Study of the Murmur Produced by Acute Pulmonary Artery Constriction

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Pulmonary artery constriction has been utilized as an experimental method of stressing the right ventricle or producing changes in the systemic circulation. As a result, the basic circulatory features of pulmonary artery constriction have been documented. The murmur that is produced by this procedure has not received as much attention. It therefore seemed desirable to investigate this aspect of pulmonary artery constriction. The purpose of this report is to summarize these observations.

Methods

Dogs were anesthetized with intravenous sodium pentobarbital (30 mg. per Kg.) and the chest opened using a sternal-splitting procedure under mild positive pressure respiration. The right ventricle was cannulated through the azygos vein and the left pulmonary artery via a side branch. A phonocatheter was also placed in the pulmonary artery by way of a second side branch. This was connected to a Electronics-for-Medicine logarithmic heart sound amplifier. Right ventricular and pulmonary artery pressures were measured electrically using Statham strain gauges and, together with the intrapulmonary sounds, were recorded photographically using both an Electronics-for-Medicine recorder and a Sanborn twin-beam recorder. Respirations were interrupted during all recordings to avoid artifacts due to lung inflation. The zero level for nil pressure recordings was the level of the right atrium.

A ligature was placed around the base of the main pulmonary artery and attached to a screw device similar to that described by Finberg and Wiggers. By tightening this instrument, the loop around the pulmonary artery was reduced in size. This instrument was calibrated so that the reduction in the size of the loop was known for each setting. In addition, immediately following each experiment, the segment of the pulmonary artery containing the stenosis was removed without disturbing the ligature, and the internal diameter was measured for each degree of constriction utilized in that study. As a result, it was possible to quantitate with considerable accuracy the internal cross sectional area of the constriction that was produced in the pulmonary artery by each setting. During a typical experiment the ligature was tightened. Sound and pressure recordings were made and other data obtained. The ligature was then loosened to permit a recovery period before the procedure was repeated at an increased degree of constriction.

Cardiac output was determined in some animals during each of the experimental procedures. The dye-dilution technique, as modified by Opdyke and Sniffen, was utilized with triiodobenazine dye (Cardio-Green*) as the indicator. The dye was injected into the right atrium via a cannula in the right jugular vein, and blood was withdrawn from the femoral artery through a Colson densitometer. Heparin was used as the anticoagulant.

Results and Discussion

Right ventricular and pulmonary artery pressure pulses and intraluminal sound records from the pulmonary artery that were suitable for analysis were obtained from 13 dogs following various degrees of pulmonary artery constriction. In 6 of these animals, the cardiac output was determined during each increment of pulmonary artery stenosis. Right ventricular and pulmonary artery pressure pulses and the intrapulmonary sound tracing from a typical experiment are shown in figure 1. The pressure tracings illustrate the classical changes that have been described for increasing degrees of pulmonary constriction and which are confirmed by our data. Right ventricular pressure increases with only a slight increase in pulmonary artery systolic pressure until a critical degree of stenosis is
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FIGURE 1
A series of records from the same experiment showing the acute effect of varying degrees of pulmonary artery constriction on the intrapulmonary sound, right ventricular pressure, and pulmonary artery pressure. Pressure in mm Hg.

reached, at which point pulmonary artery pressure begins to fall. In our studies, this critical level was reached when the lumen of the pulmonary artery was reduced about 45 per cent. If the constriction is carried much beyond this point, acute right ventricular failure may result. This is shown in the last record of figure 1 where right ventricular diastolic pressure is elevated, systolic pressure is reduced, and pulmonary pressure is markedly lower.

A small diamond-shaped systolic murmur was present in the control record in most of our experiments. This murmur is illustrated in the first record of figure 1. In a few experiments, this murmur was very soft or absent. This is the case in the control record shown in figure 2. A slight degree of pulmonary artery constriction in this animal caused the murmur to appear. This murmur is separated from the vibrations of the first sound and extends nearly to the onset of the second heart sound. A somewhat similar murmur is frequently found in intraluminal recordings from the pulmonary artery of patients and is considered a normal finding in man.

As the lumen of the pulmonary artery was gradually reduced, the first heart sound became more intense. This increase is undoubtedly related to the greater force of right ventricular contraction which accompanies partial occlusion of the pulmonary artery.

At the same time, the duration of the murmur increased. As a result, the murmur tended to merge with the first sound so that the sound vibrations filled the entire interval between the first and second heart sound. The intensity of the murmur tended to increase with moderate constriction of the pulmonary artery, however, a constant relationship was not evident between the intensity of the murmur and degree of pulmonary artery stenosis over the entire range of constriction utilized in this study. In most cases, the peak intensity of the murmur occurred with constriction in the range of 30 to 40 per cent. Constriction of the pulmonary artery beyond this range frequently did not materially alter the loudness of the murmur. In many experiments, the murmur persisted with a moderately loud intensity, even when the stenosis was sufficient to markedly reduce pulmonary flow, as shown by a precipitous drop in pulmonary pressure. Such a murmur is shown in the last record of figure 1.

The failure of the murmur intensity to correlate always with the degree of stenosis may be because the intensity is more directly related to the velocity of flow than to the size of the stenotic area. Bruns has suggested on theoretical grounds that the intensity should be related to the fourth power of the velo-
Intraluminal phonocardiograms recorded from the pulmonary artery and right ventricular pressure recorded from the same animal during various degrees of pulmonary artery constriction. The sound tracing has been darkened for better reproduction. Pressure in mm. Hg.

Dexter and co-workers, however, were unable to find a direct correlation between the intensity of the murmur of clinical aortic stenosis and the calculated velocity of flow through the valve. In an attempt to clarify this point, the velocity of flow through the stenotic portion of the pulmonary artery in our experiments was calculated and compared to the intensity of the murmur. The average velocity of flow per beat was determined by dividing the cardiac output in milliliters per minute by the product of the heart rate in beats per minute, ejection time in seconds, and the cross sectional area of the constriction in square centimeters. The intensity of the murmur was quantitated by measuring its maximum height in millimeters. This measurement gives a relative index of the intensity of the sound for each experiment. The results of four experiments were suitable for analysis. A typical plot of one experiment is shown in figure 3. While there was some grouping of the points at the upper levels of intensity and velocity, it was felt that these plots showed a roughly linear relationship between the intensity of the murmur and the velocity of flow through the constricted area. This relationship becomes more apparent if one considers that the murmur intensity is zero at zero velocity.

The velocity of flow through any tube is inversely related to the cross sectional area and is directly related to the volume flow. It was, therefore, of interest to examine the flow parameter in our experiments. A summary of the cardiac output data, expressed as per
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Plot of the relative intensity of the murmur in one experiment related to the velocity of flow through the stenotic portion of the pulmonary artery. See text for the discussion.

The effect of these changes in cardiac output on the intensity of the murmur was investigated by calculating the volume flow per beat through the stenotic portion of the pulmonary artery. This was determined in the same manner as the velocity except that the cross sectional area was not utilized in the calculation. The average velocity and flow per beat for each experiment, plus the murmur intensity index, were plotted against the per cent reduction in the lumen of the pulmonary artery. A typical plot for one animal is shown in figure 5. This plot shows that both flow and velocity increase with a slight degree of pulmonary artery stenosis. The murmur also increases in intensity. With more severe constriction, the flow begins to return toward control levels; however, the velocity and intensity of the murmur continues to increase. With high degrees of pulmonary artery stenosis, flow falls below control levels; however, velocity remains reasonably fast and the intensity of the murmur is at or near its high point. The divergence between flow and velocity with moderate to severe pulmonary artery stenosis apparently results because the drop in flow is proportionately less than the corresponding reduction in the lumen of the pulmonary artery.
The reasonably close correlation between the intensity of the murmur and the velocity of the flow through the constricted area is again shown by the similarity of the plots of these two parameters in figure 5.

A diamond-shaped systolic murmur, similar to that found in our experiments, is a characteristic auscultatory finding in patients with stenosis of the pulmonary valve. This similarity is not surprising as the dynamics of pulmonary valve stenosis and constriction of the pulmonary artery are essentially the same. Clinical observations have suggested that the peak intensity of the murmur due to pulmonary valve stenosis occurs later with increasing degree of obstruction. It was, therefore, of interest to examine our data in this regard. The time from the onset of the first heart sound to the peak intensity of the murmur was measured and grouped according to the degree of stenosis. The results are summarized in table 1. In three of these experiments the peak intensity did occur later with increasing degree of stenosis; however, in two animals the peak intensity occurred later with moderate degrees of stenosis than with mild or severe constriction, and in five experiments there was no consistent change in the timing of the peak intensity.

The explanation for our inability to confirm a constant relationship between the time of the peak intensity of the murmur and the degree of pulmonary artery constriction is not clear from our data. Perhaps it is because the small vascular volume between the pulmonary valve and the constricted portion of the pulmonary artery acts as a buffering chamber and tends to prevent changes in the timing of the peak flow through the stenotic area.

**Summary**

The murmur produced by acute graded constriction of the base of the pulmonary artery was studied in the anesthetized dog by means of intraluminal phonocardiograms recorded from the pulmonary artery. In addition, right ventricular and distal pulmonary artery pressures were recorded, and the cardiac output was determined for each degree of pulmonary artery stenosis utilized in the study. A small diamond-shaped midsystolic murmur is normally recorded from inside the pulmonary artery. This murmur increases in intensity and duration with constriction of the base of the artery. A constant relationship was not found between the degree of pulmonary stenosis or pulmonary flow and the intensity of the murmur. The intensity did correlate, in an essentially linear fashion, with the average velocity of flow through the constricted area. The timing of the peak intensity of the murmur did not change in any consistent fashion with increasing degrees of pulmonary artery stenosis. Cardiac output and the velocity of flow through the constricted area of the pulmonary artery tended to increase with moderate degrees of constriction. When the lumen of the pulmonary ar-

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**TABLE 1**

*Time in Seconds from Onset of First Heart Sound to Peak Intensity of Murmur*

<table>
<thead>
<tr>
<th>Experiment number</th>
<th>Per cent reduction in lumen of pulmonary artery</th>
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<tr>
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tery was reduced 40 to 45 per cent, cardiac output and pulmonary artery pressure fell below control levels. With further constriction, cardiac output and pulmonary flow decreased; however, the velocity of flow through the stenotic area remained relatively rapid.

References
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