Pulsus Alternans
A Case Study

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Critical care nurses care for patients with various degrees of illness. Unfortunately, a critical illness may be masked by the signs and symptoms of a secondary abnormality, for example, severe abdominal pain masking heart failure. Good assessment skills and clinical knowledge are required to ensure that important findings are not overlooked. One physiological finding that may be overlooked is pulsus alternans.

Pulsus alternans is a cardiovascular phenomenon characterized by alternating strong and weak pulse pressures during a sinus rhythm. This alternation is evident predominantly in the arterial waveform because the amplitude of the systolic beat differs with every other beat. No changes are apparent on electrocardiograms or in diastolic filling time.1,2

Pulsus alternans may indicate severe ventricular failure.3 Pulsus alternans occurs in aortic and mitral valve stenosis, hypertrophic and congestive cardiomyopathy, effusive pericarditis, and instances in which general anesthesia is used.1,4 Its occurrence always warrants further evaluation to determine the cause.

Case Study

I.B., a 30-kg, 11-year-old boy, had a 5-month history of nausea and vomiting and a 10-kg weight loss. His primary care physician ordered computed tomography of the abdomen to rule out an abdominal process. The scan revealed a mass on the right kidney. The differential diagnosis included renal cell carcinoma, Wilms tumor, oncocytoma, and pheochromocytoma. I.B. was transferred to a children’s hospital for further evaluation.

Upon arrival, computed tomography of the abdomen and chest showed bilateral pleural effusions and a dilated heart. The pediatric cardiologist was consulted because of the enlarged size of the heart. Physical examination revealed diaphoresis, jugular venous distention, S3 heart sounds and a grade 1/6 systolic ejection murmur over the left lower sternal border. I.B. had a positive hepatojugular reflux and cold extremities. His heart rate was 110/min, and his blood pressure was 143/103 mm Hg. Echocardiography revealed an enlarged left ventricle, with ventricular septal bowing toward the right ventricle, severe hypokinesis of the left ventricle, severe mitral regurgitation, and a shortened ejection fraction of 0.05.

I.B. was transferred to the pediatric intensive care unit for further evaluation of heart failure. He was given nitroprusside at a dose of 1 µg/kg per minute for afterload reduction and heparin at a dose of 20 IU/kg per hour to prevent thrombosis in the dilated ventricle. The healthcare team decided that he should be given nothing by mouth for 8 hours and to wait until morning before placing an arterial catheter and central venous access. Cardiorespiratory monitoring was instituted, and pulsus alternans was evident on the plethysmography waveform (Figure 1). This waveform is generated by the oxygen saturation probe; its characteristics are similar to those of an arterial waveform.

The next morning, arterial and central venous catheters were placed. The arterial waveform also showed the beat-to-beat alteration found in pulsus alternans (Figure 2).

As part of the diagnostic evaluation for a pheochromocytoma, a 24-
hour urine sample for measurement of catecholamines was collected. The results indicated an elevation in catecholamine level, confirming the diagnosis of a pheochromocytoma. A pheochromocytoma is a rare tumor of the chromaffin cells located in the adrenal medulla superior to the kidney. These tumors secrete large amounts of catecholamines, including epinephrine and norepinephrine. The catecholamines cause an increase in systemic vascular resistance, leading to hypertension.11 In I.B., the long-term hypertension and decreased afterload led to dilated congestive cardiomyopathy. The degree of pulsus alternans varied throughout I.B.’s hospitalization. The characteristic waveform was present on his plethysmography waveform on hospital day 13, the day before his discharge. The occurrence of pulsus alternans 1 day before his discharge reflects the severity of the heart failure.

I.B.’s clinical condition improved, with an increase in ejection fraction to 0.15, and he was sent home. His medications were phenoxybenzamine 10 mg twice a day, baby aspirin 162 mg/day, enalapril 400 µg 4 times a day, furosemide 10 mg twice a day, and ranitidine 75 mg twice a day. I.B.’s healthcare providers decided to wait 2 months before surgically removing the pheochromocytoma. This time would allow cardiac function to improve and would decrease I.B.’s risk of cardiovascular failure during anesthesia.

Pathophysiology of Pulsus Alternans

The theories about the cause of pulsus alternans can be narrowed down to 2 basic physiological actions. These physiological activities provide the basis for the clinical findings, such as the distinct pattern of the arterial waveform.

The first action is an alteration in end-diastolic pressure that affects the efficiency of the Frank-Starling mechanism.12 The Frank-Starling mechanism accounts for the ability of the heart to increase its output in response to alterations in venous return. When an increased amount of blood enters the ventricle during diastole (ie, when preload increases), the heart is stretched to accommodate the increased volume. Filling of the ventricle is similar to the stretching of a rubber band. The farther the rubber band is stretched, the greater recoil it has. Similarly, the ventricle pumps with a greater force in response to an increased preload.13 Thus, an increase in end-diastolic pressure causes an increase in the force of the contraction. This mech-
anism prevents an increase in the venous pressure during an increase in circulating blood volume. The Frank-Starling mechanism does have its limitations. The ventricle eventually reaches a point at which an increase in preload no longer leads to an increase in contractility (Figure 3).

In a study of 5 children with myocardial disease who had pulsus alternans, Harris et al. found that 3 factors influenced the production of pulsus alternans in regard to left ventricular end-diastolic volume. (1) The diastolic interval preceding the small beats was shorter than the interval preceding the large beats, even when the R-R interval was kept constant by pacing the heart. (2) The left ventricular residual volume was larger after a small beat than after a large beat. This increased volume led to a larger end-diastolic volume preceding the large beat. And as Lee and Sutton reported, a smaller end-diastolic volume preceded the small beat because of the increased emptying with the previous large beat. (3) The left ventricle did not recover after a large beat. The lack of recovery led to a decrease in diastolic compliance, thus inhibiting ventricular filling for the preceding small beat.

The second physiological action that may be the cause of pulsus alternans is the alteration in cellular handling of calcium. Calcium acts directly on the cardiac muscle as a catalyst that leads to a contraction. As the action potential is conducted through the heart, it passes through the T tubules and enters the sarcoplasm. The entry into the sarcoplasm causes a release of calcium from the sarcoplasmic reticulum. Calcium also enters the myofibrils by way of the T tubules. These tubules act as conduits that connect the myofibrils to the extracellular fluid. The calcium traveling into the myofibrils causes a contraction via the sliding of the actin and myosin filaments. At the end of the contraction, the calcium goes back into the sarcoplasmic reticulum and exits via the T tubules.

Calsequestrin is a protein in the inner membrane surface of the sarcoplasmic reticulum that binds and stores calcium. Schmidt et al. found that mice that overexpressed calsequestrin had pulsus alternans during high heart rates. Schmidt et al. concluded that the mice had a delay between the uptake and release of calcium from the sarcoplasmic reticulum and that this alteration in calcium cycling and use led to pulsus alternans.

**Clinical Features of Pulsus Alternans**

Pulsus alternans can be detected via palpation of the artery, use of a sphygmomanometer, and examination of arterial and plethysmographic waveforms. Pulsus alternans is difficult to assess by palpation when the difference between large and small systolic beats is less than 20 mm Hg. The difference in the beats is best detected by palpating the femoral pulses rather than the brachial, radial, or carotid pulses.

For detection with a sphygmomanometer, the cuff is inflated to a pressure greater than peak systolic pressure. The cuff is then slowly deflated while the operator listens for the first audible systolic beats. At this point, only the strong pulses are heard. The cuff is then deflated until all of the systolic beats can be heard. Subtracting the pressure noted at the point when the first beats were heard from the pressure noted at the point where all the beats were heard can be used to quantify the severity of pulsus alternans.

The tracings of I.B. reflect the ability to detect pulsus alternans with the plethysmographic waveform. Monitoring the plethysmographic waveform can be an alternative diagnostic tool if no indwelling arterial catheter is in place. The only other documented use of plethysmographic waveforms to detect pulsus alternans was published by Saghaei and Mortazavian. Pulsus alternans was determined by examination of the plethysmographic waveform in patients undergoing surgery of the
lower extremities. Saghaei and Mortazavian concluded that plethysmographic waveforms could be used to diagnose pulsus alternans, although the sensitivity and specificity of this method have not been determined.

**Nursing Considerations**

In most cases, pulsus alternans is an ominous sign that suggests severe cardiac failure. Critical care nurses must be aware of pulsus alternans and its implications. Patients with this abnormality require further assessment of cardiovascular function. Indications of heart failure may include increased adrenergic activity, congestive hepatomegaly, edema, ascites, pulmonary crinkles, and fever.

During severe heart failure, sympathetic and renin-angiotensin activities increase as a compensatory mechanism in response to a decreased cardiac output. These increases lead to an initial increase in circulating catecholamines and an increase in angiotensin II, which is a potent vasoconstrictor. The peripheral vasoconstriction leads to pallor, diaphoresis, and cyanosis of the nail beds. As heart failure progresses, venous engorgement occurs because of the heart’s inability to eject a large blood volume. The liver often becomes engorged before overt edema develops. The edge of the liver may be palpable below the costal margin, and eliciting the hepatojugular reflux can be used to assess the amount of engorgement. This assessment can be performed by manually pressing in the right upper quadrant of the abdomen for 1 minute. The jugular veins will become engorged as the edematous liver is emptied.

The venous engorgement associated with heart failure also can cause generalized edema, ascites, and pulmonary crinkles. As the systemic venous pressure increases, extracellular fluid is transduced into the surrounding tissue, including the alveoli, leading to the edema and crinkles. Peripheral vasoconstriction may also lead to fever. The vasoconstriction causes an increase in temperature by decreasing the amount of surface area available for conductive loss of heat.

Pulsus alternans can also occur in patients who are not in heart failure. Thus, it is not always an ominous sign. Schaefer et al found that pulsus alternans could be induced in patients undergoing cardiac catheterization. Atrial pacing techniques such as premature atrial contraction and rapid atrial pacing were used to induce pulsus alternans. All of the patients had cardiac disease that required cardiac catheterization, but they did not have pulsus alternans at rest.

**Conclusion**

Pulsus alternans may be a sign of severe ventricular failure. This cardiovascular phenomenon can be caused by alterations in both the Frank-Starling mechanism and intracellular calcium cycling. Pulsus alternans is manifested in various disease states, including mitral and aortic valve disease and hypertrophic and congestive cardiomyopathy. The patient described in the case study had pulsus alternans and congestive cardiomyopathy associated with a pheochromocytoma. Any patient with pulsus alternans should be assessed for signs and symptoms of severe ventricular failure. Such assessment may decrease the prevalence of undiagnosed heart failure.

**References**

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