Unequal Pressures in the Central Pulmonary Arterial Branches in Patients with Pulmonary Stenosis

The Influence of Blood Velocity and Anatomy

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SUMMARY. Significantly different pressures in the right (RPA) and left (LPA) pulmonary artery were observed at catheterization in patients with pulmonary valvar stenosis and no branch stenosis. The lower pressures in the RPA showed a “valley” during systole and were similar in contour and amplitude to the main pulmonary arterial (MPA) pressure; the LPA pressure, however, had a normal contour, and the peak systolic and mean pressures were higher than those in the MPA and RPA.

Angiocardiograms, phonograms, and a simple analysis of fluid mechanics suggest that this pressure pattern is related to (1) the high-velocity jet in the MPA and (2) the anatomy of the central pulmonary arterial branches (bifurcation), the LPA originating more distally than the RPA. The high-velocity jet bypasses the origin of the RPA and breaks up in the distal MPA near the origin of the LPA. The kinetic energy is then reconverted into pressure, causing the higher LPA pressures.

In patients with transposition of the great arteries and subvalvar pulmonary stenosis, the anatomy of the main pulmonary arterial bifurcation is different from normal, the RPA originating more distally than the LPA. The high-velocity jet may bypass the origin of the LPA and break up near the more distal origin of RPA, and the pressures in the RPA can be higher than those in the MPA and LPA.

KEY WORDS: Hemodynamics — Central pulmonary arterial branches — Pulmonary valvar stenosis — Bernoulli effect

Significant systolic pressure differences across valvar, subvalvar, or vascular narrowings have been a common observation at cardiac catheterization in patients with congenital or acquired heart disease [3, 4, 12, 15]. Even when there are no demonstrable structural abnormalities, small pressure differences can be observed between the main pulmonary artery and its two primary branches [19]. We have observed that the recorded blood pressures in the left and right pulmonary arteries were significantly different from each other in some patients with pulmonary valvar stenosis even in the absence of branch stenosis and that the peak systolic and mean pressures in the left pulmonary artery were higher than those recorded proximally in the main pulmonary artery.

In patients with pulmonary valvar stenosis, the relation between the severity of the obstruction and the pressure drop across the pulmonary valve was quantitated by Gorlin and Gorlin [12]. Bouchard and Cornu [2], Watson and Lowe [21], and Kaindl et al [13] observed the presence of a depression during systole in the main pulmonary arterial pressure recording and an increase in the pulmonary arterial systolic pressures more distally.

The present study concerns an unusual pressure
relationship observed in patients with pulmonary valvar stenosis, where the recorded blood pressure in a primary branch of the main pulmonary artery is higher than that recorded in the main pulmonary artery and the contralateral primary branch. Similar observations were made in patients with transposition of the great arteries and pulmonary subvalvar stenosis. Our study emphasizes the importance of the architecture of the main pulmonary arterial bifurcation and of the increased blood velocity in the main pulmonary artery in the genesis of these unusual pressure patterns. It also provides further insight into the possible role of these factors in the unequal vascularity [22] and blood flow distribution pattern [5, 17] observed between the right and left lungs.

Clinical Material and Methods

Observations on three patients are presented together with intracardiac pressure and phonogram recordings and with selective cineangiograms. Patient 1 (6 years old) had tetralogy of Fallot with severe pulmonary valvar stenosis. Patient 2 (10 years old) had moderately severe isolated pulmonary valvar stenosis. Patient 3 (17 years old) had d-transposition of the great arteries with pulmonary subvalvar stenosis and was studied after successful intra-atrial (Mustard) repair.

Catheterization studies were performed in room air. The patients were sedated with Innovar Injection (fentanyl citrate and droperidol). Pressures were recorded using a fluid-filled USC1 No. 7F pediatric angiography catheter connected to a Statham P23H pressure transducer and introduced percutaneously. The pressures were also recorded using a Millar No. 5F transducer-catheter, splitting the signal for simultaneous intracardiac phonogram recordings. Changes in velocity were inferred from the phonograms. After the pressure recordings were obtained, angiography was performed using Renografin 76 with biplane 35-mm cine recordings or serial cut films.

The Millar transducer is positioned in a slight depression on the distal lateral catheter wall and has been shown to record accurately lateral intravascular pressures in large vessels [16].

Results

The resting peak-to-peak systolic pressure difference across the pulmonary valve in patient 1 was 62 mm Hg. Patient 2 had a resting pressure difference of 45 mm Hg; after intracardiac administration of isoproterenol (Isuprel), the pressure difference increased to 120 mm Hg. Pressures recorded in the central pulmonary arterial system indicate that in these two patients with pulmonary valvar stenosis, the left pulmonary arterial peak systolic and mean pressures were significantly higher (8 to 10 mm Hg and 4 mm Hg, respectively) than those recorded in the main or right pulmonary artery. These absolute pressures were determined from the recordings obtained with liquid-filled angiography catheters, since pressures measured with Millar catheters may be influenced by the gravitational level of the transducer.

The pressure waves recorded in the main, right, and left pulmonary arteries in patient 1 (Fig. 1) showed a marked difference between the waveform in the left pulmonary artery and those in the right and main pulmonary arteries. The pressure wave contour in the main pulmonary artery showed that at the beginning of the ejection period the pressure initially increased rapidly (a-b); however, early in systole the pressure started to decrease (b). After reaching a nadir (c), the pressure again increased in the latter half of the ejection period (c-d), resulting in an ejection "valley" pattern. During the remainder of the cardiac cycle (after d), the pressure decreased as in the normal circulation with end-systole indicated by the incisura (e). The pressure wave in the right pulmonary artery had a contour similar to that recorded in the main pulmonary artery. In the left pulmonary artery, however, the pressure wave was markedly different and had an essentially normal arterial configuration.

The intracardiac phonograms (Fig. 1) indicate that the systolic murmur was loudest in the main pulmonary artery but faded abruptly in both the right and left pulmonary arterial branches. The contour of the systolic murmur (crescendo-plateau-decrescendo) in the main pulmonary artery can be correlated with the pressure events. The onset of the murmur, reflecting the development of turbu-