27 Diastolic Murmurs

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Definition

A *diastolic murmur* is a sound of some duration occurring during diastole. All diastolic murmurs imply some alteration of anatomy or function of the cardiovascular structures. The four most commonly encountered diastolic murmurs include aortic and pulmonary valve regurgitation, and mitral and tricuspid valve rumbles (Table 27.1). Compared to most systolic murmurs, diastolic murmurs are usually more difficult to hear, and certain auscultatory techniques are essential for their detection.

Technique

The murmur of *aortic regurgitation* begins with the aortic component of the second sound and is decrescendo in intensity for a variable duration of diastole. It is usually a high-frequency, "blowing" sound, most often heard best along the left lower sternal border, although occasionally only in the second right intercostal space. It may be of maximum intensity along the right sternal border (see discussion later in this chapter). Rarely, the murmur may be isolated at the apex impulse.

For detection, first think of a blowing, high-frequency sound coming from a distance (to simulate it, purse your lips very tightly and blow). Place the diaphragm of the stethoscope along the left sternal border with very firm pressure, enough pressure to leave a slight indentation on the skin when removed. The fingers may be used to hold the stethoscope, but to avoid the extraneous noise from tremor of the finger muscles, the palm of the hand may be better. The patient should be instructed "don't breathe" at end expiration, or told to "take a deep breath, blow it all out, then relax and don't breathe." A command to "hold your breath" may cause the patient to take in a deep breath and hold it. If the murmur is not heard at the left lower sternal border with the patient supine, auscultation in a similar fashion should be performed at the second right intercostal space and along the right sternal border. The murmur may only be heard by listening in one of these areas with the patient sitting, leaning forward in relaxed expiratory apnea. Any bedside maneuver that transiently increases blood pressure may intensify or bring out the murmur. Hand grip or squatting can be useful. Proper timing of the cardiac cycle is essential. A heart rate of 100 or greater abbreviates diastole so that systolic and diastolic duration are nearly equal. In this situation even a loud murmur of aortic regurgitation may be mistaken for a systolic murmur. Simultaneous palpation of the carotid pulse is essential to avoid this error.

Table 27.1
Diastolic Murmurs

- I. Aortic regurgitation
- II. Pulmonary valve regurgitation
- III. Mitral rumble
  - A. Obstruction to flow
    1. Mitral stenosis (rheumatic, congenital)
    2. Left atrial myxoma
    3. Cor triatriatum
    4. Localized pericardial constriction
  - B. Increased flow
    1. Mitral regurgitation
    2. Ventricular septal defect
    3. Patent ductus arteriousus
    4. Complete heart block
- IV. Tricuspid rumble
  - A. Obstruction to flow
    1. Tricuspid stenosis (rheumatic, Ebstein's anomoly, carinoid)
    2. Right atrial myxoma
    3. Localized pericardial constriction
  - B. Increased flow
    1. Atrial septal defect
    2. Tricuspid regurgitation

The murmur of *pulmonary valve regurgitation* associated with pulmonary hypertension is an early diastolic, decrescendo murmur beginning with the pulmonary component of the second sound, best heard along the upper left sternal border. Auscultatory techniques are like those for aortic regurgitation. The quality of pulmonary valve regurgitation is similar to that of aortic regurgitation, and differentiation may be difficult. The murmur of pulmonary valve regurgitation may increase in intensity with inspiration. In association with mitral regurgitation, intensity may actually decrease with inspiration. The presence of bounding pulses and a wide pulse pressure support the diagnosis of aortic regurgitation. Pulmonary valve regurgitation frequently results from severe pulmonary hypertension. When the murmur is associated with mitral stenosis and pulmonary hypertension, it usually represents trivial aortic regurgitation simply because aortic regurgitation is more common than pulmonary valve regurgitation in this clinical setting.

The murmur of pulmonary valve regurgitation without associated pulmonary hypertension, as in pulmonary valve endocarditis or congenital abnormalities of the pulmonary valve, is of lower frequency and may be middiastolic with a crescendo–decrescendo pattern of intensity.

A *mitral valve rumble* is a diastolic murmur of low frequency occurring in middiastole and/or late diastole (presystole). It is frequently localized to a small area at the apex impulse. The patient should be relaxed in a left lateral decubitus position and the apex impulse localized. The bell of the stethoscope should be applied with *very light* pressure, just enough to make contact with the skin. Concentrate on diastole and move the bell over and just adjacent to the apex impulse. Listen in middiastole and just before the first sound. If the murmur is due to mitral stenosis, there may be accentuation of the first sound and an opening snap. The opening snap is a high-frequency sound that introduces the

middiastolic component of the rumble and occurs .03 to .14 second after the second sound. Maneuvers that transiently increase cardiac output, such as sit-ups, coughing, or squatting, may aid in detection. When the apex impulse is not easily located, scanning the area, listening for the point of maximum intensity of the heart sounds, can help identify the apex impulse and the area on which to concentrate for the mitral diastolic rumble.

A *tricuspid valve rumble* has similar characteristics as the mitral rumble, but is localized along the left lower sternal border and increases in intensity with inspiration. The bell should be placed, again with *very light* pressure, exploring from the third to the fifth interspaces, concentrating in diastole both during inspiration and expiration. Similar maneuvers to increase venous return may augment the murmur. The inspiratory accentuation aids in differentiation from the mitral rumble, although the latter does not usually radiate to the left sternal border. Accentuation of the first sound (tricuspid component) and a tricuspid opening snap may also be present. The presystolic component of the tricuspid valve rumble is often crescendo–decrescendo, unlike the crescendo pattern of the mitral rumble.

Basic Science

Aortic valve regurgitation is the result of a loss of perfect apposition of the aortic cusps in diastole. There may be deformity of the cusps or the supporting structures. The problem may develop gradually, as in rheumatic heart disease, or acutely, as in bacterial endocarditis. The symptoms and physical findings depend on the severity of the regurgitation and the duration of its development. A minimal regurgitant volume causes no abnormalities other than the presence of the murmur. The regurgitant volume increases diastolic filling of the left ventricle, and as this gradually progresses, there is an increase in stroke volume, left ventricular dilatation, and hypertrophy. The peripheral pulses are hyperdynamic, and the pulse pressure is widened. An apical diastolic rumble (Austin-Flint) may be heard at the apex. This is due to partial closure of the anterior leaflet of the mitral valve. The apex impulse becomes laterally displaced and sustained. Resting and exercise cardiac output is maintained until progressive left ventricular dilatation causes impairment of myocardial function. Easy fatigue and dyspnea ensue. Severe left ventricular dilatation causes inadequate apposition of the mitral leaflets, and a murmur of mitral regurgitation is heard. As left ventricular diastolic pressure increases and is reflected into the left atrium and pulmonary veins, eventual pulmonary hypertension, right ventricular dilatation, tricuspid regurgitation, and elevated jugular venous pressure develop. The presence of a widened pulse pressure, a murmur that continues throughout diastole, and an apical diastolic rumble (Austin-Flint rumble) imply significant aortic regurgitation even before the development of pulmonary hypertension and right heart failure.

Pulmonary valve regurgitation is the result of imperfect cusp apposition resulting from deformity of the cusps or the supporting structures. It is most frequently secondary to severe pulmonary hypertension with dilatation of the supporting structures. In such cases, the regurgitant volume is small and of no hemodynamic importance. Pulmonary valve regurgitation may also occur with normal pulmonary artery pressures. In such cases, even a large regurgitation volume is usually well tolerated by the right ventricle. There is right ventricular dilatation and elevation of the jugular venous pressure. Rarely will severe right heart failure develop.

The *mitral valve rumble* may be caused by normal or reduced blood flow through a stenotic valve, as in mitral stenosis. Left atrial myxoma, cor triatriatum, and localized left atrioventricular groove pericardial constriction may also cause a left atrial left ventricular diastolic blood pressure gradient and a mitral valve rumble. Situations that cause an increase in diastolic mitral valve blood flow through a normal valve may also cause a rumble, as in mitral regurgitation, ventricular septal defect, patent ductus arteriosus, and complete heart block. Aortic regurgitation of moderate severity is also associated with a diastolic rumble (Austin-Flint) and results from the regurgitant volume causing vibrations of the mitral leaflet and/or displacement of the anterior leaflet toward the closed position.

Mitral valve stenosis is most commonly the result of previous rheumatic fever that later causes leaflet scarification and fusion commissures. With only mild reduction in orifice size, a middiastolic or presystolic murmur is heard (time of peak diastolic flow), and similarly, only a small pressure gradient is present. As the anterior mitral leaflet stiffens, but is still mobile, its opening produces a sound, the opening snap, and its closure accentuates the intensity of the first heart sound. As stenosis progresses, left atrial pressure rises in proportion to orifice reduction and flow (cardiac output). Eventual rises in left atrial pressure are reflected into the pulmonary veins, capillary bed, and pulmonary arteries. Even with mild stenosis, pulmonary edema may develop acutely with a rapid heart rate causing abbreviation of diastolic filling time. As left atrial pressure increases, the leaflets open more quickly in diastole, and the opening snap moves closer to the second sound. As pulmonary hypertension develops, the pulmonary component of the second sound is accentuated. Symptoms at rest may be minimal, but with exercise left atrial pressure increases further, and dyspnea is a prominent symptom. With further progression, cardiac output does not increase appropriately with exercise, and easy fatigue occurs. Pulmonary hypertension initially results in right ventricular hypertrophy and a parasternal lift is felt; if pulmonary hypertension is of long standing, right ventricular dilatation, tricuspid regurgitation, and systemic venous hypertension develop. "A" waves are prominent in the jugular venous pulsations until the development of tricuspid regurgitation, when the "v" wave is large. Calcification of the mitral leaflets may produce immobility, and the opening snap and accentuated first sound are lost. As right ventricular dysfunction develops, even resting cardiac output is reduced; this reduction in mitral valve flow will soften the intensity of the rumble.

The *tricuspid valve rumble*, caused by valvular deformity and orifice stenosis, may be secondary to rheumatic heart disease, Ebstein's anomaly, or carcinoid heart disease. Right atrial myxoma may also cause a diastolic tricuspid obstruction. The rumble may also result from increased flow across a normal valve as in atrial septal defect or tricuspid regurgitation.

Tricuspid valve stenosis may be congenital in origin but is most commonly the result of rheumatic heart disease and is rarely an isolated valvular lesion. In such cases the manifestations may be subtle. The diastolic rumble and opening snap are similar to mitral stenosis. The location, left lower sternal border, and inspiratory accentuation aid in differentiation. The jugular venous pulsations show attenuation of the "y" descent and prominence of the "a" wave. More

severe tricuspid stenosis can cause hepatomegaly, ascites, peripheral edema, and exercise intolerance.

Clinical Significance

Any of the diastolic murmurs may be present without any alteration of cardiac function. Their detection remains important for proper care of the patient. The presence of any murmur that could be caused by an alteration in cardiac structures indicates the need for bacterial endocarditis prophylaxis. These patients should be evaluated on a regular basis for progression of the valvular problem. If prior rheumatic fever is a possible cause, prophylaxis for recurrence should be given.

For these reasons, diastolic murmurs should be diligently sought in every patient who is examined. In patients with certain symptoms, physical findings, or laboratory abnormalities, specific valvular problems should be considered. The presence of other apparent noncardiac problems may also be helpful clues.

Aortic valve regurgitation should be considered in patients with any of the following complaints or findings: dyspnea, fatigue, angina pectoris, congestive heart failure, chest pain suggestive of aortic dissection, symptoms or signs suspicious for endocarditis, the presence of a wide pulse pressure or hyperdynamic pulses, radiographic evidence of an enlarged aorta (especially the ascending portion). All demand a careful search for the murmur of aortic valve regurgitation. Once the diagnosis is established, etiology and severity should be addressed. There are many causes of aortic valve regurgitation, some of which have an important influence on management and prognosis. A patient with only mild to moderate aortic valve regurgitation due to rheumatic heart disease would not require cardiac surgery, but if aortic dissection is the etiology, emergent surgery may be necessary. With bacterial endocarditis, prompt antibiotic therapy is essential, and the patient should be monitored for rapid progression of the aortic regurgitation requiring emergent surgery.

Pulmonary valve regurgitation is most commonly caused by pulmonary hypertension. Although pulmonary hypertension of any cause may result in pulmonary valve regurgitation, that due to mitral stenosis, cor pulmonale, recurrent pulmonary emboli, or primary pulmonary hypertension is most common. Less common causes of pulmonary valve regurgitation include endocarditis, rheumatic fever, cardiac tumors, and carcinoid. Bacterial endocarditis isolated to the pulmonary valve may present as a protracted febrile illness. The character of the murmur may change from one associated with a normal pulmonary artery pressure to one associated with pulmonary hypertension if the illness is complicated by recurrent septic pulmonary emboli. Echocardiography, both M-mode and two-dimensional, and Doppler studies may be useful tools in the recognition of pulmonary valve regurgitation and aid in the determination of etiology.

The presenting problems of patients with *mitral valve stenosis* are also variable. Exertional dyspnea, hemoptysis, paroxysmal or sustained atrial fibrillation (especially with a vertical QRS axis), systemic emboli, acute pulmonary edema (especially developing coincident with atrial fibrillation), hoarseness, radiographic evidence of enlarged pulmonary arteries (especially with left atrial enlargement) demand a careful search for mitral valve stenosis. In cases of severe, long-standing mitral valve stenosis, signs and symptoms of congestive heart failure, ascites, jaundice, and peripheral edema may be the presenting problem. Left atrial myxoma may mimic mitral valve stenosis by both symptoms and physical findings. This diagnosis should be considered if there are signs and symptoms of a systemic illness (e.g., weight loss, fever, anemia or polycythemia, elevated sedimentation rate or immunoglobulins). An early diastolic heart sound, the tumor plop, should be sought on auscultation. M-mode and two-dimensional echocardiography is a valuable tool not only for the diagnosis of mitral stenosis but also for the recognition of left atrial myxoma; it should be utilized in any patient in whom mitral stenosis is suspected.

Since *tricuspid valve stenosis* most commonly occurs concomitantly with other rheumatic valvular lesions, it may not be easily recognized. Tricuspid valvular obstruction may cause prominent "a" waves in the jugular venous pulsations, hepatomegaly, ascites, and peripheral edema. Electrocardiographic evidence of right atrial abnormality in a patient with mitral valve stenosis may also serve as a helpful clue to the presence of tricuspid valve stenosis.

The carcinoid syndrome should be considered in patients with either *tricuspid* or *pulmonary valve* lesions, since stenosis or regurgitation of either valve can occur. Cutaneous flushing, bronchospasm, diarrhea, and symptoms of peptic ulcer disease are common noncardiac problems for these patients.

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