Diagnoses

16.1.3.1 Pulsus Paradoxus

Normally, the arterial and venous blood pressures fluctuate throughout the respiratory cycle, decreasing with inspiration and rising with expiration. Yet, this fluctuation in the blood pressure under normal conditions is less than 10 mmHg. Inspiration increases venous return, therefore increasing the right heart output transiently, according to the Frank–Starling law. As the blood is sequestered in the pulmonary circulation during inspiration, the left heart output is reduced and is expressed as a lower systolic blood pressure. The right ventricle contracts more vigorously and mechanically bulges the interventricular septum toward the left ventricle, reducing its size and accounting for even lower systolic blood pressure. Pulsus paradoxus is defined as an inspiratory decrease of systolic blood pressure by more than 10 mmHg.

Certain conditions drastically reduce the transmural or distending (filling) pressure of the heart and interfere with the diastolic filling of the ventricles. In such cases, there is

typically an exaggeration of the inspiratory fall in the systolic blood pressure, which results from reduced left ventricular stroke volume and the transmission of the negative intrathoracic pressure to the aorta. Common causes for such reduction include pericardial effusion, adhesive pericarditis, cardiac tamponade, pulmonary emphysema, severe asthma, paramediastinal effusion, endocardial fibrosis, myocardial amyloidosis, scleroderma, mitral stenosis with right heart failure, tricuspid stenosis, hypovolemia, and/or pulmonary embolism. Associated clinical signs include a palpable decrease in pulse with inspiration and decrease in the inspiratory systolic blood pressure of more than 10 mmHg compared to the expiratory pressure. On clinical examination, one can detect extra beats on cardiac auscultation during inspiration, when compared to a peripheral pulse.

16.1.3.2 Pulsus Alternans

Pulsus alternans is an alternating weak and strong peripheral pulse, caused by alternating weak and strong heart contractions. A weak contraction will decrease the ejection fraction, hence increasing the end-diastolic volume (the volume of blood remaining in the ventricle after a weak heart contraction). As a result, in the next cardiac cycle, the heart will be stretched more; then, according to the Frank–Starling mechanism, this will generate higher pressures and a stronger perceived pulse. Pulsus alternans may be found in patients with severe heart failure, various degrees of heart block, and/or arrhythmias. Pulsus alternans is characterized by a regular rhythm and must be distinguished from pulsus bigeminus, which is an irregular heart rate (see below).

16.1.3.3 Bigeminal Pulse

A bigeminal pulse is caused by occurrences of premature contractions (usually ventricular) after every other heartbeat, resulting in an alternation in the relative pulse strength. Bigeminal pulse can often be confused with pulsus alternans. However, in contrast to the latter, in which the rhythm is regular, in pulsus bigeminus the weak beat always follows a shorter pulse interval; thus there is an arrhythmia.

16.1.3.4 Pulse Deficit

Pulse deficit is the inability to detect some arterial pulsations when the heart beats, as can be observed in patients with atrial fibrillation, in states of shock, or with premature ventricular complexes. The easiest way to detect pulse deficit is to place a finger over an artery while monitoring the QRS complexes on an electrocardiogram monitor. A QRS complex without a detected corresponding pulse represents a pulse deficit. In the presence of atrioventricular dissociation, when atrial activity is irregularly transmitted to the ventricles, the strength of the peripheral arterial pulse depends on the relative timing of the atrial and ventricular contractions. In a patient with rapid heartbeats, the presence of such variations suggests ventricular tachycardia. With an equally rapid rate, an absence of variation of pulse strength suggests a supraventricular mechanism. See Chapters 11 and 26 for an additional description of arrhythmias.

16.1.3.5 Wide Pulse Pressure

Wide pulse pressure, often called "water hammer pulse," is observed in cases of severe aortic regurgitation and consists of an abrupt upstroke (percussion wave) followed by a rapid collapse later in systole with no dicrotic notch.

16.1.3.6 Pulsus Parvus et Tardus

The phenomenon of pulsus parvus (small) et tardus (late) is observed in cases of aortic stenosis and is caused by reduction in stroke volume and prolonged ejection phase, producing reductions and delays in the volume increments inside the aorta. "Tardus" refers to delayed or prolonged early systolic acceleration; "parvus" refers to diminished amplitude and rounding of the systolic peak.

16.1.3.7 Bisferiens Pulse

A bisferiens (biphasic) pulse is characterized by two systolic peaks—the percussion and tidal waves—separated by a distinct midsystolic dip. The peaks are often equal, or one may be larger. Bisferiens pulse occurs in conditions in which a large stroke volume is ejected rapidly from the left ventricle and is observed most commonly in patients with either pure aortic regurgitation or with a combination of aortic regurgitation and stenosis. A bisferiens pulse can also be elicited by patients with hypertrophic obstructive cardiomyopathy. In these patients, the initial prominent percussion wave is associated with rapid ejection of blood into the aorta during early systole, followed by a rapid decline as the obstruction becomes prominent in midsystole followed by a tidal wave. Very rarely, bisferiens pulse occurs in individuals with a normal heart.

16.1.3.8 Dicrotic Pulse

Not to be confused with a bisferiens pulse, in which both peaks occur in systole, the dicrotic pulse is characterized by a second peak positioned in diastole immediately after the second heart sound. The normally small wave that follows aortic valve closure (dicrotic notch) is exaggerated and measures more than 50% of the pulse pressure on direct pressure recordings. A dicrotic pulse usually occurs in conditions such as cardiac tamponade, severe heart failure, and hypovolemic shock, in which a low stroke volume is ejected into a soft elastic aorta. Rarely, a dicrotic pulse can be noted in healthy adolescents or young adults.

16.2 Heart Tones

16.2.1 Physiology and Normal Heart Sounds

The primary heart tones are caused by vibrations created by pressure differentials during closure of the heart valves. Normal valve opening is relatively slow and makes little or no audible sounds. In general, heart tones are brief and characterized by varying intensity (loudness), frequency (pitch), and quality (timbre). To understand heart tones better, let us briefly review the physiology of the cardiac cycle. The electric impulse for cardiac contraction starts from the sinus node, located in the right atrium, thus causing the right atrium to contract first. Contraction of the ventricles begins with the left ventricle, resulting in mitral valve closure slightly before closure of the tricuspid valve. Ejection, on the other hand, starts in the right ventricle, because the right ventricular ejection normally occurs at a much lower pressure than the left ventricular ejection. Yet, ejection ends first in the left ventricle, causing the aortic valve to close slightly before the pulmonic valve.

The first heart sound (S1) arises from closure of the mitral valve (M1), and shortly thereafter by closure of the tricuspid valve (T1). The initial component of the first heart sound (M1) is most prominent at the cardiac apex. The second component (T1), when detected, normally presents at the left lower sternal border; it is less commonly heard at the apex and is seldom heard at the base. When the first heart sound noticeably splits, like in Ebstein's anomaly (often associated with delayed right ventricular activation), its first component is normally louder.

As mentioned previously, the intensity of heart sounds will increase with increases in pressure gradients across a particular valve. With an increasing pressure gradient, the blood velocity and the resultant force causing valve closure will increase, producing louder and more easily detectable sounds. Another factor affecting the intensity of the sound produced by an atrioventricular valve is the valvular position at the onset of systole. Note that when ventricular contraction occurs against a wide open valve, the leaflets will achieve higher velocity and thus the heart sound will be louder compared to a sound produced by a valve with partially closed leaflets at the beginning of systole.

The second heart sound (S2) is caused by closure of the aortic and the pulmonic valves. Normally the first component of this second heart sound is caused by the aortic valve closure (A2), followed shortly thereafter by the pulmonic valve closure (P2).

The physiological third heart sound (S3) (Fig. 16.1) occurs shortly after A2, and is a low-pitched vibration caused by rapid ventricular filling during diastole. Physiologic S3 sounds can commonly be heard in children, adolescents, and young adults. When detected after 30 years of age, S3 is referred to as "ventricular gallop" and is considered a sign of possible pathology. In most of the S3 ventricular gallop cases, there is diastolic dysfunction associated with ventricular failure. Normal third heart sounds sometimes persist beyond age 40 years and are more commonly found in women.

The physiological fourth heart sound (S4) (Fig. 16.2) is typically soft and low-pitched, and is best heard in late diastole just before S1. S4 is generated by rapid ventricular filling during atrial systole, causing vibrations of the left ventricular wall and the mitral apparatus. Normally, S4 is primarily heard in infants, small children, and adults over the age of 50 years. A loud S4, which can be

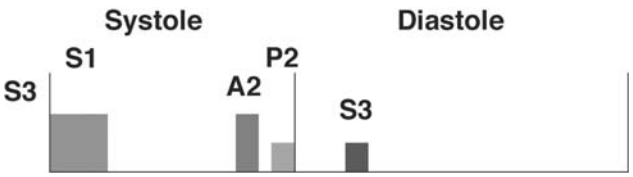

Fig. 16.1 Third heart sound (see text for details). A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound; S3 = third heart sound

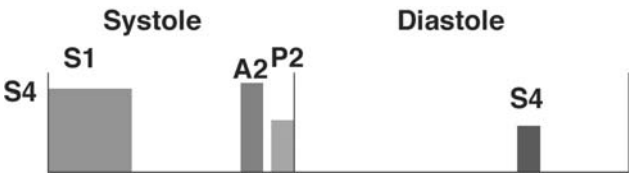

Fig. 16.2 Fourth heart sound (see text for details). A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound; S4 = fourth heart sound

associated with shock, is considered as a pathological sign and is referred to as an "S4 gallop."

16.2.2 Auscultatory Areas

Heart sounds generated by different valves are best heard over certain auscultatory areas, which bear the valve names but do not specifically correspond to the anatomical locations of the valves.

The aortic auscultation area is located over the second intercostal space at the right sternal border (Fig. 16.3). The pulmonic auscultation area is located in the second intercostal space at the left sternal border. The mitral auscultation area is just over the heart's apex, located in the fifth intercostal space, left of the sternum; this area is also called a left ventricular or apical area. The tricuspid auscultation area is located at the left lower sternal border. For patients with a left thoracic heart position (normal situs), auscultation should begin at the cardiac apex and continue with the left lower sternal border (following the inflow), and then the session should proceed interspace after interspace, up the left sternal border, up to the left myocardial base, and then move to the right base (outflow). This type of examination permits clinicians to think physiologically, i.e., following the inflow–outflow direction of the blood flow.

To most easily distinguish between the first and second heart sounds, one should take into account that there is a longer pause between S2 and S1 than between S1 and S2, caused by the fact that systole is shorter than diastole. S1 is also of longer duration and lower pitch, compared to the shorter duration and higher pitch of S2. In general, S1 is best heard at the heart's apex, and S2 is best auscultated over the aortic and pulmonic areas.

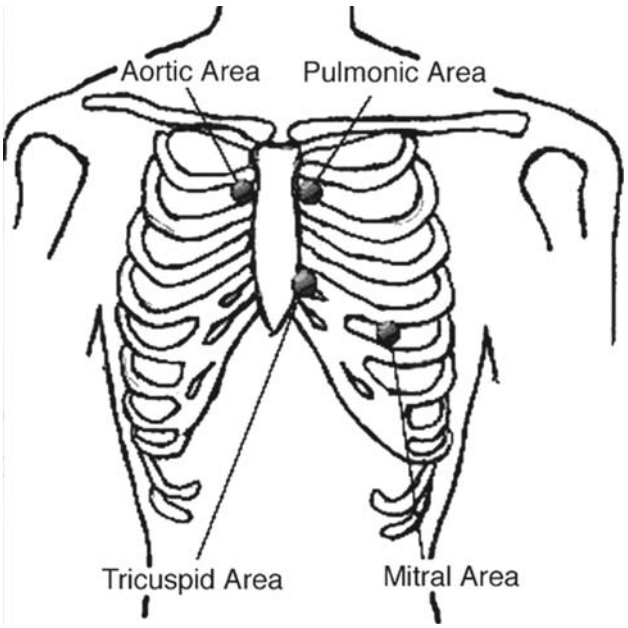

Fig. 16.3 Auscultatory areas, including the aortic, pulmonic, mitral, and tricuspid auscultation areas

The normal heart sounds represent normally occurring phenomena in a normal heart, whereas cardiac pathologies can change the intensity and/or the timing of the occurrence of the sounds and/or even create new ones, often called "murmurs."

16.2.3 Abnormal Heart Sounds

Conditions that accentuate S1 sounds include mitral stenosis (most often), left-to-right shunts, hyperkinetic circulatory states, accelerated atrioventricular conduction, and/or tricuspid stenosis. A diminished S1 can be caused by mitral and tricuspid stenosis, moderate or severe aortic regurgitation, slow atrioventricular conduction, and/or hypocontractility states. A diminished S1 sound can also be observed in patients with thick chest walls, such as in individuals with excessively developed body musculature, obese patients, or in patients with emphysema.

Variability in the S1 sound can be observed in states causing variation in the velocity of atrioventricular valve closure, such as ventricular tachycardia, atrioventricular block, ventricular pacemakers, atrial fibrillation, and so on. An accentuated S2 is commonly detected in patients with: (1) diastolic or systolic hypertension; (2) aortic coarctation; (3) aortic dilation; (4) atherosclerosis of the aorta; and/or (5) pulmonary hypertension which is characterized by a loud pulmonary component of the second heart sound. A diminished S2 sound is detected most often in aortic valvular stenosis, pulmonic stenosis, and/or pulmonary emphysema.

Some degree of the splitting of the S2 sound can be normally heard during inspiration. Yet, abnormal or persistent S2 splitting is associated with: (1) delayed activation of the right ventricle; (2) prolonged right ventricular ejection time relative to left ventricular ejection time, like in pulmonic stenosis, mitral regurgitation, or ventricular septal defect; or (3) increased impedance of the pulmonary vasculature, as in massive pulmonary embolism or pulmonary hypertension. Persistent S2 splitting may also be observed in cases in which the aortic and pulmonic components of the second heart sound remain audible during both inspiration and expiration. This type of persistent splitting is often caused by a delay in the pulmonary component, such as that occurring in complete right bundle branch block, but it can also be caused by an early

timing of the aortic component often associated with mitral insufficiency. Changes in the duration of the split interval (greater with inspiration, lesser with exhalation) in the presence of both components define the split as persistent and not fixed.

Fixed splitting of S2 is commonly found in patients with atrial septal defect, severe pulmonic stenosis, or right ventricular failure. S2 fixed splitting is characterized by wide and persistent intervals between the aortic and pulmonary components, remaining unchanged during the respiratory cycle.

"Paradoxical splitting" refers to a reversed sequence of the semilunar valve (aortic and pulmonic) closures, with the pulmonary component (P2) preceding the aortic component (A2). Paradoxical splitting of S2 is caused by a delay in the A2, varying with the inspiratory cycle and can be caused by: (1) a complete left bundle branch block; (2) premature right ventricle contractions; (3) ventricular tachycardia; (4) severe aortic stenosis; (5) left ventricular outflow obstruction; (6) hypertrophic cardiomyopathy; (7) coronary artery disease; (8) myocarditis; and/or (9) congestive cardiomyopathy.

Normally, blood flow in the heart is laminar and does not produce vibrations. "Murmurs" are created whenever there is a pathology causing increased turbulent flow, often associated with abnormal shunts, obstructions, or even the reversing of flow. In other words, turbulent flow within the heart creates vibrations that can be heard as murmurs. The various murmurs are described both on the basis of appearance in relation to the cardiac cycle and on the basis of changes in features such as intensity (loudness), frequency (pitch), configuration (shape), quality, duration, and/or direction of radiation. For example, intensity or loudness can be graded from 1 to 6. Based on time of existence relative to the cardiac cycle, murmurs are classified as systolic, diastolic, or continuous. Depending on their life cycle during systole or diastole, they are further subclassified into early, mid, and late systolic or diastolic murmurs such that:

- Early systolic murmur begins with S1 and ends before the midsystole.
- Midsystolic murmur starts after S1 and ends before S2.
- Late systolic murmur begins in the middle of systole and ends at S2.
- Holosystolic murmur starts with S1 and continues for the duration of the whole systole.
- Early diastolic murmur begins with S2.
- Middiastolic murmur begins after S2.
- Late diastolic murmur begins just before S1.
- Continuous murmur continues during both systole and diastole.

Based on changes in intensity, murmurs are commonly described as: (1) crescendo, increasing in intensity; (2) decrescendo, decreasing in intensity; (3) crescendo–decrescendo, when the intensity of the murmur first increases and then decreases; or (4) plateau, the intensity of the murmur remains constant.

16.2.4 Dynamic Auscultation

The term dynamic auscultation refers to a technique of adjusting circulatory dynamics by means of respiration or various other planned physiological or pharmacological maneuvers and then determining their effects on the dynamics of the heart sounds and murmurs. Commonly altered variables that can affect sound and murmurs include: (1) changes in venous return affecting cardiac preload; (2) changes in systemic vascular resistance; (3) changes in contractility; (4) changes in heart rate or rhythm; and/or (5) maneuvers affecting pressure gradients within the heart. To date, diagnostic maneuvers frequently used for altering heart sounds include inspiration, expiration, exercise level, or body position (e.g., recumbent position, left semilateral position, standing up, sitting up, or leaning forward).

In general, spontaneous inspiration causes decreased intrathoracic pressure, increased venous return, increased right ventricular preload, and decreased pulmonary vascular resistance. Thus, modifying inspiration is used to increase the intensity of S3 and S4 gallops, tricuspid and pulmonic stenosis or regurgitation murmurs, or mitral and tricuspid clicks. Inspiration also causes some degree of splitting of S2, caused by prolonged right ventricular ejection. In contrast, expiration causes just the opposite—decreased venous return (preload) and decreased right-sided flow. Note that sounds and murmurs originating on the left side of the heart tend to be accentuated during expiration.

In addition, changes in a patient's positioning can affect heart sound intensity; this is caused by relative changes of ventricular preload, ventricular size, and movement of the heart closer to or farther from the chest wall. A recumbent position typically accentuates murmurs of mitral and tricuspid stenosis. Similarly, a left semilateral position accentuates left-sided S3 and S4, mitral opening snap, and mitral regurgitation murmurs. Standing will affect general hemodynamics by pooling blood in the lower extremities and decreasing heart filling pressure and ventricular size. Thus, standing up is clinically used to accentuate mitral and tricuspid clicks. Sitting up accentuates tricuspid valve opening snap, and sitting up and leaning forward are the best maneuvers for enhancing murmurs due to aortic and pulmonic regurgitation or aortic stenosis. Exercise causes increases in heart rate,

shortens diastole, elevates left atrial pressure, and shortens the time for closure of the heart valves. Hence, physiological changes during exercise increase amplitudes of S1, S2, S3, S4, mitral opening snap, existing mitral regurgitation or stenosis, and/or patent ductus arteriosus murmur.

Two other pathological sounds, heard when auscultating the heart, are "clicks" and "opening snaps." Clicks are caused by the rapid movements of valvular structures. Systolic clicks are referred to as ejection or nonejection clicks, depending on their timing relative to systole. Ejection clicks, occurring early in systole, commonly indicate semilunar valve anomalies and, in rare conditions, great vessel lesions. Nonejection clicks are heard in mid to late systole and represent mitral (more often) or tricuspid valve prolapse. In contrast, a tricuspid valve opening snap is often heard when there is tricuspid valve stenosis and in conditions associated with increased blood flow across the tricuspid valve (e.g., presence of large atrial septal defect). Similarly, a mitral valve opening snap is caused by elevated left atrial pressure, resulting in rapid valve opening to the point of maximum excursion. Mitral valve opening snap is often associated with mitral stenosis and less often with ventricular septal defect, second or third degree atrioventricular block, patent ductus arteriosus, and hyperthyroidism. The mitral valve opening snap is similar in quality to normal heart sound and is often clinically confused with a splitting of S2.

16.2.5 Specific Murmurs

The murmur caused by aortic stenosis (Fig. 16.4) is characterized as a holosystolic crescendo–decrescendo murmur which is best heard at the aortic auscultation area. The high-velocity jet within the aortic root results in radiation of the murmur upward, to the right second intercostal space, and further into the neck. Although the murmur in the second right intercostal space is harsh, noisy, and impure, the murmur heard when auscultating over the left apical area can be pure and often considered musical. The harsh "basal murmur" is believed to be caused by vibrations created when high-velocity blood jet is ejected through the aortic root. The musical second component of the aortic stenosis murmur originates from periodic high-frequency vibrations of the fibrocalcific aortic cusps and can be quite loud and heard even from a distance without an auditory aid. Murmurs of aortic stenoses are accentuated by expiration, sitting up, and/or leaning forward. The high-frequency apical midsystolic murmur of aortic stenosis should be distinguished from the high-frequency apical murmur of mitral regurgitation, a distinction that may be difficult or even impossible to detect, especially if the aortic component of the second heart sound is soft or absent. In such patients, echocardiography will likely be required to determine the relative cardiac pathophysiology; see Chapter 20 for more information on echocardiographic methods.

A murmur caused by pulmonary valve stenosis is a characteristic midsystolic murmur originating in the right side of the heart and best auscultated over the pulmonic auscultation area. This murmur begins after the first heart sound, rises to a peak in crescendo, and then decreases in slow decrescendo, finishing before a delayed or soft pulmonary component of the second heart sound. In general, the length and the profile of this murmur depend on the severity of the pulmonary valve obstruction.

The murmur caused by aortic insufficiency (Fig. 16.5) is an early decrescendo diastolic murmur originating in the left side of the heart and best heard over the aortic and pulmonic auscultation areas. This murmur begins with the aortic component of the second heart sound (S2). The intensity and configuration of such a murmur tend to reflect the volumes and rates of regurgitant flows. Radiation of this murmur to the right sternal border can signify aortic root dilation, often associated with Marfan's syndrome. In chronic aortic regurgitation, the aortic diastolic pressure always significantly exceeds the left ventricular diastolic pressure, so the decrescendo is subtle and yet the murmur is well heard throughout diastole. The diastolic murmur of acute severe aortic regurgitation differs from the chronic aortic regurgitation murmur primarily in that

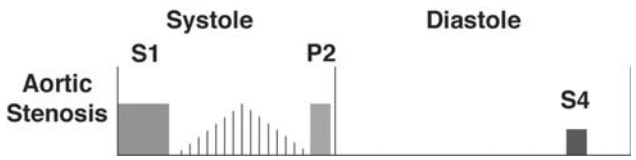

Fig. 16.4 Aortic stenosis murmur caused by stenotic aortic valve. Normally a holosystolic crescendo–decrescendo murmur is best heard at the aortic auscultatory area. P2 = pulmonic valve closure; S1 = first heart sound; S4 = fourth heart sound

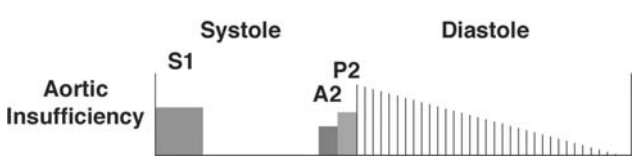

Fig. 16.5 Aortic insufficiency murmur. Early decrescendo diastolic murmur originating in the left side of the heart and best heard over the aortic and pulmonic auscultation areas. A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

the diastolic murmur is relatively short. The short duration of the acute severe aortic regurgitation murmur is due to the fact that aortic diastolic pressure rapidly equilibrates with the rapidly rising diastolic pressure in the nondilated left ventricle. The aortic insufficiency murmur is accentuated during expiration, sitting up, or leaning forward.

A pulmonary regurgitation murmur is an early diastolic murmur originating from the right side of the heart. In this case, the second heart sound is often split with the murmur proceeding from its latter part; it is loud because of relatively high transvalvular pressure. The high pulmonary diastolic pressure generates high-velocity regurgitant flow resulting in a high-frequency blowing murmur that may last throughout diastole. Because of the persistent and significant difference between the pulmonary arterial and right ventricular diastolic pressures, the amplitude of this murmur is relatively uniform throughout most of the diastole.

Mitral stenosis murmur (Fig. 16.6) is caused by a stenotic mitral valve and is decrescendo–crescendo holodiastolic by nature. It is heard best at the mitral auscultation area and is typically accentuated both by exercise and assuming a recumbent position. Mitral stenosis murmur that lasts up to the first heart sound, even after long cardiac cycle, indicates that the stenosis is severe enough to generate a persistent gradient even at the end of long diastole.

The murmur caused by tricuspid stenosis is relatively middiastolic in origin and differs from the mitral stenosis middiastolic murmur in two important aspects: (1) the loudness of the tricuspid stenosis murmur increases with inspiration; and (2) to best hear this murmur, one auscultates over a relatively localized area along the left lower sternal border. Detectable inspiratory increases in loudness occur because of augmentation of the right ventricular volume, decreases in right ventricular diastolic pressure, and increases of the gradient and flow across the stenotic tricuspid valve. This murmur is best detected over the left lower sternal border, as it originates within the

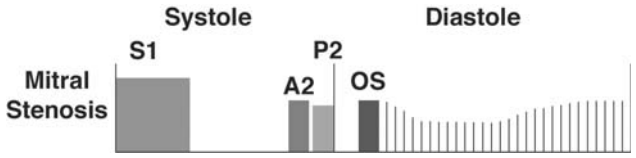

Fig. 16.6 Mitral stenosis murmur. Decrescendo–crescendo holodiastolic murmur, caused by a stenotic mitral valve. Best heard at the mitral auscultation area, this murmur is accentuated by exercise and by assuming a recumbent position. A2 = aortic valve closure; OS = opening snap; P2 = pulmonic valve closure; S1 = first heart sound

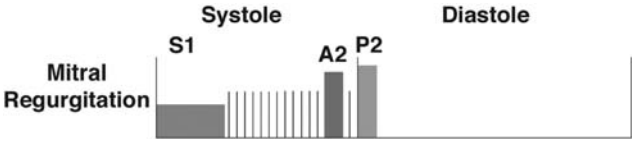

Fig. 16.7 Caused by an insufficiency of the mitral valve, the mitral regurgitation murmur is a systolic murmur best heard over the mitral auscultation area. A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

inflow portion of the right ventricle and is then transmitted to the overlying chest wall.

Mitral regurgitation murmur (Fig. 16.7) is considered to be caused by an insufficiency of the mitral valve, and is a systolic murmur best heard at the mitral auscultation area; it is accentuated by exercise and left semilateral position. Acute severe mitral regurgitation is often accompanied by an early systolic murmur or holosystolic murmur that has a decrescendo pattern, which diminishes or ends before the second heart sound. The physiological mechanism responsible for this early systolic decrescendo murmur is acute severe regurgitation into a relatively normal size left atrium with limited distensibility. In such patients, the regurgitant flow is typically maximal early in systole and reaches minimum by late systole.

Another early systolic murmur is the "tricuspid regurgitation murmur" (Fig. 16.8), which is often associated with infective "endocarditis." The mechanisms responsible for the timing and configuration of this murmur are analogous to those described for mitral regurgitation. It is also a systolic murmur best heard over the tricuspid area and accentuated by inspiration.

The typical murmur associated with an atrial septal defect (Fig. 16.9) is also a systolic murmur which is caused by increased blood flow through the pulmonary valve, and thus is best heard over the pulmonic auscultation area. Most often, the atrial septal defect involves the fossa ovalis, which is midseptal in location, and is of the ostium secundum type (see Chapters 3 and 9 for more details). This type of defect is a true deficiency of the atrial septum and should not be confused with a patent foramen ovale. The magnitude of the left-to-right shunt through an atrial septal defect depends on the size of the defect and also the relative

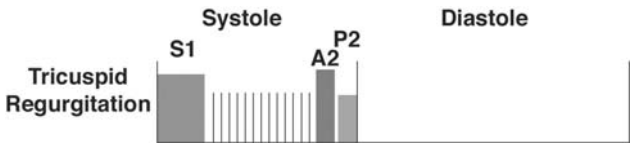

Fig. 16.8 The tricuspid regurgitation murmur is a systolic murmur best heard over the tricuspid area and is accentuated by inspiration. A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

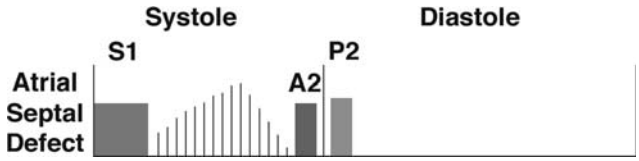

Fig. 16.9 The murmur associated with atrial septal defect is systolic and caused by increased blood flows through the pulmonic valve. Atrial sepal defect murmur is best heard over the pulmonic auscultation area. A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

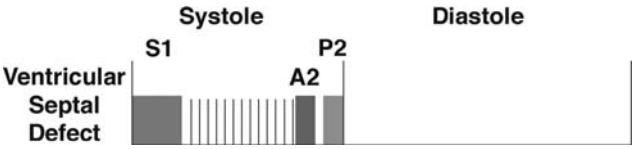

Fig. 16.11 Murmur associated with ventricular septal defect is systolic, caused by blood flow from the left to the right ventricle (or the right to the left ventricle in Eisenmenger's syndrome). A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

compliance of the ventricles, as well as the relative resistance in both the pulmonary and systemic circulations. The increased pulmonary valve flow causes a delay (splitting) of the pulmonic component of the second heart sound.

The murmur associated with a patent ductus arteriosus (Fig. 16.10) is caused by an abnormal continuous turbulent flow through a patent ductus connecting the aorta with the main pulmonary artery. It is also considered a continuous "machinery murmur," heard in both systole and diastole, because aortic pressure is higher than the pressure in the pulmonary artery throughout the cardiac cycle. Other associated clinical findings in patients with patent ductus arteriosus include bounding peripheral pulses, wide pulse pressure, an infraclavicular and interscapular systolic murmur, precordial hyperactivity, hepatomegaly, bradycardia, episodes of apnea, and/or respiratory insufficiency.

The resultant murmur associated with a ventricular septal defect (Fig. 16.11) is systolic, with its intensity depending on the relative size of the defect. This ventricular sepal defect murmur is caused by abnormal blood flow from the left to the right ventricle or from the right to the left ventricle, as in Eisenmenger's syndrome. This latter syndrome is the reversing of the left-to-right shunt in patients with atrial septal defect, ventricular septal defect, or patent ductus arteriosus, all caused by increased pulmonary and right-sided pressures, secondary to increased pulmonary blood flow. This murmur is best heard over the mitral auscultation area located over the myocardial apex. Septal defects are commonly treated, sometimes noninvasively, thus eliminating these murmurs. See Chapter 34 for more details.

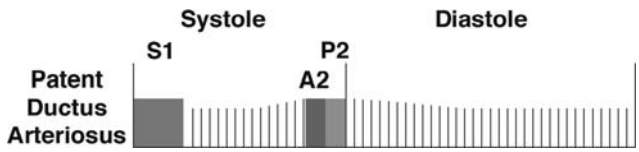

Fig. 16.10 Commonly associated with patent ductus arteriosus, this murmur is caused by a turbulent continuous flow through the ductus connecting the aorta with the main pulmonary artery. A2 = aortic valve closure; P2 = pulmonic valve closure; S1 = first heart sound

16.3 Recent Developments

The clinical application of cardiac auscultation has remained the same for centuries until recently, when some companies took auscultation to a whole new level by developing portable electronic stethoscopes. The central idea behind the electronic stethoscope is that human hearing is imperfect (or changes in its ability, i.e., aging) and can only detect a small fraction of the vast frequency range produced by the human heart. Therefore, recording the whole sound spectrum would provide novel information and thus increase our ability to diagnose pathology. More specifically, using electronic auscultation, heart sounds can be recorded for storage, later direct or remote playback or processing, such as e-mail, digital amplification, filtering for noise reduction, or electronic manipulation for accentuating particular sounds or noise. Today, electronic stethoscopes can easily record a phonocardiogram, providing visual sound representation, and can be viewed equally well by everyone thus eliminating errors associated with different hearing levels and/or clinical skills.

Currently several companies involved with digital stethoscope production include 3M Littmann, American Diagnostics Corp, Cardionics, Thinklabs, Welch Allyn, Doctors Research Group, and Andromed. Products from these companies typically employ proprietary technology and software, and therefore offer different filtering capabilities, presentation of information, size, weight, and/or ease of use.

It should be noted that an important part of each electronic stethoscope is the signal processing. There are several signal processing techniques employed, each providing slight advantages or disadvantages in obtaining different parts of the sound spectrum. Currently Fourier transform, short time Fourier transform, wavelet transform, and discrete wavelet transform are the incorporated sound processing techniques used in digital stethoscopes. Nevertheless, the main limitation of digital stethoscopy is the somewhat subjective nature of sound interpretation. Despite the superior quality of heart tones and murmurs, sounds obtained with digital stethoscopes have to be interpreted by physicians just like sounds obtained with acoustic stethoscopes. One way to deal with this

limitation is to run the digitally obtained and processed sound through an implemented algorithm to identify key parameters and yield relative diagnoses. In the near future, promising technology for achieving this diagnostic approach may be through the use of the artificial neural network, so that input data can be compared against learned data. The potential advantage of using an artificial neural network is that the network is adaptive and able to learn based on information fed to the system during a learning phase. For example, Zargis Medical recently developed Cardioscan, which is the first computer-aided medical device able to analyze heart sounds and identify suspected murmurs. Interestingly, Zargis has partnered with 3M Littmann and claims that using Cardioscan can reduce unnecessary referrals by 41%.

16.4 Summary

It remains the general consensus that even the most sophisticated electronic monitors cannot fully reduce the need for sound clinical skills like proper patient inspection, palpation, percussion, and auscultation. Furthermore, it is important to reinforce the need for a deep understanding of basic physiological principles when interpreting physical examination findings. In general, the application of technology in this area is viewed as helpful and can speed up decision making, but the knowledge of basic physiology and technology limitations can help us utilize these advancements to their maximum.

The primary purpose of this chapter was to familiarize the reader with the basic concepts of blood pressure, heart tones, and some common diagnoses. Many descriptions of the general scientific and clinical principles used in the chapter are simplified for clarity, along with their described underlying physiology and pathology; by no means is this chapter a complete review of the presented topics. There are many medical books dedicated to each topic alone and readers are strongly encouraged to further research sources and review in-depth information of interest.

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