Near Syncope and Fatigue; Warning Signs for Aortic Stenosis

Kendra L. Newland

Otterbein College

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Chief Complaint: "I have been too weak to do my work."

History of Present Illness: Eli Miller is a 65-year-old, married, white man. He has been experiencing constant weakness and paroxysmal lightheadedness for the past three to four days. Onset was sudden. He reports five or six episodes of lightheadedness in the past four days. He denies any previous episodes. His lightheadedness worsens with exertion; however it also occurs without any precipitating factors. He describes this lightheadedness as a "feeling that everything is turning black" and states that he must either sit or lay down for this to subside, which it does after a "few minutes". He denies losing consciousness or sensations of the room spinning. He denies headaches or recent trauma. His weakness he describes as a general lack of energy. He states that he "feels tired all of the time". He denies specific muscle pain or muscle weakness. He is having trouble carrying out his normal daily activities and he is losing productivity at work.

Current medications: Mr. Miller takes the following herbals and other supplements for heart health: Lilly of the valley 1 tsp. daily, alfalfa 2 tabs three times daily, CoQ10 twice daily, activated charcoal four times daily.

Allergies: NKDA

Past Medical History: Positive for hypertension. He is unsure of what, if any, childhood illnesses he had. He does not recall being sick as a child. He has not received any immunizations.

Surgical History: No past surgeries.

Social History: Mr. Miller is an Amish gentleman who is married with 10 children. He does not smoke, drink alcohol or use illicit drugs. He drinks one cup of coffee per day. He eats three meals per day. He completed the 8th grade. He works in a harness shop. He gets regular exercise with farm chores daily. He sleeps eight hours per night.

Family History: Father died at age 89 in an accident; no known medical problems. Mother died in an accident at age 48; no known medical problems. He had a brother who died at the age of 41 of probable heart disease. He has a sister, in her 40's, alive with valve disease. He has 10 children, all living with no known health problems, ages ranging from 42 years to 10 years old. His grandparents died while he was young and he does not recall their causes of death.

Decision Point #1: Focused history, physical exam, laboratory tests

Focused History-

The possible etiologies of Mr. Miller's vague symptoms are extensive. Based on the history of present illness, it can be determined that Mr. Miller is experiencing near syncope. Near syncope can be defined as, "the sense of imminent loss of consciousness without frank syncope" (Magaziner & Walker, 2007, p. 1540). Further questioning aims to identify potential origins of Mr. Miller's near syncope and fatigue. This is best achieved by attaining a focused history guided by plausible causes.

Near syncope and fatigue could arise from a neurologic pathology. A detailed history that would give suspicion to a neurologic etiology is performed, supplementing the data that was gathered in the history of present illness. Mr. Miller denies experiencing the feeling of losing his balance. He denies any hearing or vision changes or trouble with speech or memory. Mr. Miller denies numbness or tingling in his extremities or headaches. He denies loss of consciousness, disorientation, tremors, or involuntary movements.

Infection and malignancy can present with the symptoms that Mr. Miller presents with. He denies having a fever or chills. Mr. Miller denies having a cough, choryza, nasal congestion, ear pain, sore throat. He denies nausea, vomiting, diarrhea or bloody stools. He denies burning or

pain with urination or flank pain. Mr. Miller denies any changes in his appetite or recent weight loss.

Metabolic disorders such as hypothyroidism, hyperthyroidism, hypoglycemia and hyperglycemia can cause a patient to have fatigue and near syncope. Mr. Miller denies being increased thirst or hunger. He denies frequent urination. Mr. Miller denies dry skin or constipation. He denies intolerance to heat or cold.

Cardiac etiologies are important to consider when obtaining a history from a patient with near syncope and fatigue. Mr. Miller denies heart palpitations, chest pain, shortness of breath or edema. He does not recall ever having been told that he has a heart murmur. He does not know if anyone in his family was ever diagnosed with cardiomyopathy or an arrhythmia.

The practitioner must establish if the symptoms are psychogenic in nature. Mr. Miller denies feeling nervous or anxious. He denies a significant change in his mood or dysthymia. Mr. Miller has not had any significant changes in his life.

The detailed history does not give suspicion to any specific cause. Since these symptoms are vague and could indicate any number of disease processes, a thorough physical examination is performed.

Physical Examination-

*Vital Signs***:**

Height is 5’5”; **weight** is 150 lbs, **BMI** 24.5

BP: 160/80 (right arm, lying); 154/72 (standing) H**R** 72 (regular) **RR** 18 (regular) Temp 98.6 F

*General***:** Mr. Miller is a 65-year-old, normal weight man, well-groomed, and in good spirits. He has no obvious physical deformities. He has a steady gait, good posture, is able to get up and

down from exam table without difficulty. His speech is clear. He is able to hear normal conversational tone without difficulty. Body and breath without pungent odor.

Skin: Color pink. Skin warm and moist. No rash, petechiae, or ecchymoses. Hair with average texture, normal distribution. Turgor good.

HEENT: Head-The head is normocephalic/atraumatic. Face is symmetric with appropriate expression. *Eyes*- Symmetric, no erythema or exudate. Sclera white, conjunctiva pink. PERRLA. Red reflex noted. Visual acuity 20/20 on Snellen chart without correction. *Ears:* Acuity good to normal conversational tone at 3 feet. External ears symmetric without erythema, masses or edema. TMs with good cone of light. *Nose:* External nose symmetric, without erythema, edema. Nasal mucosa pink. No sinus tenderness. *Mouth and throat:* Oral mucosa pink, dentition good, pharynx without exudates. Gums pink. Tongue midline and pink. Soft palate and uvula mobile. Uvula midline. *Neck:* Trachea midline. Neck symmetric, supple, thyroid isthmus palpable, lobes not felt. No visible lumps or pulsations. No bruits. Full range of motion, strong muscle strength.

Lymph nodes: Tonsillar, submandibular, submental, anterior and posterior cervical, preauricular, posterior auricular, occipital, inguinal, axillary, and inguinal nodes without noted lymphadenopathy or tenderness.

Chest: Thorax is symmetric with good expansion. Respirations even and unlabored. Clear breath sounds bilaterally anteriorly and posteriorly.

Cardiovascular: Apical pulse with regular rate and rhythm. Distant S1, S2, no S3 or S4. Grade 3/6 pansystolic harsh murmur heard best at sternal notch. No rubs, gallops or clicks. No thrill palpable. Radial pulses palpable, regular and strong bilaterally. Pedal pulses palpable, weak and regular bilaterally.

Peripheral Vascular System: Extremities are warm and without edema. No varicosities or stasis changes.

Gastrointestinal: Abdomen is flat, soft, non-distended and non-tender. Active bowel sounds in all 4 quadrants. No masses or hepatosplenomegaly. No costovertebral angle tenderness. No inguinal lymphadenopathy or tenderness.

Musculoskeletal: No joint deformities. Good range of motion and strong muscle strength in hands, wrists, elbows, shoulders, spine, hips, knees, ankles.

Neurologic: Mental Status: Alert and cooperative. Thought coherent. Oriented to person, place and time. *Cranial Nerves:* CN II-XII intact. No nystagmus. *Motor:* Good muscle bulk and tone. Gait stable, fluid. Negative Dix-Hallpike maneuver. Negative Brudzinski's sign. Rapid alternating movements, finger-to-nose and heel-to-shin intact. Romberg – maintains balance with eyes closed. No pronator drift. Deep tendon reflexes 2+ bilaterally with plantar reflexes downgoing.

Preliminary Labs and Testing:

With syncope or near syncope, there is no one test identified as a gold standard for diagnosis. The practitioner must order diagnostic testing based on sound clinical judgment. From the findings of the physical exam, explicitly the murmur that was noted, an echocardiogram is warranted. Echocardiography is the preferred method to identify the cause and severity of heart murmurs (McCannon, 2004). An electrocardiogram is also indicated to identify a cardiac etiology of Mr. Miller's near syncope. It is important to note that Mr. Miller does not have medical insurance. This fact, along with cultural beliefs and practices, greatly influence the degree of testing that is performed initially. The Amish are cautious and conservative in their use of modern medical technology (Armer & Radina, 2006). Although it was highly recommended,

he did not wish to have an echocardiogram or electrocardiogram performed at this time. The community hospital where Mr. Miller lives offers health profile screenings for its residents for a very minimal fee. He agreed to have this lab work done which consists of a complete metabolic panel, a lipid panel, a serum iron level, and a complete blood count. While the laboratory testing will not provide any diagnostic information related to the heart murmur, it can effectively rule out other etiologies of Mr. Miller's symptoms. Mr. Miller's initial test results are found in Table 1.

Table 1

Test	**Result**		**Indication**	**Test**	**Result**		**Indication**
CMP	Sodium	141	BUN elevated (normal reference range 6-24 mg/dl) All other within normal limits	Lipid Panel	Cholesterol	168	LDL elevated (normal reference range 0-99 mg/dl) All other within normal limits
	Potassium	4.6			Triglycerides	91	
	Chloride	105			HDL	35	
	CO2	28			LDL **H**	115	
	BUN **H**	27			Calculated LDL/HDL	3.28	
	Creatinine	1.0			Cholesterol/HDL	4.80	
	Glucose	83					
	Calcium	9.1					
	AST (SGOT)	23					
	ALT (SPGT)	40					
	Total bilirubin	0.6					
	Total protein	6.5					
	Albumin	3.6					
	Alkaline Phosphatase	121					
	GGT	37					
CBC	WBC	10.5	Within normal limits	Serum Iron	Iron	63	Within normal limits
	RBC	5.49					
	HGB	14.9					
	HCT	45.9					
	MCH	27.1					
	MCHC	32.5					
	Platelet	309					
	MC	84					

Decision Point #2: Differential diagnoses

Symptoms of near syncope and fatigue are rather vague and not specific to any one health alteration; rather, the list of differential diagnoses for these complaints is vast and encompasses many body systems. The differential diagnoses can be categorized into five general causes: hypotension, cardiac disease, metabolic conditions, intracranial conditions, or psychiatric disorders (Magaziner & Walker, 2007). For this 65 year old man, the list of differential diagnoses that must be considered based on the history of present illness is found in Table 2.

Table 2

Hypotension	• Medication effects • Vasovagal or neurocardiogenic syncope • Decreased blood volume (hemorrhage, dehydration) • Nutritional deficiencies (hypoalbuminemia) • Orthostasis of aging • Cervical osteoarthritis (causes compromise of cerebral blood flow) • Carotid sinus hypersensitivity
Cardiac Disease	• Arrhythmia • Aortic or mitral valve stenosis • Acute myocardial infarction • Mitral valve prolapse • Angina • Cardiomyopathy • Coronary artery disease
Metabolic Conditions	• Anemia • Infective processes (including meningitis) • Cancer • Hypoglycemia or Hyperglycemia • Hypothyroidism or Hyperthyroidism • Electrolyte imbalances (hypokalemia, hyponatremia, hypocalcemia)
Intracranial Conditions	• Transient ischemic attack • Migraine • Seizure disorder
Psychiatric Disorders	• Anxiety • Depression

A thorough history and review of symptoms is crucial to narrowing the list of differentials and assist the practitioner in focusing the physical exam. The physical exam will help guide the practitioner to further limit the probable diagnoses and to determine what testing is indicated. It is vital that the practitioner distinguishes between benign and life-threatening causes of syncope.

In the case of Mr. Miller, the detailed history effectively ruled out a number of possible diagnoses. Mr. Miller denied any symptoms of an acute infectious process. He denied any symptoms of anxiety or depression. He denied a headache, effectively ruling out migraines. A diagnosis of seizure is five times more likely if the patient is disoriented after the event (Magaziner & Walker, 2007). Mr. Miller denied feeling disoriented after episodes of lightheadedness, in effect ruling out seizures. He denies any chest pain therefore ruling out angina. He denies symptoms of palpitations or heat intolerance which guides the practitioner away from hyperthyroidism. He also denies symptoms of cold intolerance and constipation which direct the practitioner away from hypothyroidism. Similarly, he denies symptoms of hyper- and hypoglycemia (hunger, palpitations). The laboratory testing excluded anemias (normal hemoglobin, hematocrit, red blood cell count), electrolyte imbalances (normal sodium, potassium, calcium carbon dioxide levels), dehydration (normal creatinine, BUN, electrolytes), and nutritional deficiencies (normal albumin, electrolytes). The blood work did reveal a slight elevation in his LDL level. The physical exam ruled out orthostatic hypotension, which is defined as a decrease in systolic blood pressure of 20 mmHg or more when patient changes from a supine to standing position. Mr. Miller's orthostatic blood pressure measurements did not indicate orthostatic hypotension. Meningitis was ruled out with a negative Brudzinski's sign.

The physical exam did reveal a grade 3/6 harsh pansystolic murmur which indicates a possible cardiac etiology for Mr. Miller's symptoms. Organic heart disease may be a life-threatening cause of near syncope (Magaziner & Walker, 2007). Other possible diagnoses include neurocardiogenic syncope, medication effects, transient ischemic attack, corornary artery disease, arrhythmia, and aortic stenosis (among other diseases that inhibit cardiac outflow).

Medication effects

Alternative medicine, including the use of herbal supplementation, is routinely practiced in the Amish culture (Armer & Radina, 2006). Many patients will not include herbals and other supplements in their list of medications, so the practitioner must explicitly question the patient about the use of alternative therapies. People consider herbs to be safe because they are "natural". Unlike prescription drugs, the Food and Drug Administration does not regulate herbs and supplements, therefore potency and purity is not guaranteed. Many herbs have not been thoroughly tested and safety and effectiveness are not proven. Furthermore, herbal supplements do occasionally have serious side effects and may interact with prescription drugs. Mr. Miller takes activated charcoal, alfalfa, Co-Q 10, and Lily of the Valley on a daily basis. Activated charcoal is said to help lower cholesterol. Activated charcoal absorbs many materials, including prescription drugs. Alfalfa, thought to lower cholesterol and glucose, may cause hypoglycemia which can lead to syncope (U.S. National Library of Medicine, 2008). Coenzyme Q 10 is produced by the body and is necessary for the basic functioning of cells. Levels are thought to be low in some chronic conditions such as heart conditions, cancer, diabetes and Parkinson's disease. Coenzyme Q10 may cause hypoglycemia and dizziness, and it may lower blood pressure, among other side effects (U.S. National Library of Medicine, 2008). Lily of the Valley is another herb thought to help the heart. Some concerning side effects reported are heart failure,

coma and death (Thomson Reuters, n.d.). The side effects of herbs as a causative factor of Mr. Miller's symptoms cannot be ruled out without stopping the supplements to determine if the symptoms cease. In light of the cardiac murmur that was identified with the physical exam, medication effect is not explored as a causative factor at this time.

Neurocardiogenic syncope

Neurocardiogenic syncope, or vasovagal syncope, is the most common cause of syncope in adults and children, accounting for 50-66% of inexplicable syncope (Chen-Scarabelli & Scarabelli, 2004). A benign condition, it is caused by an abnormal autonomic response to stimuli and results in self-limited bradycardia and hypotension. This bradycardia and systemic hypotension leads to syncope; consciousness is regained promptly after the patient lies down. Often, there is a prodromal period in which the patient may experience lightheadedness, pallor, flushing, nausea, palpitations and throat tightness (Magaziner & Walker, 2007). Syncope can be avoided if the patient lies down during this prodromal period. After a syncopal episode, the patient often complains of tiredness which resolves. In more than 50% of patients with neurocardiogenic syncope, the history and physical are non-diagnostic (Chen-Scarabelli & Scarabelli), therefore, this is often a diagnosis of exclusion. Tilt-table testing can be done to confirm the diagnosis.

Mr. Miller has near syncopal episodes that completely resolve with lying or sitting down. He describes general fatigue, not associated specifically with the near syncopal episodes. In the case of Mr. Miller, the discovery of the heart murmur during the physical exam leads the practitioner to consider a cardiac cause rather than the benign diagnosis of neurocardiogenic syncope. If further workup indicates that the near syncope that Mr. Miller is experiencing is not related to a cardiac etiology, this diagnosis may be reconsidered.

Transient ischemic attack (TIA)

Mr. Miller is at increased risk for cerebrovascular disease due to hypertension and hyperlipidemia. The identification of a heart murmur in his physical exam also adds to the suspicion of a TIA. It is known that 15% to 30% of all ischemic strokes are attributed to an embolus originating in the heart which is highly associated with cardiac arrhythmias, valvular disease, recent myocardial infarction and dilated cardiomyopathy (Llinas & Johnson, 2007). Hypertension and hyperlipidemia also place Mr. Miller at risk for decreased cerebral blood flow due to diseased carotid arteries. TIA is defined as a "transient episode of focal cerebral dysfunction, rapid in onset (from none to maximum symptoms in less than five minutes), that usually lasts from two to fifteen minutes but always resolves completely within 24 hours (Llinas & Johnson, 2007, p. 1573). Mr. Miller's near syncope completely resolved within a few minutes, as would be expected with a TIA. However, Henry and Johnston (2004) report that if syncope (or, in this case, near syncope) occurs with a TIA, more typical TIA symptoms generally accompany (such as slurred speech or one-sided weakness). Also, it is not likely that a transient episode with altered consciousness is vascular in nature (Llinas & Johnson, 2007). Taking this into account, TIA is not considered a likely diagnosis; however, it should not be ignored due to the potential mortality and morbidity. Additionally, the clinical evidence of heart disease, specifically the cardiac murmur, would direct the practitioner to obtain an echocardiogram (which would identify a cardiac source of emboli) and electrocardiogram (which would identify an arrhythmia).

Coronary Artery Disease, Arrhythmias, and Myocardial Infarction

Coronary artery disease (CAD) caused by atherosclerosis affects over 13,000,000 Americans (Chandra-Strobos & Hirsch, 2007). Mr. Miller is at increased risk for coronary artery

disease from hypertension and hyperlipidemia (based on recent laboratory findings) as well as a family history of heart trouble. Patients with CAD are at risk for having a myocardial infarction (MI). An acute MI may present without the classical symptoms, particularly in an older adult. In fact, the sole presenting manifestation of an MI may be syncope (or near syncope) (Kyrillos, Carissa, & Pineda, 2005). For Mr. Miller, coronary artery disease and myocardial infarction are possible diagnoses due to his risk factors. While he does not have the classic symptoms of an acute MI, the practitioner should not rule this out without first checking a 12-lead electrocardiogram. An electrocardiogram may be normal in CAD, but would be abnormal in a patient with a recent or past history of an MI.

Cardiac arrhythmias can be symptoms of disease or they can cause disease. They can be relatively benign or they can be deadly. Arrhythmias should always be considered with near-syncope, particularly in the older adult (Magaziner & Walker, 2007). Mr. Miller's symptom of lightheadedness might be caused by decreased cardiac output as a result of an arrhythmia. Mr. Miller has a history of hypertension and has a cardiac murmur, two underlying diseases that may be associated with arrhythmias (Gottlieb, Marine, & Calkins, 2007). Mr. Miller denies palpitations; however, the presence or absence of symptoms does not confirm or eradicate the possibility of an arrhythmia. Upon physical assessment, his heart rate was within normal limits with a regular rhythm. Arrhythmias may be paroxysmal in nature, so they may not be identified during an outpatient office visit. A single electrocardiogram tracing may not identify a paroxysmal arrhythmia. If arrhythmia is still suspected after a normal 12-lead electrocardiogram, a 24 hour ambulatory electrocardiogram is indicated. In Mr. Miller's case, arrhythmia is of high concern and cannot be ruled out at this time.

Aortic Stenosis

Aortic stenosis (AS) is an abnormal narrowing of the aortic valve. It is a significant cause of morbity and mortality among the elderly population (Mallavarapu, Mankad, & Nanda, 2007). Aortic stenosis has been documented in 2% to 4% of patients over 65 years of age (Lauck, Mackay, Galte, & Wilson, 2008). Its precursor, aortic sclerosis, is present in an estimated 25% of the population over 65 years of age (Keong Yeo & Low, 2007).In the older adult, the most common cause is calcification of the aortic valve. Evidence indicates that the same factors that influence the development of CAD, like smoking, hypercholesterolemia, hypertension and diabetes, also influence the progression of AS (Lauck et al., 2008). Historically, AS has been considered to be a degenerative disease. However, new evidence suggests that AS may be an active disease similar to CAD involving lipoprotein deposition, chronic inflammation and active calcification of the leaflets (Lauck et al., 2008). As calcification progresses, the valve leaflets become stiff and narrow, causing impaired valve opening. The outflow of blood from the left ventricle is obstructed; the pressure in the left ventricle increases, leading to concentric hypertrophy of the left ventricle. This hypertrophy enables the heart to compensate and maintain adequate cardiac output for a period of time during which the patient is clinically asymptomatic. However, this hypertrophy also decreases coronary blood flow and leads to diastolic and systolic left ventricular dysfunction. When the compensatory mechanisms fail, the patient starts exhibiting classic symptoms of AS: heart failure, angina or syncope. Initially, many patients experience decreased exercise tolerance and other vague signs and symptoms (Lauck et al., 2008).Once symptoms develop, the average survival is two to three years. It is important to note that the severity of AS is poorly associated with signs and symptoms. Patients with aortic stenosis generally have a grade 1-4/6 harsh, crescendo-decrescendo, systolic murmur heard best at the second right intercostal space or at the apex. The murmur may radiate to the carotid

arteries. The murmur often softens with standing and increases when the patient leans forward. S1 and S2 may be diminished. The patient with AS may complain of fatigue, dyspnea on exertion or angina. Mr. Miller's cardiac exam revealed distant S1, S2, grade 2/6 pansystolic harsh murmur heard best at the sternal notch. He complains of fatigue, but denies angina or dyspnea. He does have exertional near syncope, which is a classic symptom of AS. Elderly patients with severe aortic stenosis may present with subtle symptoms (Grimard & Larson, 2008). Exertional dyspnea is the most common initial complaint (Talano & Melek, 2007). In light of the harsh systolic murmur and clinical presentation, as well as the prevalence of the disease among the older population, aortic stenosis is a viable diagnosis. It is important to note that in the case of Mr. Miller rheumatic valve disease should be considered since he is unable to recall his history of childhood illnesses. Also, the Amish culture often relies on alternative therapies to treat disease, so he may have had an untreated streptococcal infection at some point.

Decision Point #3: Further work-up

At this point, further work-up includes an echocardiogram to visualize the heart valves and an electrocardiogram to identify any cardiac arrhythmias and rule out myocardial infarction. The patient called the office two weeks after his original office visit and informed the practitioner that he was experiencing dyspnea with exertion. He agreed to have an echocardiogram and electrocardiogram performed.

Decision Point #4: Management

The presumptive diagnosis prior to the echocardiogram and electrocardiogram is aortic stenosis based on the auscultation of a harsh systolic murmur and the presence of the classic symptom of exertional near-syncope. A cardiology referral is made. He is given a prescription for lisinopril 5 mg daily for his hypertension. It is estimated that 40% of patients with AS also

have hypertension (Grimard & Larson, 2008). Hypertension increases systemic vascular resistance and further creates an increase in left ventricular afterload. Angiotensin-converting enzyme inhibitors are well tolerated and will improve exercise tolerance in the symptomatic patient (Grimard & Larson). Mr. Miller is also prescribed lovastatin 20 mg daily for hyperlipidemia. There is conflicting evidence on the use of statins to slow the progression of AS. Larger studies are expected to conclude in 2009 (Grimard & Larson). Mr. Miller is encouraged to take an 81 mg aspirin daily due to his risk of cardiovascular disease. The use of herbal medications and their possible risks is discussed. He is instructed to seek immediate medical attention if his symptoms suddenly worsen. In 2008, the American Heart Association stated that antibiotic prophylaxis was no longer indicated for prevention of infective endocarditis in patients with aortic stenosis (Nishimura et al., 2008).

Watchful waiting is recommended in most asymptomatic patients, with survival rates comparable to those in patients without AS (Grimard & Larson, 2008). Doppler echocardiology should be performed every year for the asymptomatic patient with severe AS, every one to two years with moderate AS and every three to five years for mild AS (Grimard & Larson, 2008). Symptomatic patients with severe aortic stenosis have poor prognosis without valve replacement; 52% of these patients who did not receive surgery were dead at 5 years according to the National Institutes of Health (Aronow, 2007).The only definitive treatment for symptomatic moderate to severe AS in the elderly is aortic valve replacement (Aronow, 2007). In fact, the 10-year-survival rate after aortic valve replacement in patients over 65 years of age is almost the same as age- and sex-matched patients without AS (Grimard & Larson, 2008). The degree of severity corresponds to the peak gradient across the aortic valve and the aortic valve area (as measured by

echocardiography) and is classified as mild, moderate or severe (see Table 3 for more information). Mr. Miller is symptomatic with near-syncope and dyspnea.

Table 3 (Grimard & Larson, 2008, p.720)

Severity	Aortic valve area (cm2)	Aortic jet velocity (m per second)	Mean Gradient (mmHg)
Normal	3 to 4	< 2.5	--
Mild	1.5 to 2	2.5 to 2.9	<25
Moderate	1 to 1.5	3 to 4	25 to 40
Severe	<1	>4	>40

Decision Point #5: Final diagnosis

The electrocardiogram was abnormal, showing sinus rhythm at 91 beats per minute with a marked left ventricular hypertrophy with strain pattern. The echocardiogram showed severe calcification of the aortic valve with severe stenosis (valve area 1 cm2, peak gradient 68 mmHg), concentric left ventricular hypertrophy, and mild left ventricular systolic dysfunction (ejection fraction 50%). The electrocardiogram showed no evidence of a myocardial infarction, so this can be ruled out. Based on the results of the echocardiogram and electrocardiogram, the diagnosis of aortic stenosis is verified and left ventricular hypertrophy is diagnosed. When the echocardiogram shows severe aortic stenosis in a symptomatic patient, symptoms must be presumed to be caused by the AS, even if other potential causes are present (Grimard & Larson, 2008).

The patient did follow up with the cardiologist who recommended a heart catheterization and angiogram to assess the aortic valve and to examine the coronary arteries. The probability of a patient requiring an aortic valve replacement was discussed. The patient wanted to consider it

and has not followed up with the cardiologist. On his return visit to the family practitioner, he reported that he did not wish to have surgery. He stated that he has not filled the prescriptions given to him and he is continuing to use the herbal supplements on a daily basis. The practitioner discussed with Mr. Miller the seriousness of this condition and the poor prognosis without surgical intervention. The risk of congestive heart failure, heart attack or sudden cardiac death was discussed at length. He was instructed to follow a low salt diet so as not to develop fluid volume overload. He was, again, strongly encouraged to take the lisinopril, lovastatin, and baby aspirin. Mr. Miller understood all that was discussed and wished to go home and think about it.

Case Study Summary

When a patient presents with near syncope and fatigue, the practitioner must rely on a thorough history and expert physical assessment skills to guide clinical decision making. Whenever these symptoms are present with a cardiac murmur, a cardiac etiology must be considered. In the case of Mr. Miller, the finding of a harsh, crescendo-decrescendo systolic murmur indicated aortic stenosis therefore, an echocardiogram was ordered. Aortic stenosis is a common cause of morbidity and mortality among the older population. While valvular heart disease may be the cause these symptoms in a patient, it is not always the case, and they could arise from another source. For this reason, other diagnosis should remain on the differential list until proven otherwise. In Mr. Miller's case, neurocardiogenic syncope, arrhythmia, coronary artery disease, myocardial infarction, effects of herbal medications, and transient ischemic attack remained in the back of the practitioner's mind until further testing was performed. The echocardiogram showed severe aortic stenosis, which leads the practitioner to presume that the symptoms are related to the valvular disease versus another cause. In the case of Mr. Miller, the practitioner prescribes an ACE inhibitor for hypertension and a statin for hyperlipidemia. He is

encouraged to take a baby aspirin daily and to discontinue the use of herbal medications. The patient is referred to a cardiologist for further work-up and treatment. Cultural beliefs and practices play a role in the management of Mr. Miller, and the practitioner must be sensitive to this.

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