

CLINICAL COLUMN

Heart Sounds: Are You Listening? Part 2

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Abstract

The first of this two-part article on heart sounds was in the Spring 2013 issue of the *Canadian Journal of Cardiovascular Nursing* (Reimer-Kent, 2013). Part 1 emphasized the importance of all nurses having an understanding of heart sounds and being proficient in cardiac auscultation. The article also focused on an overview of the fundamentals of cardiac auscultation and basic heart

sounds. This article provides an overview of the anatomy and pathophysiology related to valvular heart disease and describes the array of heart sounds associated with stenotic or regurgitant aortic and mitral valve conditions.

Key words: valvular heart disease pathophysiology, systolic murmurs, diastolic murmurs, aortic stenosis, mitral stenosis, aortic regurgitation, mitral regurgitation

Anatomy of the Aortic and Mitral Valves

Understanding normal valve anatomy is foundational to understanding abnormal heart sounds associated with valvular heart disease. Figure 1 illustrates the four cardiac valves. Emphasis in this article is on the mitral valve (MV), also known as an atrioventricular valve and the aortic valve (AV), also known as a semilunar valve. Valve orifices (annulus) are formed from the fibrous skeleton of connective tissue that separates the atria and ventricles.

Connected to the AV annulus are three half-moon-shaped cusps, which are named according to the proximity of their respective coronary arteries (i.e., left coronary cusp, right

coronary cusp, and non-coronary cusp). When open the AV measures approximately 3 to 4 cm² (Bonow et al., 2006; Cary & Pearce, 2013; Klabunde, 2011).

Connected to the MV annulus are the anterior and posterior leaflets. Unlike the attachment free AV, the MV is a sophisticated structure. The leaflets are attached to chordae tendineae, which are connected to papillary muscles, which anchor the entire valve to the left ventricular wall. For the MV to function properly, there needs to be integrity among all of these components. When open, the MV area measures approximately 4 to 5 cm² (Bonow et al., 2006; Klabunde, 2011; Turi, 2004).

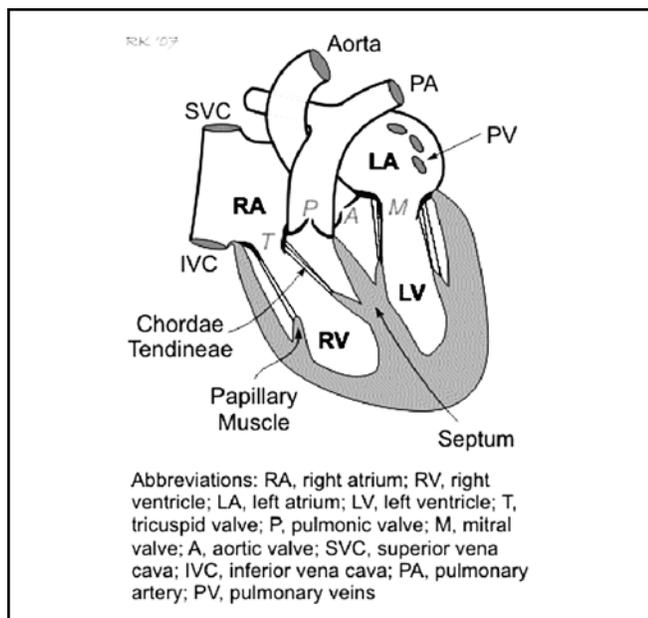


Figure 1: Anatomy of the Aortic and Mitral Valves
 Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD001.htm>
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Systolic and Diastolic Murmurs

Skilled clinicians can diagnose the cause of a heart murmur by pinpointing the sound in relation to its timing within the cardiac cycle, location, radiation, and intensity. To discern the significance of a murmur requires critical thinking on the part of the practitioner and correlating the findings with the patient's appearance and history. Murmurs arise from stenotic (obstructive) or regurgitant (incompetent) valve conditions, abnormal connections between right and left heart circulations, and conditions that obstruct flow across the valve that mimic stenosis such as a left atrial myxoma. Functional systolic and diastolic murmurs can develop as the heart adapts to stenotic or regurgitant states. Innocent systolic flow murmurs can also develop from high output states (e.g., pregnancy) (Alpert, 1990; Choudhry & Etchells, 1999; Crawley, 1990; Curtin, & Griffin, 2010; Turi, 2004; University of California San Diego, 2008; University of Washington, n.d.).

To distinguish the timing of the murmur within the cardiac cycle (systole or diastole), palpate the carotid pulse or observe the cardiac rhythm as outlined in Part 1 (Reimer-Kent, 2013). A systolic murmur is heard between the first heart sound (S₁) and the second heart sound (S₂) and accompanies aortic

stenosis (AS) or mitral regurgitation (MR). A diastolic murmur is heard between S_2 and S_1 and accompanies aortic regurgitation (AR) or mitral stenosis (MS).

Diastolic murmurs are classified as early, mid, or late. Systolic murmurs are also classified as early, mid, late, or holosystolic (i.e., pansystolic). An early systolic murmur obliterates S_1 and extends to mid or late systole and sounds like “Shshsh Dub”. With a mid-systolic murmur both early and late systole are murmur free and it has a crescendo-decrescendo character and sounds like “Lub Shshsdub”. A late systolic murmur starts with the second half of systole and obliterates S_2 and sounds like “Lub Shshshs”. A holosystolic murmur obliterates S_1 and S_2 and sounds like “Shshshshshshshshs” (Alpert, 1990; Crawley, 1990; Klabunde, 2011; University of California San Diego, 2008; University of Washington, n.d.).

It is also important to note the location of where the murmur is loudest as well as whether and in which direction and areas it radiates.

Murmurs are rated for intensity using a six point grading system expressed as a fraction. Grade 1/6 is very faint; grade 2/6 is still quiet but heard immediately; grade 3/6 is moderately loud; grade 4/6 is loud with a palpable thrill; grade 5/6 is heard with the stethoscope partly off the chest wall; and grade 6/6 is so loud no stethoscope is needed (Choudhry & Etchells, 1999; University of California San Diego, 2008; University of Washington, n.d.).

Stenotic and Regurgitant Valvular Heart Disease

Pathophysiology of Aortic Regurgitation

In AR the AV cusps are still open during diastole. This causes hemodynamic volume overload on the left ventricle (LV) (see Figure 2). In diastole retrograde blood flows from the aorta (AO) into the LV and antegrade blood flows from the left atrium (LA) into the LV.

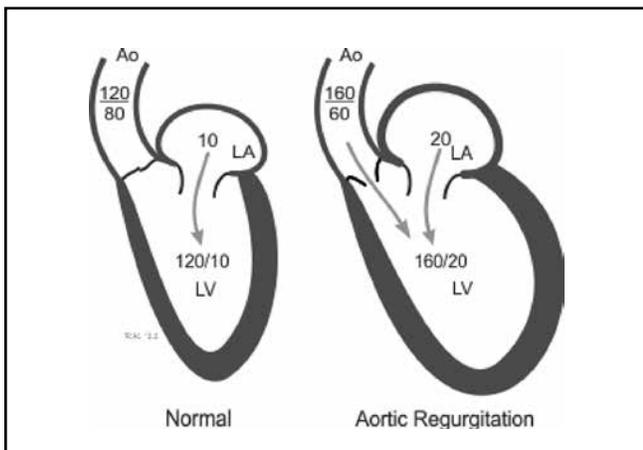


Figure 2: Pathophysiology of Aortic Regurgitation
Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD005.htm>
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AR can develop acutely as when the valve is destroyed by infective endocarditis (IE) or result from damage to the AO near the AV (e.g., acute aortic dissection/aneurysm or chest trauma). Acute AR is accompanied by pulmonary edema, shock and early death as the result of the sudden volume overload on a normal LV. Both left ventricular end-diastolic pressure (LVEDP) and left atrial pressure (LAP) increase rapidly and dramatically with a corresponding decrease in forward stroke volume (SV) and cardiac output (CO). Acute AR can be misdiagnosed as a non-valvular problem as the classical heart sounds of AR may be missing. These patients require vigilant monitoring as immediate cardiac surgery may be warranted (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Hamirani et al., 2012; Klabunde, 2011; Novaro, 2010; Vahanian et al., 2007).

AR can also develop gradually and be chronic, which creates a combined volume and pressure overload problem for the LV. The LV remodels to become more spherical in shape and compliant to regurgitant volume giving SV a chance to increase to maintain an effective CO. These patients can be asymptomatic for many years. In time, the heart will decompensate and pulmonary hypertension and right-sided heart failure will occur. Chronic AR arises from rheumatic heart disease (RHD), annular stretch from left ventricular dilatation, diseases that affect the connective tissue of the AO (e.g., Marfan's syndrome), degenerative deterioration with age, iatrogenic (e.g., catheter perforation of the AV), congenital heart defect, or inflammatory processes (e.g., vasculitis, aortitis, arteritis, reactive arthritis) (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Hamirani et al., 2012; Klabunde, 2011; Novaro, 2010; Reimer-Kent, 2007; Vahanian et al., 2007).

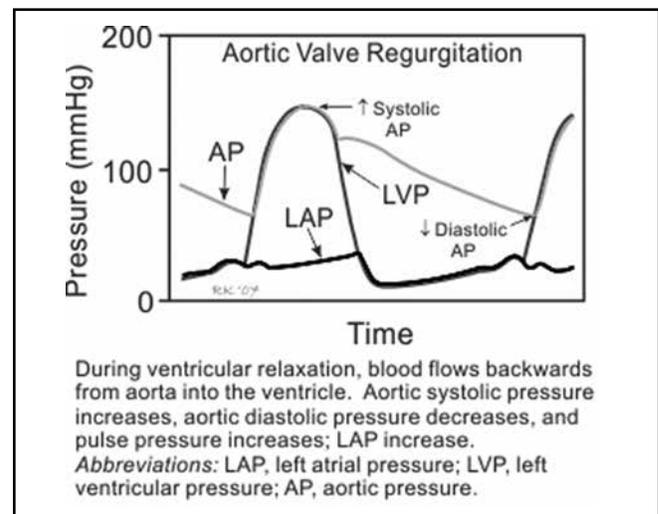


Figure 3: Aortic Regurgitation in Relation to the Cardiac Cycle
Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD005.htm>
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With AR there is no true isovolumetric relaxation or isovolumetric contraction (see Figure 3). The LV does not get a chance to relax because as soon as diastole begins, even before the MV opens, blood from the AO begins to fall back into the LV. Once the MV does open the LV must accommodate the continual retrograde blood flow from the AO as well as the antegrade blood flow from the LA. This double source of blood flow will significantly increase left ventricular end-diastolic volume (LVEDV) (i.e., preload). The Frank-Starling mechanism will be activated and initially SV will increase. The pulse pressure will also widen (e.g., greater than 50 mm Hg) with a higher than normal systolic pressure, from the force with which the large volume of blood is ejected forward during systole and a lower than normal diastolic pressure, from the rapid fall in aortic pressure as blood flows backward into the LV during diastole (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Hamirani et al., 2012; Klabunde, 2011; Novaro, 2010; Vahanian et al., 2007).

Heart Sounds Associated with Aortic Regurgitation

Several heart sounds are associated with AR. The retrograde blood flow through an incompetent AV into the LV causes a high-pitched, quiet, blowing, puffing, decrescendo, early diastolic murmur, which sounds like “Lub Pewwwwww” (see Figure 4). The murmur is early as this correlates with high aortic pressure and decrescendo as this correlates with the fall in aortic pressure that happens with retrograde blood flow into the LV. A high-pitched AR murmur is best heard by placing the diaphragm of the stethoscope firmly on the chest wall. Listen along the left sternal border at the third or fourth intercostal space with the patient sitting upward, leaning forward and at the end of expiration. Manoeuvres such as isometric exercise will increase systemic vascular resistance and accentuate the AR murmur. There is a positive relationship between the duration of the diastolic murmur and the severity of AR (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Hamirani et al., 2012; Klabunde, 2011; Novaro 2010).



Figure 4: Aortic Regurgitation Murmur
<http://www.fastbleep.com/medical-notes/heart-lungs-blood/1/23/151>
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Due to the large SV travelling across the AV a short systolic aortic flow murmur that sounds like “Lub shshs Pewww” may be heard.

There may also be an apical low-pitched, soft, rumbling Austin Flint murmur late in diastole or presystole. Best heard at the apex with the patient left-side lying, this murmur is thought to be related to functional mitral stenosis (MS) where the normal anterior MV leaflet is compressed by regurgitant blood flow causing an obstruction to antegrade blood flow from the LA to the LV during diastole (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Hamirani et al., 2012; Klabunde, 2011; Novaro 2010; University of California San Diego, 2008; University of Washington, n.d.).

Pathophysiology of Mitral Regurgitation

In MR the MV leaflets are still open during systole. This causes hemodynamic volume and pressure overload on the LA and LV (see Figure 5). During systole the LA receives retrograde blood flow from the LV, as well as antegrade blood flow from the pulmonary veins.

MR can develop acutely as when the valve is destroyed by IE, with myocardial ischemia that affects the wall of the LV and parts of the MV apparatus (e.g., papillary muscle/chordea tendinea rupture/dysfunction), or trauma (Bonow et al., 2006; Crawley, 1990; Curtin & Griffin, 2010; Klabunde, 2011; Turi, 2004; Vahanian et al., 2007).

Acute MR causes a sudden volume overload on both the LA and LV as it provides a low impedance pathway for retrograde blood to flow into the low pressure LA during LV systole. This will increase LV preload but forward SV and CO will be decreased. To compensate the LV will dilate and become hyperdynamic. Patients with acute MR develop pulmonary edema from the quick onset of high LAP and pulmonary venous pressure and this condition necessitates urgent cardiac surgery.

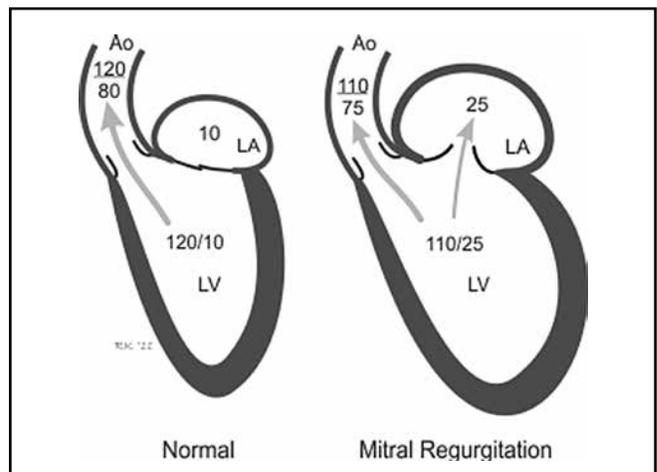


Figure 5: Pathophysiology of Mitral Regurgitation
 Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD005.htm>
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MR can also develop gradually and be chronic from conditions such as RHD, annular stretch (e.g., left ventricular dilatation/cardiomyopathy from conditions such as untreated hypertension, alcohol abuse, or certain drugs), coronary artery disease, or myxomatous degeneration (common cause for MV prolapse). The etiology of myxomatous degeneration is unknown but thought to result from connective tissue matrix degeneration and loss of collagen fibres in the MV leaflets. Patients with chronic MR can be asymptomatic for many years as the LA and LV compensate and remodel their size and become compliant (Bonow et al., 2006; Crawley, 1990; Curtin & Griffin, 2010; Klabunde, 2011; Reimer-Kent, 2007; Turi, 2004; Vahanian et al., 2007).

The effects of MR on the cardiac cycle are depicted in Figure 6. Due to the large volume overload (retrograde LV blood flow and antegrade pulmonary venous blood flow) there is a large increase in LAP throughout the cardiac cycle. The high LAP produces a tall “v” wave (e.g., 70 to 80 mmHg) at the end of LV systole. With MR there is no true isovolumetric contraction or isovolumetric relaxation because as soon as LV systolic contraction begins, even before the AV opens, blood from the LV begins to fall back into the LA. Once the AV does open retrograde blood flow continues into the LA and antegrade blood starts to flow into the LV. This will increase LAP and also increase LVEDP and LVEDV (i.e., preload).

The Frank-Starling mechanism will be activated and initially SV will increase; however, the net amount of blood ejected into the AO will decrease because some of this volume is also flowing retrograde into the LA. Eventually the LV will dilate, afterload will increase and cardiac decompensation with right and left heart failure will ensue. Increased LA size increases the likelihood of developing atrial fibrillation and the potential for thromboembolic events (Bonow et al., 2006; Crawley, 1990; Curtin & Griffin, 2010; Klabunde, 2011; Turi, 2004; Vahanian et al., 2007).

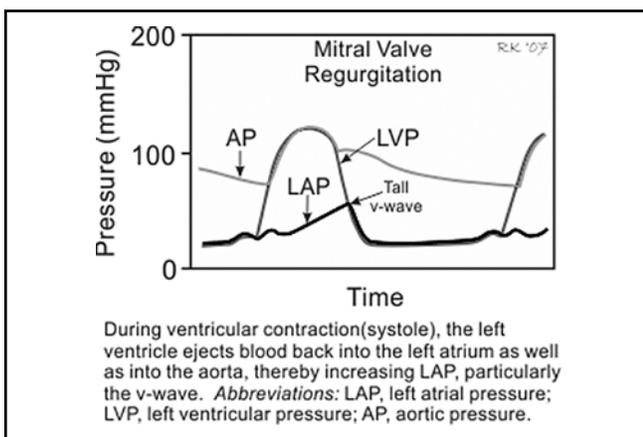


Figure 6: Mitral Regurgitation in Relation to the Cardiac Cycle

Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD005.htm>

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Heart Sounds Associated with Mitral Regurgitation

There are a variety of heart sounds with MR. The retrograde blood flow through an incompetent MV into the LA during systole causes a blowing holosystolic murmur that sounds like “Shshshshshshshshs” obliterating S_1 and S_2 (see Figure 7). The murmur is heard best at the cardiac apex (5th or 6th left intercostal space) with radiation towards the left anterior axillary line and left axilla. Listen with the diaphragm of the stethoscope with the patient supine and hear how the murmur intensifies when the patient moves into the left side lying position.

Due to the volume of retrograde blood flowing across the MV there may be a rumbling, early diastolic flow murmur. A third heart sound (S_3) (left atrial blood entering an already full LV in early diastole) or fourth heart sound (S_4) (left atrial blood entering a stiff, non-compliant LV during atrial systole during late diastole) may also be heard (Alpert, 1990; Bonow et al., 2006; Crawley, 1990; Curtin & Griffin, 2010; University of California San Diego, 2008; University of Washington, n.d.).

Pathophysiology of Mitral Stenosis

In MS the MV creates an obstruction that prevents blood flow from the LA into the LV during diastole. This causes hemodynamic pressure overload on the LA and pulmonary venous system (see Figure 8). The obstruction is most commonly caused by RHD with a latency period of approximately two decades before the onset of symptoms. RHD causes the MV orifice to become smaller and funnel-shaped as the MV thickens and calcifies, the leaflets scar and fuse at the commissures, and the chordae tendineae contract. Other less common causes include MV annular calcification, IE, systemic lupus erythematosus, rheumatoid arthritis, and intra-cardiac tumors [i.e., carcinoid or benign (e.g., atrial myxomas)] (Bonow et al., 2006; Crawley, 1990; Curtin & Griffin, 2010; Klabunde, 2011; Reimer-Kent, 2007; Turi, 2004; Vahanian et al., 2007).

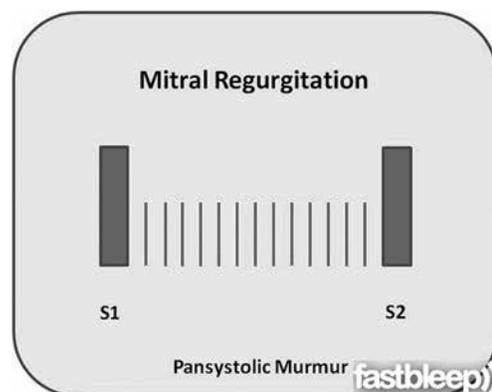


Figure 7: Mitral Regurgitation Murmur

<http://www.fastbleep.com/medical-notes/heart-lungs-blood/1/23/151>

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Normally at the end of diastole LAP equals left ventricular pressure. With the high resistance across the MV, pressure in the LA is higher than the pressure in the LV creating a diastolic pressure gradient across the MV (see Figure 9).

LAP, which is normally between 8 and 10 mmHg, is significantly elevated with MS (e.g., 25 to 30 mmHg) leading to LA enlargement. This in-turn will predispose the patient to atrial fibrillation and potential for systemic thromboemboli. Patients are often intolerant of atrial fibrillation as CO decreases from the loss of atrial contraction (kick) and a further decrease in diastolic filling of the LV.

High LAP is reflected back on the pulmonary venous system and right ventricle (RV). The RV hypertrophies because it is forced to generate pressures high enough to drive blood across an obstructed MV in an attempt to fill the LV and maintain CO. In time, pulmonary congestion/edema,

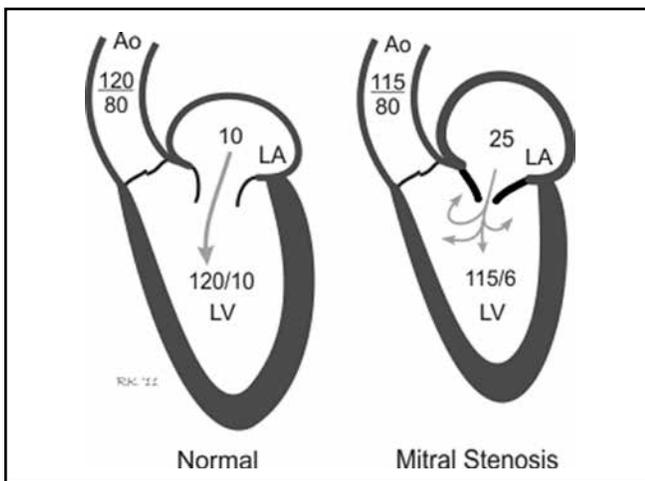


Figure 8: Pathophysiology of Mitral Stenosis
 Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD004.htm>
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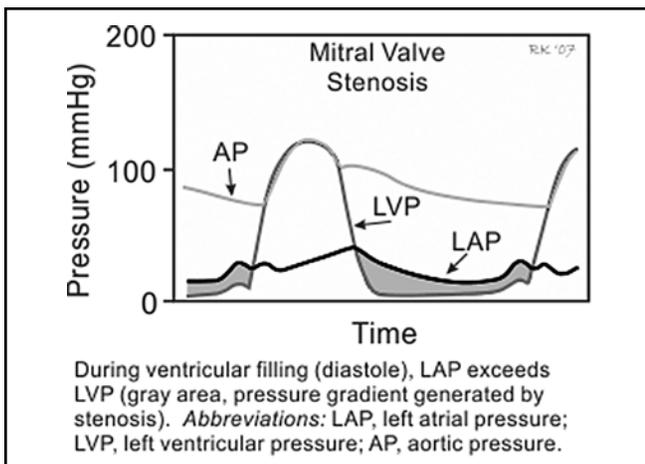


Figure 9: Mitral Stenosis in Relation to the Cardiac Cycle
 Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD004.htm>
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pulmonary hypertension, and right-sided heart failure will develop. The valve orifice is halved in size (e.g., less than 2.5 cm²) before symptoms develop.

In MS the LV is not subjected to undue pressure and remains small and thin-walled. Impaired filling of the LV will decrease preload. In an attempt to compensate for this reduced SV the Frank-Starling mechanism will be activated. However, as the disease progresses, the heart will decompensate and CO and aortic pressure will decrease (Bonow et al., 2006; Curtin & Griffin, 2010; Crawley, 1990; Klabunde, 2011; Turi, 2004; Vahanian et al., 2007).

Heart Sounds Associated with Mitral Stenosis

The obstruction to diastolic blood flow from the LA to the LV in MS creates a unique array of heart sounds: an opening snap, a loud S₁, a low-pitched, mid-diastolic, rumbling murmur that sounds like whispering the letter “r” out loud, and a loud pulmonic component (P₂) of S₂ (see Figure 10). These sounds are best heard at the apex with the bell of the stethoscope and the patient in the left side-lying position.

The opening snap is caused by the flow of blood across the obstructed MV leaflets, which is followed by a loud S₁ from the stiff MV leaflets closing, which is followed by a mid-diastolic murmur. The duration of the murmur is proportional to the severity of the MS and with critical MS the diastolic murmur becomes diminished. With pulmonary hypertension an accentuated P₂ would follow the murmur (Bonow et al., 2006; Choudhry & Etchells, 1999; Crawley, 1990; Curtin & Griffin, 2010; Klabunde, 2011; University of California San Diego, 2008; University of Washington, n.d.).

Pathophysiology of Aortic Stenosis

In AS the AV creates an obstruction that prevents blood flow from the LV into the AO during systole. This causes hemodynamic pressure overload on the LV (see Figure 11). The obstruction develops when the AV calcifies (i.e.,

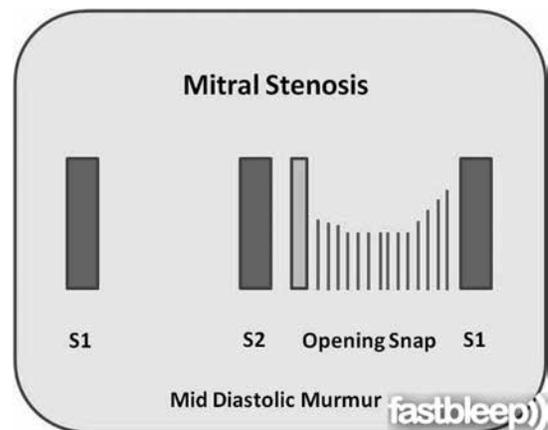


Figure 10: Mitral Stenosis Murmur
<http://www.fastbleep.com/medical-notes/heart-lungs-blood/1/23/151>
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degenerative or senile AS) or is congenitally deformed with only two cusps (i.e., bicuspid). Less common causes include RHD, radiation effects on the heart, and congenital aortic stenosis. The latter occurs when the AV commissures fail to fully develop in utero and the child is born with a unicuspid or bicuspid AV. The fusion of stiff, fibrotic cusps and commissures cause the AV orifice to take on a fish mouth-like appearance during systole (Bonow et al., 2006; Carabello, 2002; Cary & Pearce, 2013; Novaro, 2010; Vahanian et al., 2007).

Normally during systole, the pressure in the LV and AO are equal. With the high resistance across the AV, pressure in the LV is significantly higher than pressure in the AO creating a systolic pressure gradient across the AV (e.g., 50 mmHg or higher) (see Figure 12). Corresponding concentric hypertrophy of the LV develops to compensate for this pressure in order to maintain forward blood flow and CO. High peak left ventricular systolic pressure leads to an increase in afterload, a decrease in SV, and a resulting increase in left ventricular end-systolic volume (LVESV). This residual LVESV will be augmented by venous volume received during diastole, which will increase LVEDV (i.e., preload). The Frank-Starling mechanism is activated in an attempt to increase the driving force across the stenotic AV. As the LV becomes less compliant there is less capacity for LV diastolic pressure. This vicious cycle further impedes antegrade blood flow from the LA into the LV (Bonow et al., 2006; Carabello, 2002; Cary & Pearce, 2013; Novaro, 2010; Vahanian et al., 2007).

To protect the pulmonary venous system from the high systolic pressures of AS and to maintain forward SV requires an adequately functioning MV. Functional MR may develop as LAP is easier for the LV to overcome than AO pressure. This further compromises the delicate balance that comes with the low, fixed CO associated with AS. Efforts at manipulating any components of CO (i.e., heart rate or SV [preload,

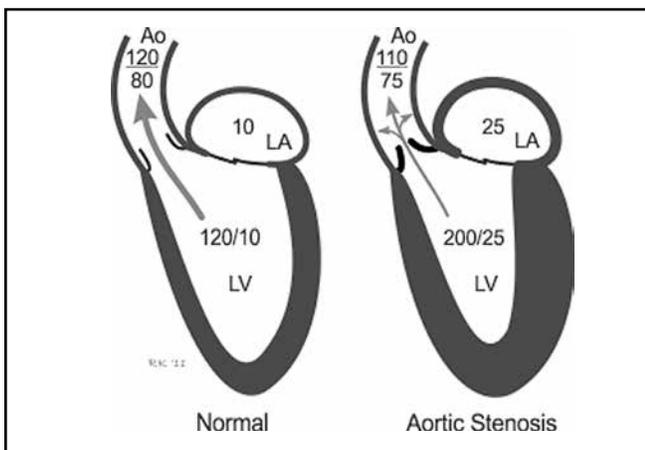


Figure 11: Pathophysiology of Aortic Stenosis
Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD004.htm>
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contractility, and afterload]) with medical management once AS is severe are of little help and may possibly be detrimental.

When the disease is advanced, the classical triad of severe AS symptoms appear (i.e., chest pain, syncope, dyspnea). The angina-like chest pain is not necessarily related to coronary artery disease, but rather to an imbalance in the oxygen supply/demand for an extremely hypertrophied LV and the poor delivery of blood to the endocardium. Syncope is caused by decreased cerebral blood flow and dyspnea comes from left ventricular failure. Severe AS is life-limiting and with the onset of symptoms life expectancy is less than two to three years (Bonow et al., 2006; Carabello, 2002; Cary & Pearce, 2013; Novaro, 2010; Vahanian et al., 2007).

Heart Sounds Associated with Aortic Stenosis

The obstruction to systolic blood flow from the LV to the AO in AS creates an array of heart sounds including a systolic ejection click, systolic murmur, paradoxical splitting of S_2 , and possibly an S_4 .

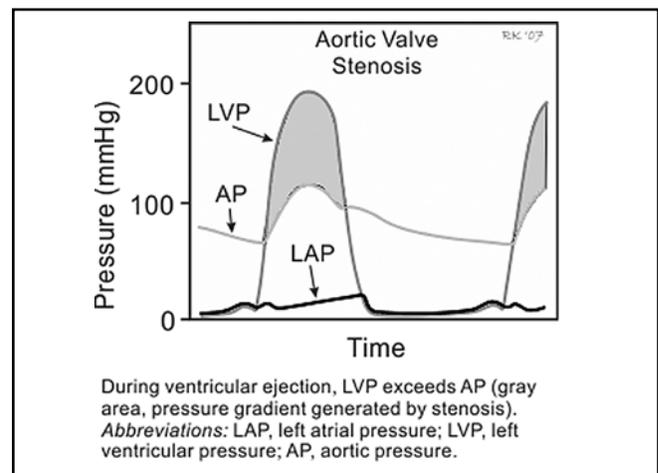


Figure 12: Aortic Stenosis in Relation to the Cardiac Cycle
Klabunde, R.E. (2011). <http://cvphysiology.com/Heart%20Disease/HD004.htm>
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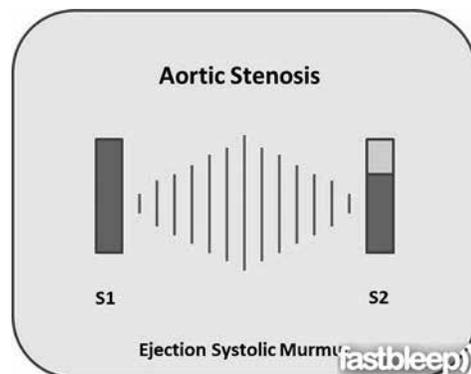


Figure 13: Aortic Stenosis Murmur
<http://www.fastbleep.com/medical-notes/heart-lungs-blood/1/23/151>
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The ejection click follows a normal S_1 and is due to the sound generated when the cusps of a stiff, non-compliant, yet mobile AV open. The crescendo-decrescendo (i.e., diamond) shaped systolic murmur is low to medium-pitched and harsh, rasping, grunting, rough, whooshing, or humming in quality. This murmur is best heard at the base of the heart in the AV area (second intercostal space right sternal border) and it radiates to the carotid arteries.

The closing of a poorly mobile AV will affect the aortic component (A_2) of S_2 . If A_2 is delayed because it was forced to close after P_2 , a paradoxical split of S_2 will be heard. An S_4 may also be heard during atrial contraction (kick) when more LA blood enters an already full, stiff and noncompliant LV in late diastole (Alpert, 1990; Bonow et al., 2006; Carabello, 2002; Cary & Pearce, 2013; Novaro, 2010; University of California San Diego, 2008; University of Washington, n.d.).

Conclusion

This concludes Part 2 with an overview of anatomy and pathophysiology of valvular heart disease and a description of the heart sounds associated with stenotic and regurgitant AV and MV disease. Heart sounds are just one part of your assessment, which must be correlated with the patient's clinical condition.

Remember it takes much practice to become proficient at this important nursing skill, so listen and learn by visiting the numerous websites that offer recordings of the sounds covered in this article like <http://medicine.osu.edu/exam/>, <http://www.med.ucla.edu/wilkes/intro.html>, <http://depts.washington.edu/physdx/heart/demo.html>, or <http://www.cardiosource.com/heartsounds/index.asp>. ♥

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