

# Aortic Valve Stenosis

## Definition

Aortic stenosis is a disease of the aortic valve in the heart. Aortic stenosis is a hardening or narrowing of the aortic valve causing restrictive opening, valvular incompetency, and pressure overload of the left ventricle of the heart (Cary & Pearce, 2013; Manning, 2013; Novaro, n.d.).

## Anatomy

There are four chambers of the heart and four valves that separate each of the chambers (Cary & Pearce, 2013). The purpose of each heart valve is to direct blood flow through the heart properly. When the valve opens it allows blood to pass to the next chamber and by closing, the valve prevents blood from flowing backwards (Novaro, n.d.). Each valve has a certain number of leaflets that open and close as blood passes through them (Cary & Pearce, 2013). The aortic valve has three, tightly-fitting, leaflets that are crescent shaped and fit together tightly when blood is not moving through them. The aortic valve is called a tricuspid, semi-lunar valve because of the three crescent-shaped leaflets. The aortic valve is the second semilunar valve in the heart with the pulmonic valve being the first. The aortic valve sits between the left ventricle and the aorta. It is the last valve that blood must pass through before exiting the heart. The aortic valve has three layers: the ventricularis, the fibrosa, and the spongiosa (Freeman & Otto, 2005). The ventricularis layer is composed of elastin-rich fibers aligned in a circular manner on the ventricular side of the valve. The fibrosa is composed of fibroblasts and collagen fibers also aligned in a circular manner on the aortic side of the valve. The spongiosa is a layer of loose connective tissue composed of fibroblasts, mesenchymal cells, and a mucopolysaccharide-rich matrix at the base of the leaflets of the valve between the fibrosa and ventricularis. These layers work together to provide the strength and flexibility of the aortic valve in order to withstand years of repetitive movement (Freeman & Otto, 2005).

## Pathophysiology

Calcification of the aortic valve is the most common cause of aortic stenosis in adults (Cary & Pearce, 2013). Calcification refers to the deposition and accumulation of calcium or lipids from the blood into the valvular tissue causing hardening and narrowing. Calcium and lipids are readily found in blood (Mayo Clinic, 2012). As blood rushes over the aortic valve surfaces, lipids and calcium are oxidized and taken up by macrophages into the endothelial layer of the valve. T lymphocytes and macrophages enter the endothelial cells of the valve and release cytokines. The cytokines act on the valvular fibroblasts and begin cellular proliferation and extracellular matrix remodeling. The fibroblasts differentiate into myofibroblasts and myofibroblasts then change into osteoblasts that are capable of promoting calcium nodule and bone formation (Freeman & Otto, 2005). The result of this cellular process is a stenotic aortic valve incapable of functioning properly. The aortic valve helps control aortic pressure. Aortic pressure is determined by the amount of blood that passes from the left ventricle to the aorta through the aortic valve (Katz, 2011). During diastolic filling, the pressure in the left ventricle is lower than the pressure in the aorta causing the healthy aortic valve leaflets to remain tightly closed. As the blood volume and pressure in the left ventricle increases and reaches its full capacity, it contracts and its pressure becomes greater than that of the aorta forcing the aortic valve to open and allow blood to pass into the aorta (Katz, 2011). When the aortic valve is stenotic and incompetent, blood flow is not regulated properly. Stenosis of the aortic valve does not allow the aortic valve leaflets to close tightly during left ventricular filling. This causes blood from the higher pressured aorta to re-enter the left ventricle during diastole. Blood is unable to be ejected properly from the left ventricle to the aorta because of more closely related pressures between the left ventricle and the aorta (Cary & Pearce, 2013). Backward flowing blood causes an increase in pressure in the left ventricle and can cause left ventricular hypertrophy and dysfunction (Katz, 2011). For further information regarding left ventricular hypertrophy see [www.mayoclinic.org](http://www.mayoclinic.org) or [my.clevelandclinic.org](http://my.clevelandclinic.org). Aortic stenosis may also be caused by rheumatic disease. Rheumatic fever can cause severe inflammation and subsequent scarring of the aortic valve (Mayo Clinic, 2012). Scar tissue can cause hardening and narrow of the aortic valve but it also causes a rough surface that attracts calcium and lipid deposits. Rheumatic fever is rare in developed countries but some older adults may have had the disease during their youth (Mayo Clinic, 2012).

## Genetics

Aortic stenosis can be congenital with a person being born with a bicuspid valve instead of a tricuspid valve thus predisposing them to incompetency of the aortic valve (Hull, 2012). Approximately 1-2% of the population is born with a bicuspid valve and of these about one-half will develop aortic stenosis (Novaro, n.d.). Bicuspid aortic stenosis affects more men than women (Novaro, n.d.).

## Epidemiology

Aortic valve sclerosis and subsequent stenosis is the most common valvular heart disease acquired in developed countries. Approximately 25% of people age 65 to 74 years and 48% of people over the age of 84 years present with aortic stenosis (Freeman & Otto, 2005). Aortic stenosis is associated with higher mortality and morbidity rates

than any other valvular disease (Cary & Pearce, 2013). Factors that increase risk of aortic stenosis are increased age, male gender, smoking, hypertension, and hyperlipidemia (Freeman & Otto, 2005). A diagnosis of aortic stenosis increases risk of myocardial infarction by 40% and cardiovascular death by 50% in persons with no preexisting cardiac conditions (Freeman & Otto, 2005).

## Disease described

Aortic stenosis refers to the obstruction of blood flow through the aortic valve of the heart (Novaro, n. d.). There are three main causes of aortic stenosis: calcification of the valve, rheumatic valve disease, and bicuspid aortic valve leading to stenosis. Aortic stenosis is defined hemodynamically by a transvalvular pressure gradient of at least 10 mm Hg (Novaro, n. d.). Aortic stenosis severity is graded as mild, moderate, severe, or critical. A valve area of 1.5-2.0 centimeters squared, aortic velocity of 2.5-3.0 m/sec, and a mean pressure gradient of less than 25 mm Hg define mild aortic stenosis. Moderate aortic stenosis is a valve area of 1.0-1.5 centimeters squared, 3.0-4.0 m/sec aortic velocity, and a mean pressure gradient of 25-40 mm Hg. Severe aortic stenosis is seen when valve area reaches 0.6-1.0 centimeters squared, aortic velocity is greater than 4.0 m/sec, and the mean pressure gradient is greater than 40 mm Hg. Critical aortic stenosis is defined as a valve area less than 0.6 centimeters squared (Cary & Pearce, 2013; Freeman & Otto, 2005; Novaro, n. d.).

## Signs and Symptoms

Aortic stenosis can often be symptomless or signs and symptoms can develop very gradually making the symptoms less noticeable (American Heart Association [AHA], 2013). Commonly, the first sign that a patient has or is developing aortic valve disease is the presence of a systolic heart murmur heard with a stethoscope during auscultation of the heart. The harsh-sounding systolic murmur is best heard in the second right intercostal space at the right upper sternal border at the base of the heart and can be transmitted to the clavicles and carotid arteries (Hull, 2012; Manning, 2013; Novaro, n. d.). Other signs include delayed carotid upstroke and sustained point of maximal impulse due to left ventricle enlargement (Manning, 2013). Symptoms most commonly associated with aortic stenosis include shortness of breath at rest and with exertion, decreased exercise tolerance, syncope or dizziness, chest pain or angina, heart palpitations, and lower extremity edema or swelling (AHA, 2013).

## Diagnosis

The first step in diagnosing aortic stenosis is auscultation of heart sounds with a stethoscope (AHA, 2013). A murmur heard during auscultation requires further evaluation. Auscultation should be executed using all five heart auscultation points. Auscultation should also be done with the patient in different positions and while holding breath and while performing the Valsalva maneuver (AHA, 2013). An echocardiogram may be done and may show changes of left ventricular hypertrophy and a chest x-ray may show an enlarged cardiac silhouette or heavy calcification of the valve or the ascending aorta but neither is a reliable tool for sole diagnosis aortic stenosis (Novaro, n. d.). The main tool for diagnosis is a non-invasive transthoracic two-dimensional doppler echocardiogram (AHA, 2013; Cary & Pearce, 2013; Novaro, n. d.). An invasive transesophageal echocardiogram may also be performed. Echocardiography allows for assessment of the valvular structures, heart chamber size and functionality, blood flow through the heart and valves, and estimation of pressure gradients and valve area (Novaro, n. d.). With a quality echocardiogram, cardiac catheterization is frequently not needed to diagnose aortic stenosis (Novaro, n. d.). If cardiac catheterization is needed, the procedure can accurately measure pressure gradients across the valve and cardiac output.

## Treatment

Currently, there is no medical curative treatment for aortic stenosis. Secondary preventative treatment such as modifying exercise and diet, lowering lipids with statin medications, and lowering blood pressure with beta-blockers can be helpful but not curative (Novaro, n.d.). If aortic stenosis is asymptomatic or mild, regular check-ups with inquiry of change in exercise tolerance or development of any symptoms is adequate. Routine transthoracic echocardiograms are also suggested with frequency based on the severity of aortic stenosis (Novaro, n. d.). Many patients with asymptomatic or mild aortic stenosis are treated medically with the secondary preventative measures noted above. Treatment for symptomatic patients with moderate to critical aortic stenosis is undergoing open-heart surgery for an aortic valve replacement. Replacement of the valve is not curative but rather palliative as the prosthetic valve can encounter problems causing incompetency as well (Hull, 2012; Novaro, n. d.).

## Links to More Information on Aortic Stenosis

American Heart Association- <http://www.heart.org/>

Cleveland Clinic- <http://www.clevelandclinicmeded.com/>

Mayo Clinic- <http://www.mayoclinic.org/>

## Related Current Articles

Please see references

## References

- American Heart Association. (2014). Heart valve problems and disease. Retrieved from [http://www.heart.org/HEARTORG/Conditions/More/HeartValveProblemsandDisease/About-Heart-Valves\\_UCM\\_450699\\_Article.jsp](http://www.heart.org/HEARTORG/Conditions/More/HeartValveProblemsandDisease/About-Heart-Valves_UCM_450699_Article.jsp)
- Cary, T. & Pearce, J. (2013). Aortic stenosis: Pathophysiology, diagnosis, and medical management of nonsurgical patients. *Critical Care Nurse*, 33(2), 58-71.
- Freeman, R. V. & Otto, C. M. (2005). Spectrum of calcific aortic valve disease: Pathogenesis, disease progression, and treatment strategies. *Circulation*, 111, 3316-3326. doi: 10.1161/CIRCULATIONAHA.104.
- Hull, C. L. (2012). Treating calcific aortic stenosis: An evolving science. *Medsurg Nursing*, 21(2), 82-88.
- Katz, A. M. (2011). *Physiology of the heart*. Philadelphia, PA: Lippincott Williams & Wilkins.
- Manning, W. J. (2013). Asymptomatic aortic stenosis in the elderly: A clinical review. *The Journal of the American Medical Association*, 310(14), 1490-1497. doi: 10.1001/jama.2013.279194
- Mayo Clinic. (2012). Aortic valve stenosis. Retrieved from <http://www.mayoclinic.org/diseases-conditions/aortic-stenosis/basics/definition/con-20026329>
- Novaro, G. M. (N. D.). Aortic valve disease. Retrieved from <http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/aortic-valve-disease>