The Pulmonary Pressures in Pulmonary Venous Obstruction

By Merritt C. Warren, M.D., Philip M. Benaron, M.D., and Norman J. Sissman, M.D.

SUMMARY

The contour of the pulmonary artery wedge pressure tracing in a case of pulmonary venous obstruction was unique and may be diagnostic. Wedged pressures were obtained in both a pulmonary artery and a pulmonary vein on recatheterization of a 5-year-old boy with total anomalous pulmonary venous drainage to the superior vena cava on whom an anastomosis of the confluence of the pulmonary veins to the left atrium with loose ligation of the vertical vein had been done at 2 months of age. The pulmonary artery wedge pressure (oxygen saturation, 97%) was 31/18 (mean, 22) mm Hg and its contour was almost identical to that of the pulmonary artery pressure. A similar contour was obtained in the pulmonary vein, for both wedged and free pressures. The similarity of the pulmonary artery wedge pressure to that in the free pulmonary artery may lead to the conclusion that the catheter is not wedged when actually it is. Doubt can be resolved by withdrawing fully saturated blood. Extreme stenosis between the confluence of the pulmonary veins and the left atrium may cause the pulmonary artery wedge to reflect pulmonary arterial rather than left atrial pressure and pulse form.

Additional Indexing Words:
Pulmonary artery wedge pressure
Pulmonary hypertension
Anomalous pulmonary venous drainage
Pulmonary venous wedge

SINCE first described by Hellms and associates in 1945, determination of the pulmonary artery wedge pressure has come into routine use in right heart catheterizations. Following some initial controversy on the subject, the publications of Werkö,2,3 and Connolly4–6 and their associates reported a good correlation in humans between the level of the pulmonary artery wedge pressure and the left atrial pressures and often a close similarity in contour. Epps and Adler7 also found a good correlation. Pedersen8 found that the pulmonary artery wedge pressure had pulsations in 50% of the cases in which pressures were normal and in 90% of those in which pulmonary artery wedge pressures were elevated. He noticed that the configuration was venous and that the a waves never exceeded the v waves.

The pulmonary artery wedge pressure in a case of pulmonary venous obstruction which we recently studied was much higher than normal, and the contour was almost identical with that of the pressure wave in the pulmonary artery. These features may lead to failure in recognizing such a pressure as being a true wedge pressure. While the pulmonary artery wedge pressure has been reported as elevated in many cases of pulmonary venous obstruction, few cases have been reported in which the contour was similar to that found in this case and, to our
knowledge, this point has not been emphasized. Accordingly, the case history and pertinent catheterization data are presented.

**Report of Case**

R. F. (P.A.-S. 09-43-81), born on March 29, 1962, had tachypnea from birth and poor weight gain during the first few weeks of life. At 6 weeks of age, dyspnea, fever, and tachycardia developed for which he was hospitalized and digitalized. Physical findings at that time included mild cyanosis with crying, a right ventricular lift, a grade III/VI rough, systolic murmur at the mid left sternal border transmitted to the back, a grade II/VI short, low-pitched mid-diastolic murmur at the apex and the lower left sternal border, and a high-pitched continuous murmur, grade II/VI, localized in the left infraclavicular area. Chest x-rays showed moderate cardiac enlargement and marked increase in pulmonary vascularity. The electrocardiogram showed right ventricular and right atrial hypertrophy.

Cardiac catheterization and selective cineangiography were performed on June 1, 1962. The physiological data are presented in table 1. They confirmed the clinical diagnosis of total anomalous pulmonary venous connection to a persistent left superior vena cava with moderate elevation of pulmonary vascular resistance.

At 2 months of age, two anastomoses were created surgically by Dr. Norman Shumway, one 1.2 cm in diameter between the confluence of the pulmonary veins and the left atrium and another 1.1 cm in diameter between the left atrial appendage and the vertical vein. The persistent left vertical vein was constricted by a suture to approximately half its former diameter. Following a stormy period immediately after operation, the patient was discharged as improved. Only a grade II/VI, systolic ejection murmur in the pulmonary area remained. During the next few months he had repeated episodes of acute dyspnea which responded to oxygen and therapy for congestive failure.

At 7 months of age, because of these symptoms, he was recatheterized with the results depicted in table 1. No evidence of a left-to-right shunt was found. The mean pressure in the pulmonary artery was 35 mm Hg, that in the right pulmonary artery wedge was 14 mm Hg, and the tracing showed small a and v waves with marked respiratory variation. No further surgery was performed.

After the age of 1 year the patient did well with slightly subnormal exercise tolerance. His growth was good, and there was no further need for cardiac medication. However, during the fourth year of life, a new continuous murmur and increase in the intensity of the second sound in the pulmonic area were noted. These physical findings plus continued right ventricular hypertrophy on the electrocardiogram prompted a third cardiac catheterization.

Physical examination at the time of the last study showed normal height (108 cm), weight (17.4 kg), vital signs and blood pressure (right arm, 97/58 mm Hg; right thigh, 125/65).

There was a moderate right ventricular heave but no thrill. The apical first sound was normal. The second sound in the pulmonic area was moderately accentuated and had a normal variable split. There was a grade III/VI, medium-pitched, continuous murmur, maximal in the third left intercostal space somewhat lateral to the left sternal border. The diastolic portion of

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**Table 1**

**Catheterization Data**

<table>
<thead>
<tr>
<th>Site</th>
<th>Age, 2 mo</th>
<th></th>
<th>Age, 7 mo</th>
<th></th>
<th>Age, 5 yr</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pressure* (mm Hg)</td>
<td>O₂ sat (%)</td>
<td>Pressure* (mm Hg)</td>
<td>O₂ sat (%)</td>
<td>Pressure* (mm Hg)</td>
<td>O₂ sat (%)</td>
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<tr>
<td>Innominate vein, left</td>
<td>(6)</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td>83</td>
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<tr>
<td>Innominate vein, right</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Superior vena cava</td>
<td>97</td>
<td>53</td>
<td></td>
<td>73</td>
<td></td>
<td>82</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>53</td>
<td>65</td>
<td></td>
<td>73</td>
<td></td>
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</tr>
<tr>
<td>Right atrium</td>
<td>(2)</td>
<td>96</td>
<td>(0)</td>
<td>50</td>
<td>(2)</td>
<td>76</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>52/2</td>
<td>89</td>
<td>54/2</td>
<td>52</td>
<td>45/17</td>
<td>76</td>
</tr>
<tr>
<td>Main pulmonary artery</td>
<td>46/15</td>
<td>(29)</td>
<td>52/19</td>
<td>45/17</td>
<td></td>
<td></td>
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<tr>
<td>Right or left pulmonary</td>
<td>52/2</td>
<td>93</td>
<td>52/22</td>
<td>(35)</td>
<td>39/22</td>
<td>(26)</td>
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<tr>
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<td></td>
<td>(14)</td>
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<td></td>
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<tr>
<td>Left pulmonary vein</td>
<td>38/22</td>
<td>(28)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>wedge</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td>Left pulmonary vein</td>
<td>37/21</td>
<td>(30)</td>
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<td></td>
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<tr>
<td>(free)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrium</td>
<td>—</td>
<td>(9)</td>
<td></td>
<td></td>
<td></td>
<td>96</td>
</tr>
<tr>
<td>Right femoral artery</td>
<td>63/41</td>
<td>(52)</td>
<td>76</td>
<td></td>
<td>114/50</td>
<td>(70)</td>
</tr>
</tbody>
</table>

*Pressure (mm Hg): systolic/diastolic; mean in parentheses.
the murmur was well transmitted to the apex, while the systolic portion was better transmitted to the left sternal border. The liver was not enlarged. The electrocardiogram showed right ventricular hypertrophy. Chest x-rays showed enlarged pulmonary veins and small peripheral vessels interpreted as dilated lymphatic channels.

Cardiac catheterization was done on January 30, 1967, from the right saphenous vein. The results are listed in Table 1. The catheter was manipulated across the atrial septum from right atrium to left atrium and then through the lower of the two surgical anastomoses into a left pulmonary vein. The physiological findings will be discussed in detail below. In essence, they showed marked pulmonary venous hypertension together with normal left atrial pressure and normal pulmonary arterial vascular resistance (pulmonary arterial and venous pressures were practically identical) and no shunting. There was "arterialization" of the pulmonary venous pressure tracing with an abrupt change in the level and contour of the tracings on withdrawing the catheter from the pulmonary vein into the left atrium (Fig. 1). Biplane cineangiography (Fig. 2) demonstrated the anatomy of the anastomoses and showed dilatation of the pulmonary arterial, capillary, and venous vascular beds. The left vertical vein was not patent.

On March 7, 1967, the patient underwent reoperation by Dr. Shumway, who made a new, larger anastomosis between the confluence of the pulmonary veins and the left atrium, and closed a small patent foramen ovale. No lung biopsy was obtained. The postoperative course was uncomplicated.

Discussion

The unusual opportunity to obtain, in this patient with extracardiac pulmonary venous obstruction, free and wedged pulmonary venous as well as arterial pressures yielded several findings worthy of discussion.

The outstanding phenomenon observed was the near identity both of levels of pressure and of contours of the pressure waves in the pulmonary arterial and venous systems; the latter we have labeled "arterialization" of the pulmonary artery wedge and venous pressure tracings. This extended transmission of the arterial pulse wave may be attributed to several factors. Undoubtedly the small diameters of the pulmonary capillaries play a large role in the normal damping of arterial pressure waves, as has been commented upon by Fishman. Further loss of pressure characteristics occurs, under usual circumstances, when the waves reach the large volume, relatively compliant, low pressure area of the combined venous and left atrial segments of the pulmonary circulation. In our case, the site of the stenosis effectively excluded the atrial chamber from the system. Braun and Stern pointed out that the venous component of the pulmonary circulation, at least in the isolated rabbit lung, has a high degree of compliance and is able to provide storage for as much as two thirds of the entire pulmonary volume. However, Caro and associates concluded from their studies that compliance of

![Figure 1](http://circ.ahajournals.org/)

Figure 1

Pressure tracings from recatheterization at 5 years of age. Note the basic similarity of the wave form and pressure levels in the free pulmonary artery, the right pulmonary artery wedge, the pulmonary vein wedge, and the free pulmonary vein. Also note the abrupt drop in pressure and change in pattern on withdrawal from the pulmonary vein to the left atrium.
the pulmonary veins in the dog and rabbit is markedly reduced when the mean pressure is elevated, and they implied that this has been observed in man also. Caro's group obtained "arterialization" of the pulmonary venous pulse wave after snaring segmental pulmonary veins in the dog, although delay and attenuation of the waves were greater in their experimental animals than in our patient. That even the normal capillary system is capable of transmitting arterial pulse waves has been well known since the re-

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Frames from cineangiograms obtained at 5 years of age. (A) Left lateral projection with injection into the confluence of the pulmonary veins (CPV). The arrow marks the jet of contrast material passing into the left atrium. (B) Later frame, same cineangiogram. Arrow marks jet passing through the second anastomosis into the left atrial appendage. Contrast material is now seen in the left atrium (LA). (C) Posteroanterior projection with
peated demonstration$^9,12$ that a catheter wedged in a pulmonary vein will record such waves has been well known since the retention of the capillaries and elevation of the pulmonary venous pressure would favor transmission of the arterial pulse wave. The angiocardiograms in our case (fig. 2) confirmed the presence of dilated capillaries and veins.

Morphological changes that accompany long-standing pulmonary venous hypertension tend also to decrease the compliance of the capillary and venous beds. As described by Ferencz and Dammann$^{13}$ and Harris and

Figure 2 (continued)

*inadvertent injection into the left pulmonary artery. Note the considerable dilatation of the pulmonary arteries especially peripherally. (D) Frame taken during capillary phase of the same cineangiogram. Note the dilatation of the capillary system and the blush in the lower left portion of the lung field. (E) Later frame from the same cineangiogram. Visualized are the confluence of the pulmonary veins (CPV), the left atrium (LA), the left ventricle (LV), and the aorta (A). There is no evidence of a left-to-right shunt.*
Heath, these consist of thickening of the muscular media of the veins and venules and, in some cases, intimal proliferation. In addition thickening of alveolar walls surrounding capillaries occurs. Unfortunately no lung biopsy was obtained from our patient.

In summary, the combination of exclusion by the stenosis of the compliant left atrium from the pulmonary venous system, dilatation of the capillaries, and elevation of venous pressure due to the blockage and probable morphological changes associated with chronic venous hypertension, all contributed to bringing about the observed "arterialization" of the pulmonary artery wedge and pulmonary venous pressures.

The second physiological finding of note was the normal pulmonary arteriolar vascular resistance in the presence of chronic marked pulmonary venous hypertension. The cause of pulmonary arterial hypertension in patients with left-sided obstruction has been the subject of much analysis and comment. Dexter and associates pointed out many years ago that a rise in pulmonary "capillary" pressure up to 25 mm Hg causes a passive proportional rise in the pulmonary artery pressure. In many clinical instances, however, documented since Dexter's observations, elevated pulmonary arterial resistance is found with less severe degrees of pulmonary capillary hypertension; the natural history of patients of this type is often unknown. Was there, at one time in these patients' past, a period when their pulmonary capillary pressure exceeded 25 mm Hg?

Jordan has recently reviewed various theories regarding the cause of pulmonary hypertension in mitral stenosis. He found that the most popular hypotheses, namely, that of organic obstruction of drainage from the pulmonary capillary bed followed by a passive rise in pulmonary artery pressure which initiates a vicious spiral of constriction of the pulmonary arterioles, further rise in pressure, and then further constriction, and, alternately, that of constriction of the pulmonary arterioles secondary to reflexes arising from pressure receptors in the pulmonary veins or left atrium, or both, did not fit all of the known facts. He postulated that the stimulus for constriction of the pulmonary arterioles is alveolar hypoxia caused by thickening of the alveolar membranes, due to edema and fibrosis, which occurs following elevation of pulmonary venous pressure. That pulmonary hypertension secondary to mitral stenosis can be reversed rapidly has been shown by recent studies of patients immediately after mitral valvotomy.

Perhaps the slow development of the pulmonary venous hypertension in our patient permitted the development of such efficient alternative pathways of drainage via the lymphatics that alveolar hypoxia did not occur. Or, one may fall back on the unknown factors responsible for individual as well as species variability relative to vascular reactivity, commented upon by Grover and co-workers, to explain our patient's lack of increased pulmonary arteriolar resistance. Again, a lung biopsy would have been helpful in answering some of the questions raised by the available data.

Our patient was remarkably free of the symptoms and signs of elevated capillary pressure (dyspnea, orthopnea, and pulmonary edema) usually associated with pulmonary capillary mean pressures of over 25 mm Hg. Here, too, the early age of onset of the pulmonary venous hypertension and its chronicity may have permitted the development of unusually large alternative pathways of lymphatic drainage and prevented fluid accumulation in the alveolar walls and spaces. Rabin and Meyer did not find increased lymphatic drainage in dogs with surgically produced chronic left atrial obstruction. Their experiments, however, were done on mature animals. Conventional chest x-rays in our patient did show evidence of dilated lymphatic channels in the peripheral lung fields, although classical Kerley's lines were not seen.

The occurrence of "arterialization" of the pulmonary artery wedge pressure has practical implications for assessing the results of catheterization studies in patients with pul-
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monary venous hypertension. The similarity to pulmonary artery pressures may prevent recognition of the fact that the catheter has been adequately wedged and thus result in failure to appreciate the existence of the pulmonary venous hypertension. Many examples of wedge pressure tracings with large solitary systolic waves can be found in the literature, but most of these have been in cases of mitral regurgitation; the contours of these waves are more symmetrical, do not have as clear an incisura as was seen in the tracings in our patient, and, in general, do not bear so close a qualitative or quantitative relationship to the pulmonary artery pressure. Elevation of pulmonary venous pressure has been described in cases of anomalous pulmonary venous connection alone, or together with cor triatriatum, and in cases of cor triatriatum alone. Wender and associates described "arterialization" of pulmonary artery wedge pressures in patients with anomalous pulmonary venous drainage but did not present firm evidence that their catheters were adequately wedged. In case 3 of Jegier and associates' study, interpretation of the high wedged pressure as that of a damped distal pulmonary artery pressure resulted in diagnostic error. Congenital anomalies in which circumstances similar to those present in this case would predispose to the occurrence of the phenomenon described, include those of cor triatriatum, stenosis or atresia of the common or individual pulmonary vein and anomalous pulmonary venous connection with obstruction to pulmonary venous drainage. Realization that "arterialization" of the pulmonary artery wedge pressure may occur will aid in accurate assessment of some of these patients, and if it can be obtained, withdrawal of fully saturated blood from the catheter thought to be wedged will confirm its true position.

Venables and co-workers reported a case of total anomalous pulmonary venous drainage which had a continuous murmur over the area of the vertical vein which was somewhat similar to the murmur noted in this case.

Acknowledgment

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