Murmur Alternans in Aortic Stenosis*

Morton E. Tavel, M.D., F.C.C.P.,** and William K. Nasser, M.D.**

Acquired aortic stenosis, when severe, often gives rise to pulsus alternans.1 Other relatively common causes for pulsus alternans include hypertensive heart disease, coronary artery disease, syphilitic heart disease and cardiomyopathy.2 When pulsus alternans is present, the clinician can usually appreciate it through the use of a sphygmomanometer, in which case when the cuff is inflated to near systolic levels, Korotkoff sounds are transmitted below the cuff with alternate beats. Alternating strength of cardiac contraction may sometimes be ascertained by cardiac auscultation: the first heart sound, the second heart sound and ejection sounds have all been observed to be greater in amplitude with stronger beats.3 Fort et al4 have reported a case with idiopathic cardiomyopathy manifesting pulsus alternans in whom a summation gallop sound was seen to alternate with each alternating beat. Occasional reference5-6 has been made to alternating systolic murmurs in certain cases with pulsus alternans. We wish to report, herein, the case of aortic stenosis with not only striking alternation of a systolic murmur, but also with an alternating diastolic gallop, first and second heart sounds.

CASE REPORT

A 46-year-old white man admitted to the Indiana University Medical Center for cardiac evaluation on March 7, 1969, had noted progressive exertional dyspnea for five years and anginal-type chest pain for one year. He had known of a heart murmur for approximately one year.

Physical examination disclosed blood pressure of 135/90, and approximately 7 mm Hg of pulsus alternans was found. The pulse beat was 100 per minute. The jugular veins were not distended and both carotid arteries were easily felt, with a thrill over these vessels. Cardiac examination disclosed a sustained maximal impulse in the fifth intercostal space at the mid-clavicular line. A grade IV/VI harsh, long, systolic crescendo-decrescendo murmur was heard best in the aortic area and was associated with a systolic thrill. This murmur was transmitted widely and was observed to alternate in intensity from beat to beat. An atrial gallop sound was thought to be present. The lungs were clear. The peripheral pulses were palpable throughout and manifested alternating amplitude.

The electrocardiogram showed changes indicative of left ventricular hypertrophy, but no alternation was found. Chest roentgenograms and cardiac fluoroscopy showed a normal sized cardiac silhouette with some prominence of the left ventricle and considerable calcification of the aortic valve. Phonocardiograms and external pulse tracings are discussed below. Catheterization of the right and left side of the heart gave the following findings: right atrial and right ventricular pressures were normal. The left atrial mean pressure was 6 mm Hg during rest and rose to 15 mm Hg during exercise. Systolic pressures were alternating in both the left ventricle and central aorta by 22 and 8 mm Hg respectively. Average pressure in the left ventricle was 246/12 during rest and simultaneous average pressure in

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21488/)
the ascending aorta was 166/80 mm Hg, giving a peak systolic pressure gradient across the aortic valve of 80 mm Hg. During exercise the left ventricular pressure rose to 262/16 mm Hg with simultaneous pressure in the descending aorta of 176/92 mm Hg, giving a peak systolic pressure gradient across the aortic valve of 86 mm Hg. On close scrutiny of numerous left ventricular pressure pulses, one could observe a slightly more rapid diastolic pressure rise during the diastolic periods preceding the stronger beats, compared to those before the weaker beats. Cardiac index was normal at rest, being 3.84 L/min/M². Following exercise it rose to 4.12 L/min/M². Arterial venous oxygen difference was normal during rest, being 5 volumes percent, but increased to abnormal levels during exercise, being 9.5 volumes percent. There was no evidence of other valve disease or cardiac shunts. Mild aortic insufficiency was seen with the use of selective aortic cineangiography. Calculated aortic valve area was 0.7 cm². Subsequent to hospitalization, the patient was given digitalis and diuretics with considerable relief of symptoms, following which the pulse rate slowed; the alternating pulse and auscultatory findings disappeared. One month later, the aortic valve was surgically replaced with a Starr-Edward prosthetic valve, and alternans did not appear.

**Discussion of Graphic Features**

Figure 1 shows sound tracings recorded over the aortic area and apex, together with the external carotid pulse. Here one can see alternating loud and soft systolic diamond-shaped murmurs. With cycles showing loud murmurs, the carotid pulse shows a relatively long ejection time and a greater amplitude. In addition, the first and second sounds and an ejection click are somewhat larger. Figure 2, which represents apical sounds together with the apexcardiogram, shows alternating gallop sounds along with alternating apical A wave amplitudes. Cycles containing loud murmurs are preceded by larger A waves and relatively large gallop sounds. It is possible that this gallop sound may have represented a summation sound; however, inasmuch as the sound occurred slightly late in relationship to the rapid filling wave of the preceding beat, it probably represents primarily a fourth heart sound. Figure 3 represents similar tracings taken three weeks after intensive therapy for cardiac failure. At this time the rate is considerably slower and the alternation phenomena are virtually absent. The A waves and fourth heart sounds also have become relatively smaller in amplitude.

**Discussion**

Cohn et al have recently detailed the possible mechanisms of pulsus alternans. There are two basic theories to explain its production: 1) alternation of diastolic volume might account for alternating force of cardiac contraction. This is in keeping with Starling's principle, i.e., increased fiber length precedes the more powerful beats; 2) the "myocardial theory" suggests that alternating contractile force occurs in the absence of a fluctuating ventricular diastolic volume. This theory requires either alternation of the basic inotropic state of the myocardium or alternation of the number of cardiac fibers contributing to each systole. Both theories are based upon certain experimental support and neither has been accepted without reservation. The present case fits somewhat better the first theory, inasmuch as the stronger beats...
are longer in duration and encroach more upon the following diastolic period, thus allowing less time for ventricular filling. Moreover, the diastolic pressure seemed to rise more slowly following the stronger beats, an observation consistent with the findings of Gleason and Braunwald, who noted that stronger beats cause more complete emptying of the ventricle.

The systolic murmur, resulting from ejection across the stenotic valve, becomes louder with stronger beats, paralleling a larger aortic pressure gradient, a larger stroke volume, and a greater rate of ejection during these cycles. Alternation of first and second sounds and the ejection click is best explained by the greater rates of change of the ascending and descending pressure curves in the left ventricle with the stronger beats. Alternate explanation for fourth sound alternation is provided by the concept of atrial alternans. The stronger atrial contractions result in more complete ventricular filling and thus, the subsequent ventricular contraction is also stronger. This explanation seems less likely—primarily because of the extreme rarity with which atrial alternans must occur.

Our findings are opposite to those of Fort et al. These latter authors observed a softer gallop sound preceding the strongest ventricular contractions and the louder sounds preceding the weaker ventricular contractions. It is probable, however, that these authors were describing summation gallop sounds which may have been composed chiefly of third heart sounds, rather than fourth heart sounds, as probably represented by our case. Thus, the results between our studies and theirs are probably not comparable. Katz and Feil also noted alternating third heart sounds which were louder after stronger beats in pulsus alternans.

When aortic stenosis manifests alternating strength of ventricular contraction, one is more apt to find a severe grade of aortic obstruction. Such cases more often manifest congestive heart failure, angina pectoris, significant cardiomegaly, higher ventricular pressures (average 220 mm Hg), higher aortic gradients (average 90 mm Hg), and higher left ventricular end-diastolic pressure (average 20 mm Hg), than those cases not showing alternation.

The disappearance of pulsus alternans in our case probably relates not only to increasing cardiac compensation with the use of cardiac therapy, but also to the slowing of rate, a factor which, in itself, can abolish pulsus alternans. Digitalis per se also exhibits such a property. Moreover, pulsus alternans is a relatively labile phenomenon and can manifest spontaneous changes in rapid sequence.

Certain clinical implications may be derived from our observations. In pulsus alternans associated with valvular aortic stenosis, the aortic obstruction may impair the arterial alternation. Thus, the peripheral arterial pulse changes may be difficult to determine with the use of a sphygmomanometer, despite considerable variation of ventricular dynamics and ejection murmur intensity. The auscultatory findings would therefore become quite important in detection of this phenomenon. In hypertrophic subaortic stenosis, stronger ventricular beats might result in even narrower pulse pressures than that seen with weaker beats. In this case, auscultatory findings would be extremely important. In other instances where pulsus alternans is severe, small beats may not be conducted to the periphery. This would give the impression of bradycardia rather than alternation. In such cases, alternating phenomena during auscultation would provide a clue to the presence of alternans. We must stress, however, that the heart rate must be regular before one can diagnose pulsus alternans. If the rate is irregular, longer cycle lengths would be expected to terminate in stronger beats and, therefore, alternans might be simulated.

**Summary**

We have presented a case of severe aortic stenosis with pulsus alternans manifesting alternation of the auscultatory findings, namely, the systolic ejection murmur, diastolic gallop sound, first and second heart sounds and ejection click. Physiologic considerations are discussed. In the presence of a regular heart rate, alternating auscultatory findings have the same clinical significance as the finding of pulsus alternans.

**References**

MURMUR ALTERNANS IN AORTIC STENOSIS


GOBLET CELLS IN THE MUCOSA OF THE LOWER RESPIRATORY TRACT

In chronic bronchitis goblet cells in the walls of the bronchi and bronchioli are increased in number but the increase is not uniform throughout. The degree of hypertrophy of glands and of hyperplasia of the goblet cells in the epithelium of peripheral airways is broadly the same. It may be that from the standpoint of sputum production the goblet cells are less important. But as mucus is produced in larger amounts nearer the periphery of the lung, the difference in the severity of the peripheral change may account for the variation in the natural history of the disease in individual patients. Often sputum production may persist for many years without being accompanied by shortness of breath and without diagnosis of emphysema either clinically or pathologically. The incidence of peripheral infection and distal hypersecretion of mucus may go hand in hand.


THE BLOOD-BRAIN BARRIER

Currently the problem of the nature of volume of brain extracellular fluid is one of the most exciting and important questions in neurophysiology. Electron microscopy of the brain reveals little room, if any, for extracellular fluid. The equilibration of brain fluid with substances usually excluded from cells, such as sulphate, sucrose or insulin, reveals a volume of extracellular fluid which amounts to, at most, 5% of the total fluid content of this tissue as compared to the value of 20% for muscle. It appears that the brain is unique in lacking an important extracellular space as a mediator of water and electrolyte exchange. This fact is presumably the explanation of the blood-brain barrier, which in part, is made up of the plasma membranes of glial cells.