The Cardiac Cycle

Dr. Ronald Drusin

Objective: We shall correlate heart sounds with physiologic events during the cardiac cycle. Initially, we shall review normal physiologic changes that produce normal heart sounds; later we shall present abnormal heart sounds and discuss their origin in altered physiology. Thinking in terms of etiologic, anatomic, and physiologic abnormalities will be stressed.

Prerequisite Knowledge: It is assumed that all students have completed the First Year course in Physiology and have a working understanding of the cardiac cycle.

THE HEART is a mechanical pump. Actually, it is two pumps in series which are synchronized by sharing a common origin of electrical stimulation—the sinus node. Automatic pacemaker cells in the sinus node depolarize spontaneously at a rate of 60 to 100 beats per minute. The sequential electrical excitation of the cardiac chambers is recorded from surface leads as the electrocardiogram. The P wave is generated as the stimulus spreads through the right and left atria, and it stimulates the atria to contract in end-diastole, maximally filling the ventricles just before systole begins. There is a delay in conduction through the A-V junctional tissue of up to 160 milliseconds. Electrical activation of both right and left ventricles is represented by the QRS. The T wave is a wave of repolarization of the ventricles. Systole begins with the peak of the R wave of the QRS.

Although the right and left heart are two pumps in series, timing of physiologic events is brought about by simultaneous electrical stimulation. Consequently, right and left atria are both stimulated (P wave) and contract simultaneously. Normally, both ventricles receive electrical stimulation simultaneously (QRS), and thus contract together as well.

Using a technique called cardiac catheterization, which enables us to measure changes in pressure in each of the heart chambers during the cardiac cycle, we are

able to correlate electrical and mechanical events. The process involves very narrow, fluid-filled, open tubes which are connected to pressure transducers. Pressures measured at the leading tip of the tube, or catheter, are displayed on an oscilloscope calibrated in millimeters of mercury. Peripheral arteries and veins are entered and the catheter passed antegrade under fluoroscopic control into the chambers of the right side of the heart, and retrograde into the chambers of the left side of the heart. Pressures are measured directly on the right side in the pulmonary artery, right ventricle, and right atrium. On the left side, pressures are measured directly within the left ventricle and aorta. Pressure in the left atrium is measured indirectly by advancing the right-sided catheter into a pulmonary artery, wedging it into a small vessel, and recording pulmonary capillary wedge pressure, which has been shown to be identical to left atrial pressure.

In addition to displaying the surface electrocardiogram, the timing axis, and changing pressures inside cardiac chambers on our oscilloscope, we are able to display simultaneous heart sounds. These sounds are recorded with sensitive microphones which are placed on the chest at specific auscultatory locations. The mitral valve is best heard at the apex with the patient lying supine, slightly turned to the left. The aortic valve is auscultated with the patient sitting and the microphone or stethoscope bell applied to the second or third intercostal space to the right of the sternum. The pulmonic valve is auscultated to the left of the sternum in the second intercostal space. Both valves may be heard at Erb's point, in the third intercostal space adjacent to the sternum on the left. The tricuspid valve is auscultated at the fifth intercostal space to the left of the sternum with the patient supine (Fig. 1).

Reviewing the pressure events on the left side of the heart, the left atrium receives blood from the pulmonary circulation (Fig. 2). During diastole, the atrium passively empties into the relaxing left ventricle. At the end of diastole the atrium is stimulated (P wave), and contracts, actively emptying. During atrial con-

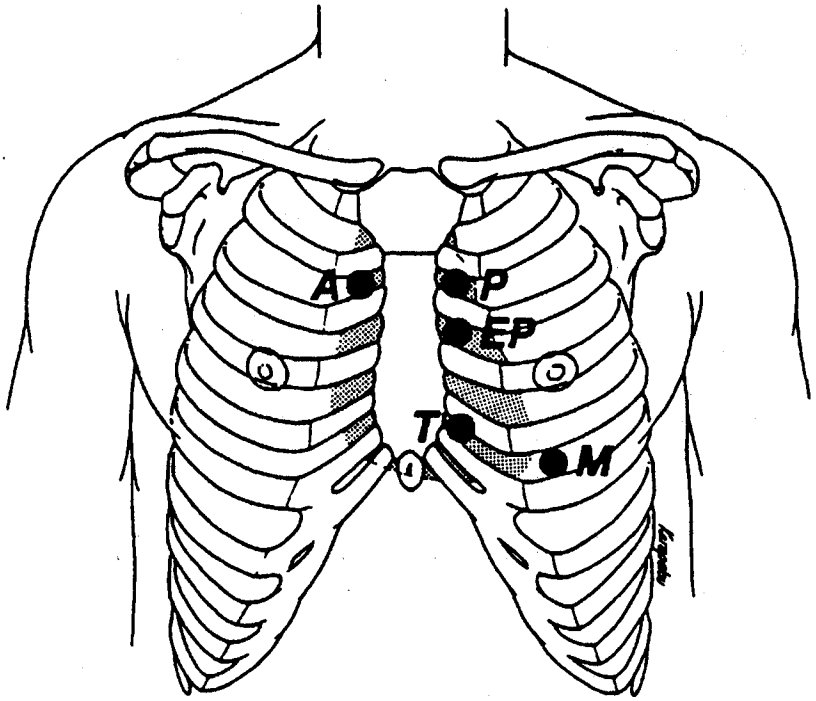

Figure 1

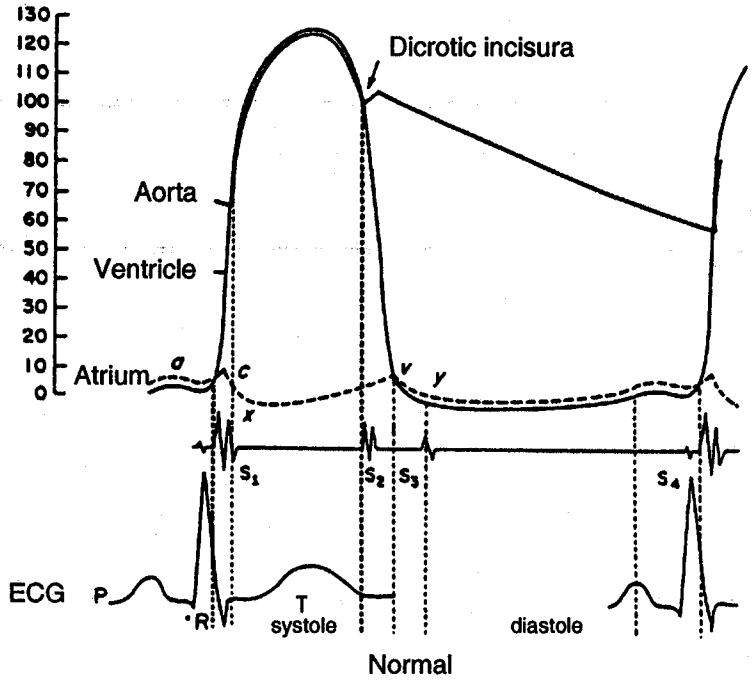

Figure 2

traction, intra-atrial and ventricular pressure rise—recorded as the "a" wave. When systole begins, the mitral valve closes with an increase in ventricular pressure, higher than atrial pressure during isovolemic contraction. As the pressure in the ventricle rises during isovolemic contraction, mitral leaflets bulge into the atrium, producing a "c" wave. During ventricular systole, the left atrium fills passively at low pressure from the pulmonary veins. Gradually, as the atrium fills, its pressure rises. The "v" wave is formed as the atrium completes filling, and reaches its peak as the ventricle relaxes. The mitral valve opens when ventricular pressure falls below atrial pressure.

The decline in atrial pressure is the Y descent. Blood in the atrium then fills the ventricle passively in early diastole.

Catheters in both chambers would record identical pressures inside the atrium and ventricle during end diastole, assuming no obstruction to blood flow between these chambers occurs. Consequently, diastolic pressure is low during passive filling, with the most rapid diastolic filling of the ventricle in early diastole. An "a" wave is recorded in end diastole with filling brought about by atrial systole. As ventricular systole begins, pressure in the ventricle rises rapidly during isovolemic contraction. When ventricular pressure exceeds atrial pressure, the mitral valve closes. When ventricular pressure is greater than aortic pressure, the aortic valve opens and ejection of blood into the systemic circulation occurs. During this phase, pressure in the ventricle and the aorta are identical in normals. The normal ventricle will eject more than 50% of its blood volume in end diastole (ejection fraction). After maximal ejection, the ventricle begins to relax and pressure falls. When left ventricular pressure falls below aortic pressure (incisura), the aortic valve closes and systole is complete. Left ventricular pressure continues to fall.

When left ventricular pressure falls below left atrial pressure, the mitral valve opens and the ventricle commences the rapid phase of diastolic filling.

Physiologic events are identical on the right side of the heart. The right atrium receives blood from the inferior and superior vena cava. During diastole the right atrium passively empties into the right ventricle. When the right atrium is stimulated to contract (P wave) at the end of diastole, it actively contracts to fill the right ventricle. Systole begins with electrical activation of the right ventricle (R wave of the QRS). Pressure in the right ventricle rises rapidly during isovolemic contraction. When ventricular pressure exceeds atrial pressure, the tricuspid valve closes and may bulge into the atrium ("c" wave). During systole the right atrium fills passively with the pressure gradually rising as filling is completed. The peak of the "v" wave in the atrium is reached as diastole begins with the opening of the tricuspid valve and filling of the right ventricle.

Pressure rises rapidly in the right ventricle during systole. When ventricular pressure is greater than atrial pressure, the tricuspid valve closes. When right ventricular pressure is greater than pulmonary artery pressure, the pulmonic valve opens and ejection of blood into the pulmonary circulation begins. Pressures measured in the pulmonary artery and right ventricle are identical during systole. When contraction is complete and the right ventricle begins to relax, pressure falls. As ventricular pressure falls below pulmonary artery pressure, the pulmonic valve closes, terminating systole and beginning diastole. Right ventricular pressure continues to decline as the ventricle relaxes. When ventricular pressure falls below right atrial pressure, the tricuspid valve opens and rapid ventricular filling in early diastole commences.

Heart Sounds

The first heart sound, S1, is heard when the mitral and tricuspid valves close during contraction. The mitral valve closes first, then the tricuspid valve. The second heart sound, S2, is heard when the aortic and pulmonic valves close during relaxation of the ventricles. Normally, aortic valve closure precedes pulmonic valve closure by a few milliseconds.

When listening at Erb's point, the second sound may be heard on deep inspiration as two separate components. With a fall in intrathoracic pressure on deep inspiration, venous return to the right atrium and diastolic filling of the right ventricle increase. In emptying the added right ventricular volume, ejection time is prolonged and pulmonic valve closure delayed. Aortic valve closure is on time. S2 is heard as two distinct components: first aortic valve closure (A2), then pulmonic valve closure (P2). On expiration, pulmonic valve closure is on time and almost coincides with A2; S2 is then a single sound. These changes in auscultation are called physiologic splitting of the second sound (Fig. 3).

Abnormal splitting of the second heart sound may be heard with high pulmonary artery pressure or with delayed electrical activation of right ventricular systole. Paradoxical splitting is heard with delayed emptying of the left ventricle or with delayed electrical activation of left ventricular systole.

To distinguish the first sound from the second sound, it is necessary to know when systole occurs. If one lightly palpates the carotid artery while auscultating

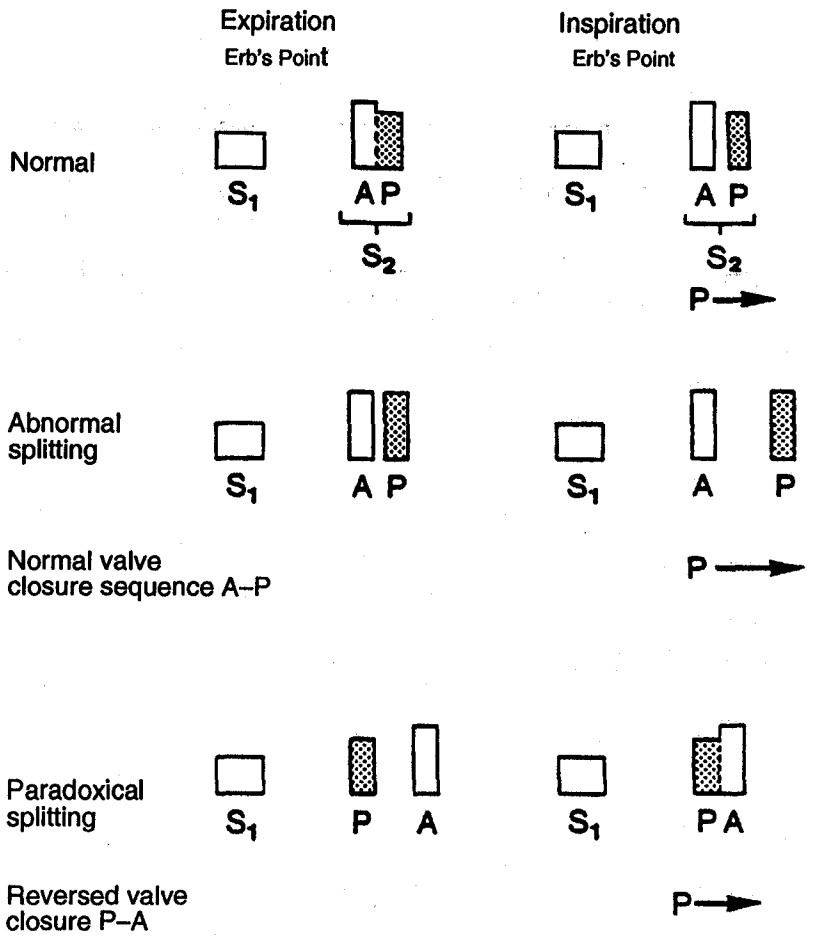

Fig. 3. Splitting of the second heart sound.

the heart, Sl will precede the carotid pulse upstroke; S2 will be heard as the pulse recedes.

Additional sounds may be heard in the cardiac cycle. These sounds may be normal or abnormal, depending on other associated changes. For example, an ejection click is a high-pitched sound in early systole. The click coincides with aortic valve opening and is thought to be made by the valve leaflets snapping open. Often the click may be so early as to appear to be a final component of Sl.

S4, a low-pitched sound that precedes systole, is auscultated at the ventricular apex. In timing, S4 coincides with atrial systole, end-diastolic ventricular filling. Most likely, S4 is made by vibrating ventricular myocardium with end-diastolic filling. Since S4 is heard in old people who have no other definable heart disease, it may be a normal sound in this group. In patients who have heart disease in which the ventricle operates at very high pressure, e.g., aortic obstruction and systemic hypertension, an S4 gallop sound is abnormal. Under these circumstances, the ventricular myocardium is hypertrophied; myocardial compliance is reduced–i.e., the ventricle is less able to expand to accommodate an added volume of blood (see discussion of diastolic properties of the ventricle in CLS 130). The S4 gallop is a low-pitched sound that coincides with atrial systole, preceding ventricular systole. When abnormal, it is not only heard but may be palpated as a presystolic impulse

at the apex of the left ventricle. S4 is never heard during atrial fibrillation, since atrial systole is absent.

S3 is a low-pitched sound heard at the ventricular apex. The S3 sound is heard during early diastole during rapid ventricular filling, about 150 milliseconds after aortic valve closure. S3 is produced by low-frequency vibration of the ventricular myocardium during rapid ventricular filling. It is a normal sound when no other indication of heart disease is apparent in patients up to 30 years of age.

S3 gallop is an abnormal sound that coincides with rapid ventricular filling, 150 milliseconds following aortic closure. It is produced by vibrations of the ventricular myocardium and is very low in pitch. In the presence of heart disease in children and adults, an S3 gallop sound is a physical sign of ventricular failure. Ventricular failure is defined as an ejection fraction less than 50% in systole. It is frequently associated with a high end-diastolic volume and end-diastolic pressure. An S3 gallop sound may be visible and palpable as an impulse following the apical impulse during ejection.

Heart Murmurs

A heart murmur is a sound produced by turbulence of blood or eddy formation. The murmurs to be discussed are caused by specific valve deformities that modify normal blood flow. Murmurs are heard in either systole or diastole. It is most important, when describing a murmur, for a physician to characterize its timing and duration; pitch and quality; its location, i.e., where it is loudest; and its radiation.

Ejection murmurs are heard in systole. The murmur begins after the first heart sound and may start with an ejection click. The murmur begins softly, becomes louder, peaks, and then softens, i.e., crescendo–decrescendo or diamond shaped. A systolic ejection murmur always ends before S2. In describing the murmur, one should note when it peaks—in early-, mid-, or late-systole. The murmur may be palpable as a "thrill" over the anatomic area of the affected valve. The presence of a systolic ejection murmur suggests obstruction to flow across a semilunar valve, aortic or pulmonic. They may, however, be heard as functional murmurs in the absence of organic heart disease. Systolic ejection murmurs are common in high output states, such as anemia or hyperthyroidism. When no valve deformity is present, the murmur may resolve with return to a normal metabolic state.

Pansystolic (holosystolic) murmurs are also systolic murmurs. They begin with Sl and are heard throughout systole, blending into the second heart sound. These murmurs are typical of atrioventricular valve regurgitation, i.e., involving mitral and tricuspid valves. Pansystolic murmurs are also characteristic of ventricular septal defect. They may be either high or low in pitch.

Diastolic murmurs will be discussed with specific valve problems, mitral valve obstruction, and aortic valve regurgitation.

Systolic and diastolic murmurs may be heard in patients with multiple valve problems, for example, patients with rheumatic heart disease having mitral valve regurgitation and aortic valve regurgitation.

Continuous murmurs are heard in all parts of the cardiac cycle and originate from one anatomic defect in which flow is continuous during systole and diastole, e.g., patent ductus arteriosus and arterial-venous fistula.

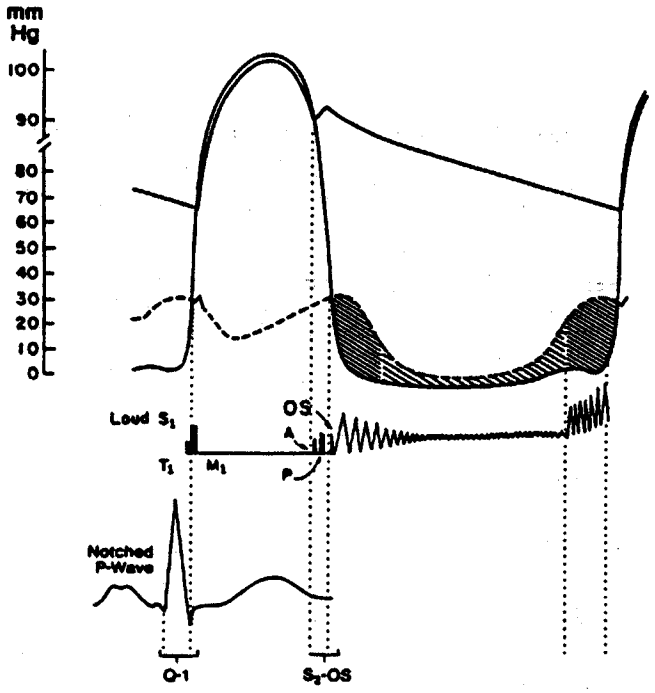

Fig. 4. Mitral valve obstruction.

EXAMPLES OF COMMONLY AUSCULTATED HEART MURMURS

Mitral valve obstruction

Mitral valve obstruction is most often caused by rheumatic heart disease with scarring of the valve leaflets, fibrosis, and shortening of the chordae tendineae. During diastole the valve leaflets open partially, limiting free flow of blood from left atrium to left ventricle. Flow across the thickened leaflets is turbulent, producing a low-pitched murmur during diastole.

If one measures left atrial pressure and left ventricular pressure simultaneously in diastole, there is a gradient across the mitral valve: i.e., diastolic pressure in the left atrium is higher than in the left ventricle. With significant mitral valve obstruction there is no rapid filling phase of the ventricle. The rate of flow across an obstructed valve is slowed, with flow extending uniformly through diastole. If sinus rhythm is present, atrial systole may slightly enhance end-diastolic flow.

With mitral valve obstruction, diastolic flow into the left ventricle may be reduced significantly if the heart rate becomes very rapid. Rapid heart rate shortens diastole, reduces time for left ventricular filling, and reduces cardiac output. Left atrial pressure increases if the atrium empties incompletely during diastole. Loss of atrial systole also reduces diastolic filling of the left ventricle.

The characteristic murmur of mitral valve obstruction is heard only at the apex of the heart. The bell of the stethoscope is lightly applied to the apex while the patient rests in the left lateral position. The murmur is a low-pitched rumbling sound that begins with the opening of the mitral valve. It is produced by turbulence during the passive flow of blood from left atrium to left ventricle. The murmur may become louder in end diastole (presystole), when atrial systole increases flow across the obstructed valve. In atrial fibrillation (a cardiac arrhythmia in which atrial systole is lost as a result of very rapid repetitive atrial stimuli at about 400 to 500 times per minute), the presystolic accentuation of the murmur is absent.

When the mitral valve is thickened but not calcified, it resists movement. When isovolemic contraction begins, the valve snaps shut quickly as ventricular pressure exceeds atrial pressure. S1 is louder than normal and may be described as snapping in quality.

Similarly, when, in early diastole, left atrial pressure is higher than ventricular pressure, the valve snaps open. Opening of a thickened, obstructed mitral valve is heard as a high-pitched sound following S2. This abnormal sound is called an ***opening snap,*** characteristic of mitral valve obstruction. Time between A2 and the opening snap may be a clue to the severity of the obstruction: the greater the mitral valve obstruction, the higher the left atrial pressure. The higher the left atrial pressure, the sooner after A2 the atrial pressure exceeds ventricular pressure, and thus, the earlier the mitral leaflets snap open. Consequently, in severe mitral valve obstruction with very high left atrial pressure, the opening snap may be heard closely following A2. However, with mild mitral valve obstruction and low left atrial pressure, the time between A2 and opening snap is long.

If the mitral valve is calcified, leaflet mobility is reduced markedly. S1 is not accentuated and may in fact be diminished. The opening snap may be absent, and the characteristic holodiastolic rumble remains.

Aortic valve obstruction

Aortic valve obstruction may be caused by a variety of etiologies. Congenital aortic valve deformity, i.e., bicuspid valve instead of tricuspid, is common. Calcification of a tricuspid valve with progressive obstruction is seen in patients in the sixth and seventh decades of life. When associated with other valve deformities, aortic valve obstruction may be rheumatic in origin.

In congenital obstruction the valve may be unicuspid or bicuspid. Often the valve is not calcified but has a fixed opening with fusion of commissures. At the start of systole the whole valve may snap open as a domed structure without apparent commissures, producing a loud ejection click. The murmur that is heard in aortic valve obstruction, which is ejection in quality, begins after S1, becomes louder until it peaks, and then declines, ending before S2. The more obstructed the valve, the later the peak of the murmur. Since ejection is delayed by aortic valve obstruction, palpation of the carotid pulse will reveal delay, too, in the peak of the pulse. Again, the more stenotic the valve, the later the peak of the carotid or brachial pulse. Aortic valve closure may also be delayed, and in severe obstruction may follow pulmonic valve closure, producing paradoxical splitting of S2. The murmur is best heard at the base to the right of the sternum, and may be harsh, low-, or high-pitched. The murmur radiates to the neck and may be heard by listening over the carotid arteries.

If one were to measure simultaneous pressures in the left ventricle and in the aorta, there would be a gradient across the aortic valve during systole: i.e., intraventricular pressure would be greater than intraaortic pressure. Assuming constant cardiac output, the higher the gradient across the aortic valve in systole, the more severe the obstruction, and the smaller the valve orifice. To achieve very high pressure within the left ventricle, the myocardium hypertrophies with time. Hypertrophy of the left ventricle decreases compliance in diastole. An S4 gallop is common in significant aortic valve obstruction.

In calcific aortic valve obstruction, as seen in older patients with tricuspid

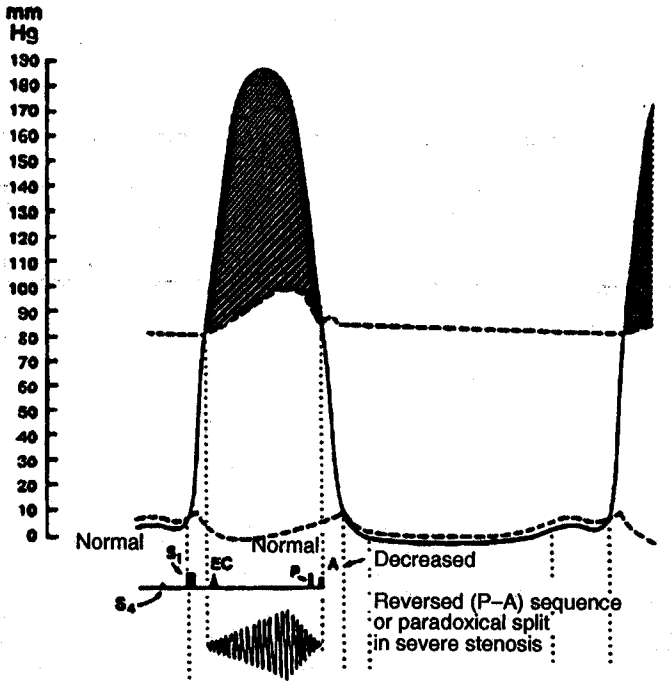

Fig. 5. Aortic valve obstruction.

valves, heart sounds are altered. The valve moves little during the cardiac cycle, and thus, no ejection click is heard when the aortic valve opens. Similarly, A2 is reduced or absent when the semilunar valves close. S2 may be a single sound made by P2 alone.

Mitral valve regurgitation

Anatomically, the mitral valve is a very complex structure. It consists of a fibrous ring and two leaflets, anterior and posterior. The anterior leaflet also forms a part of the aortic outflow tract and is continuous with the aortic wall. The posterior leaflet is continuous with the left atrium. Both leaflets are connected by delicate fibers–chordae tendineae–to the two papillary muscles, themselves intimately related to left ventricular myocardium. Any anatomic alteration in these components of the mitral valve apparatus may alter its competency. Indeed, many different pathologic processes may produce mitral valve regurgitation.

If the valve leaflets become incompetent during systole, blood will flow retrograde into the left atrium, as well as across the aortic valve into the systemic circulation. Blood flow through an incompetent mitral valve may occur when ventricular pressures are normal. Flow usually begins with isovolemic contraction and continues through systole: i.e., the murmur is holosystolic, beginning with Sl and ending after aortic valve closure when left ventricular pressure falls below left atrial pressure. The murmur may be heard all over the precordium, but is usually loudest at the apex. This murmur commonly radiates to the left axilla and may be heard in the back beneath the left scapula. The murmur is relatively high-pitched and blowing in quality.

Another murmur of mitral valve regurgitation has been described. The valve is competent during the initial portion of systole. In midsystole one or both valve leaflets may prolapse into the left atrium and be followed by mitral valve regurgita-

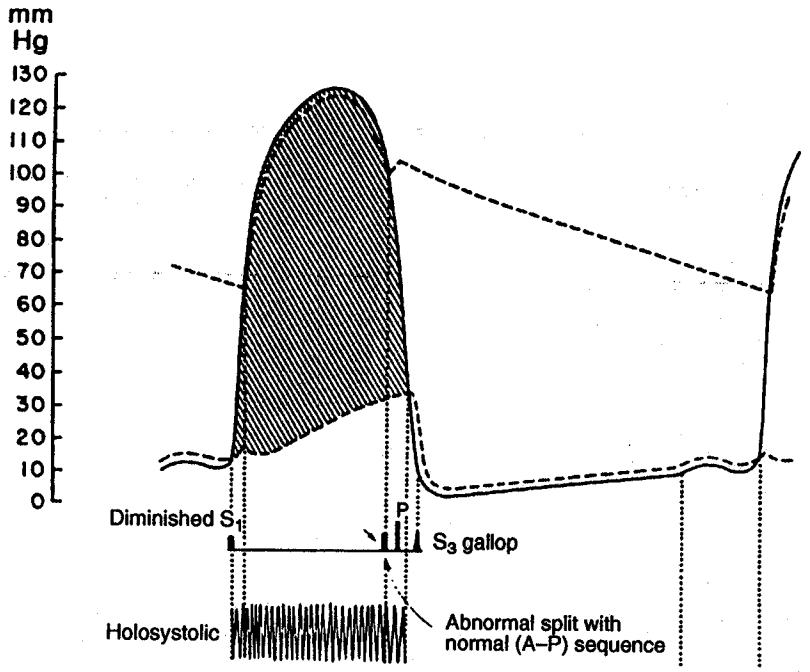

Fig. 6. Mitral valve regurgitation.

tion for the duration of systole. In this case Sl is normal, and the initial part of systole sounds normal. As the valve leaflet prolapses, a high-pitched click is heard, followed by the typical murmur of mitral valve regurgitation that continues into S2. It has been called "midsystolic click, late systolic murmur," or mitral valve prolapse.

Aortic valve regurgitation

Aortic valve regurgitation, too, may be caused by multiple etiologies: rheumatic heart disease, syphilis, dissecting aortic aneurysm, Marfan's syndrome, ankylosing spondylitis, infective endocarditis, etc. In aortic valve regurgitation the valve leaflets do not completely come together during diastole. Consequently, blood ejected during systole may flow back into the left ventricle during diastole. As with normal ventricular filling, flow is greatest in early diastole but may continue throughout diastole. To maintain a normal cardiac output, the ventricle must eject a volume of blood equal to normal flow plus the regurgitant volume. The end-diastolic and stroke volumes in aortic regurgitation are larger than normal.

Characteristically, there are two murmurs heard in a patient with severe aortic valve regurgitation. In systole, there is an early or midsystolic ejection murmur produced by the high-volume flow across the valve. There is no significant pressure gradient across the aortic valve in pure aortic valve regurgitation. In diastole, one hears a high-pitched diastolic murmur, which is loudest in early diastole during rapid flow and continuing through diastole. The murmur is decrescendo in quality, best heard at Erb's point (lower left sternal border) and apex.

The quality of the carotid pulse is pathognomonic for aortic regurgitation. In systole a large volume is ejected rapidly. The pulse is forceful and reaches its peak early. In diastole a large volume moves retrograde into the ventricle. The pulse falls off quickly as well, with low diastolic pressure recordings.

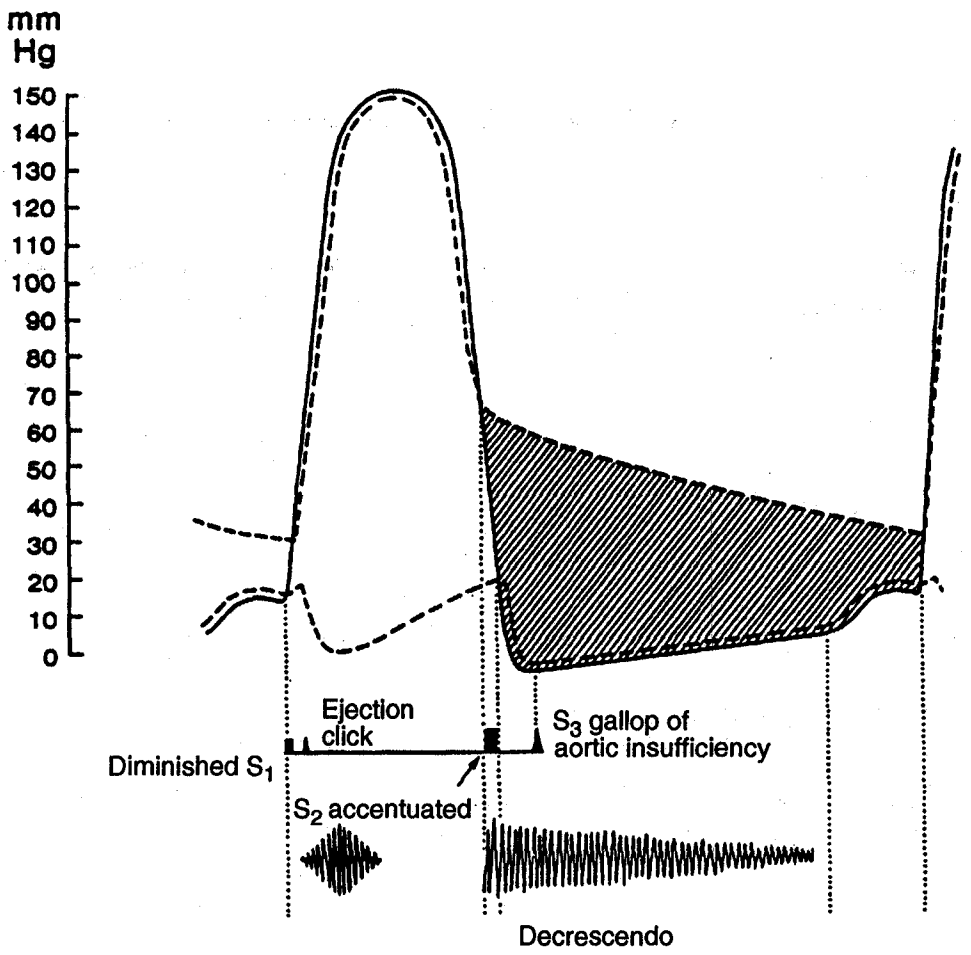

Fig. 7. Aortic valve regurgitation.

A third sound (S3) is often heard at the apex during rapid filling in early diastole.