Pulmonary Blood Flow and Venous Return During Spontaneous Respiration

By GERHARD A. BRECHER, M.D., PH.D. AND CHARLES A. HUBAY, M.D.

During normal spontaneous inspiration pulmonary blood flow increases in spite of an increase in resistance to flow in the pulmonary bed. The enhancement of pulmonary flow is caused by an augmentation of venous return due to thoracic aspiration. The right heart acts as a moderator for the pulmonary flow by temporarily storing part of the large influx of venous blood during inspiration and ejecting the stored part during expiration and the expiratory pause.

ONE of the oldest controversies in the field of pulmonary circulation concerns the effect of spontaneous respiration on blood flow in the pulmonary artery. According to pulmonary vascular bed perfusion experiments pulmonary flow diminishes due to an increase in resistance when the lungs are expanded by negative pressure in the closed chest, such as would occur during spontaneous inspiration.1, 2 On the other hand, Baxter and Pearse3 found that, in the intact animal, pulmonary flow was augmented during inspiration. These contradictory findings could be explained only if one assumes that during inspiration venous return increases to such an extent that it augments right heart output in spite of the higher pulmonary bed resistance. Recently it has been demonstrated that venous return actually can increase with inspiration.4, 6 The correlation of such flow augmentation with changes in pulmonary flow has not been established, however, and it was the purpose of this investigation to study this problem.

METHOD

In acute experiments on dogs, blood flow was simultaneously measured in both the main pulmonary artery and the superior vena cava with two 5734 vacuum tube bristle flowmeters. The superior caval flow was taken as representative of venous return since it has been found in former experiments7 that superior and inferior caval flows show the same directional changes during the respiratory and cardiac cycle. Flow in the superior cava was recorded with a bristle flowmeter of a design described previously.7, 8 The cross-sectional area of the vein was fixed for volume flow determination by the ringlike head of the flow cannula inserted into the vessel at the entrance of the right atrium. The internal diameter of the cannula head was 12 mm. Right heart output was recorded by a modified bristle flowmeter inserted into the main pulmonary artery.9 The bristle was introduced into the trunk of the pulmonary artery through a buttonhole incision without blood loss, and the cross sectional area of the vessel was kept constant by passing a suitable metal band around it.

Nine dogs, ranging from 15 to 34 Kg. in weight, were anesthetized with 1.5 mg/kg morphine sulfate and 15 mg/Kg. sodium pentobarbital. The animals were fixed in the right lateral position and the chest was entered on the left side between the 4th and 5th rib. After heparinization the flowmeters were inserted, the chest was closed, and normal intrathoracic pressure relations were re-established. Pressures from side arm tubes on the flowmeters and from the aorta, via a cannula passed down the left carotid artery, were recorded with Gregg optical manometers. Intrathoracic and endotracheal pressures were traced with Frank segment capsules. Zero flows and zero pressures were established at the end of each record. At the conclusion of each experiment the flowmeters were calibrated in situ with the animal's own blood, using steady flows of different magnitudes.

RESULTS

Figure 1 shows a segment of a typical record from a representative experiment demonstrating the effect of spontaneous respiration on venous return and cardiac output. Phasic flows in the superior vena cava and pulmonary artery were recorded. The lowest curve shows the fluctuating changes in venous return with each heart beat. They are characterized by...
systolic (S) and diastolic (D) peaks within each cardiac cycle. Their magnitude was greatly modified by respiration. The amount of blood passing through the superior cava during each heart cycle was calculated from measurements of the area under the flow curve and the figures were then entered on the record. During the expiratory pause, venous return was about the same as illustrated by the first and fifth heart beats (13.5 ml and 12.8 ml).

With the onset of inspiration, venous flow increased as early as the beginning of the second heart beat. The greatest increase (24.1 ml) took place during the third heart beat when inspiration had reached its maximum. With the onset of expiration the return flow of blood was immediately reduced (4th heart beat, 12.3 ml).

Inflow and outflow of the right heart is correlated by comparing the superior vena cava flow tracing with the pulmonary artery flow curve. The stroke volume of the first beat amounted to 30 ml. The second beat had practically the same volume as the first (35.8 ml) in spite of the fact that venous return during the same heart cycle was already greatly augmented. The first sign of an increase in stroke volume with inspiration occurred during the third beat (40.6 ml). The output became largest during the fourth beat (42 ml), though at the same time venous return had become minimal and expiration had already started. The smallest volume was ejected by the last beat (32.2 ml) when venous return was again on the increase. This record demonstrates that pulmonary artery flow increases significantly during spontaneous inspiration, and that changes in venous return are always reflected in the beat output of the following cycle. Thus, due to the sequence of the flow changes the pulmonary artery flow increase must be caused by the augmentation of venous return during inspiration.

While the actual pressures in the pulmonary artery and superior vena cava declined during inspiration, the effective pulmonary artery pressure rose with inspiration as plotted in figure 2. The values for the concomitant flow per cardiac cycle in the pulmonary artery and superior
Table 1—Respirogenic Fluctuations of Right Heart Output and Venous Return in the Closed Chest

<table>
<thead>
<tr>
<th></th>
<th>Average volume of blood inflow per cardiac cycle and outflow (stroke volume) in ml.</th>
<th>Maximal increase above average during inspiration, %</th>
<th>Maximal decrease below average during expiratory pause, %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Averages during 5 normal quiet spontaneous respirations</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior vena cava</td>
<td>17.7</td>
<td>22.0</td>
<td>19.2</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>38.4</td>
<td>8.3</td>
<td>9.9</td>
</tr>
<tr>
<td><strong>B. Averages during 6 spontaneous deep respirations with partially constricted trachea</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior vena cava</td>
<td>16.7</td>
<td>37.7</td>
<td>25.7</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>37.6</td>
<td>11.7</td>
<td>10.9</td>
</tr>
</tbody>
</table>

cava are also shown in this graph. It is noted that the effective systolic and diastolic pulmonary artery pressures increased consistently with each inspiration. This pressure increase was associated with the augmentation of stroke volume. The effective superior vena cava pressure was calculated from the actual pressure measured in this vessel during each cardiac cycle just prior to the opening of the tricuspid valves ("V" point). The effective caval pressure increased during each inspiration concomitantly with the augmentation of caval flow and the increase in effective caval pressure and caval flow preceded by one heart beat the rise of the effective pulmonary artery pressure and the stroke volume augmentation. Toward the end or after each inspiration venous return and effective pulmonary artery pressure fell and the stroke volume decreased. It is of course not to be expected that this sequence of events is demonstrable to the same extent with each inspiration since the ratio of heart cycles to respirations vary and have but a random relationship. For example, in the specific case illustrated in figure 2, there were 18 heart beats during 5 respiratory cycles. However, by analyzing long sequences of respirations in numerous experiments it could be shown that these findings represented a consistent pattern of flow and pressure relations.

Based on the values of the graph, resistance in the pulmonary artery was calculated from the effective diastolic pulmonary artery pressure and equal volume flows. The average resistance during expiration and the expiratory pause was 408 dyn. sec./cm.² and it increased to 481 dyn. sec./cm.² during inspiration.

The respirogenic variations of pulmonary and caval flows in figure 2 demonstrate furthermore that inflow and outflow of the heart are undergoing continuous dynamic changes with respiration. In fact, at no time is there a period which might be called a "steady state." One may calculate the average stroke volume and superior caval flow per heart cycle over a longer time interval, but these values do not give justice to the remarkable respirogenic fluctuations which flow undergoes from one heart beat to the next. Table 1 shows the extent of these fluctuations as they occur in a typical experiment. The figures represent the average flows per cardiac cycle and their fluctuations with respiration expressed as the percentage increase or decrease from the average. All values were taken from one continuous record. The stroke volume increased and decreased above and below the average by 8.3 to 9.9 per cent during quiet respiration, but these fluctuations became somewhat greater with deeper inspirations, though the average stroke volume remained essentially the same. The fluctuations in superior vena cava flow during normal respirations were obviously much greater than those in the pulmonary artery. With deeper inspirations, they were still more pronounced. Comparing the extent of the respirogenic fluctuations in the superior cava and pulmonary artery in table 1 reveals that caval inflow undergoes relatively much greater increases with respiration than cardiac output. Some of the rapidly inflowing blood is obviously stored in the right heart during output and this larger residual volume is then ejected during expiration and the expiratory pause when venous return is minimal. This reservoir phenomenon became even more pronounced when the fluctuations of venous return were larger during deeper inspirations. Comparison of the values in table 1 indicates that output fluctuated only slightly although the venous return fluctuations became greatly enhanced.

Furthermore, the values in table 1 demon-
strate that the greater depth of inspirations which resulted from the partial occlusion of the trachea during the last six respirations did not increase the average venous return. It only led to greater augmentation of superior caval flows during inspirations which were, however, cancelled by greater reductions during expirations and expiratory pauses. The fact that the greater average pressure gradient from the extrathoracic veins to the right atrium which was created by deeper inspirations did not augment the average venous return indicate that the depleting and partial collapse of the extrathoracic veins prevented an increase in the average return flow of the blood to the heart. Reduction of right atrial inflow during the latter part of deep inspirations owing to partial collapse of the extrathoracic veins was also demonstrated in previously described experiments.1-3

DISCUSSION

These experiments clearly reveal the effect of spontaneous respiration on pulmonary artery flow. As it is known from numerous very well documented observations1,2 that resistance to blood flow increases in the pulmonary bed during negative pressure lung expansion, one should expect a decrease in pulmonary artery flow during inspiration. Our experiments corroborated the observation of Baxter and Pearse1 that pulmonary blood flow actually increases during inspiration.

The present experiments furnish direct evidence as to the cause of the augmentation in pulmonary artery flow against a rising pulmonary resistance. We have shown that venous return increased significantly during inspiration and that the augmented inflow during a cardiac cycle always resulted in a larger heart output during the next cardiac cycle. This sequence of events indicates that the augmentation of pulmonary artery flow with inspiration is caused by increased right heart filling and is not due to a reflex or an intrinsic mechanism of the right ventricle.

Our experiments also demonstrate that the resistance in the pulmonary artery increases under the normal physiological conditions of lung expansion by spontaneous inspiration. Though the increase in resistance is only small (18 per cent during quiet inspiration), it is believed that this increase is real because it was calculated by comparing ratios of pressures to flows both under conditions of equal flows or equal pressures. The calculation of the stroke volume and caval flows per heart cycle showed that during inspiration right atrial inflow from the superior cava was relatively larger than outflow from the right ventricle. As it has been previously demonstrated that the inferior cava contribution is similarly augmented during inspiration,4,5,6 this would mean that during inspiration more blood entered the right heart than was ejected. Thus the combined action of an increase in venous return and pulmonary bed resistance must result in a dilatation of the right heart during inspiration. The surplus blood which is momentarily accommodated in the right heart during inspiration is then released into the pulmonary circuit after inspiration is over.

SUMMARY

In anesthetized closed chest dogs flow was measured simultaneously in the main pulmonary artery and the superior vena cava with two 5734 vacuum tube bristle flowmeters. Superior caval flow was taken as representative of venous return.

It was found that right heart stroke volume increased consistently during normal quiet spontaneous inspiration. Venous return also increased with inspiration but this happened always one heart beat before the augmentation of the ventricular stroke volume occurred. From this sequence of events, it was concluded that the inspiratory augmentation of venous return was responsible for the increase in pulmonary artery flow.

Augmentation of pulmonary artery flow occurred despite a small increase in pulmonary bed resistance during inspiration. As a result of this resistance increase and an augmentation of venous return, the residual volume in the right heart became larger during inspiration. During expiration and the expiratory pause, part of the residual volume was ejected.

Attention is called to the fact that at no time can one observe a "steady state" of the circu-
lation since venous return and right heart output continuously undergo marked dynamic changes during spontaneous respiration.

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REFERENCES

Preoperative Determination of the Severity and Site of Uncomplicated Pulmonary Stenosis

Catheterization studies of 12 patients with uncomplicated pulmonary stenosis confirm previous work that this lesion does not cause significant hemodynamic changes at rest or moderate exercise unless the size of the orifice calculated by Gorlin and Gorlin's formula is less than 1 cm². In the latter cases only does one encounter significant differences in pulmonary and right ventricular systolic pressures, reduction in cardiac output at rest, and failure to increase output in accordance with increased metabolism during physical effort with the result that the oxygen utilization coefficient is diminished during exercise.

The observation of Kirklin and Associates (Circulation 8: 849, 1953) is confirmed that the site of obstruction can be determined by fluoroscopic localization of the catheter while it is withdrawn from the pulmonary artery into the right ventricle, and noting the nature of the pressure changes. In infundibular stenosis the catheter passes through a zone in which systolic pressure approximates that in the pulmonary artery during systole and that in the right ventricle during diastole; in the orificial stenosis the change of pressures is abrupt when the catheter has passed the valves.

A survey of the cases reveals that all of the severe cases established by catheterization complained of dyspnea of effort; the less severe ones did not. It is obvious that, while not emphasized in this report, the presence or absence of effort dyspnea in uncomplicated pulmonary stenosis should not be overlooked in considering surgical intervention.

For details see L. Leguime, "Physiopathologic de la stenose pulmonaire isolee," Acta cardiol. 9, 298, 1951.
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