Role of Kinetic Energy in Pulmonary Valvar Pressure Gradients

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SUMMARY

Peak systolic pulmonary valvar pressure gradients are frequently seen in large intracardiac left-to-right shunt lesions. Conventionally, this has been attributed to "relative" pulmonic stenosis. Theoretical considerations suggest that this gradient is due to differences in expression of the total fluid energy. The side pressure, the downstream pressure, the end pressure, and the flow velocity in the pulmonary artery (PA) and the right ventricle (RV) pressure were measured in 11 dogs in the control state, and after increasing the velocity of the pulmonary arterial flow either by administration of isoproterenol or atropine, or by creating an arteriovenous fistula. The RV pressure and the end PA pressure were not significantly different ($P > 0.05$), and were higher ($P < 0.01$) than the side and downstream PA pressure in the control state, but increased to higher levels ($P < 0.01$) after increasing the velocity of flow in the pulmonary artery. The mean pulmonary valve peak systolic gradient (RV–side PA pressure difference) was 8.8 mm Hg in the control state and increased to 19.1 mm Hg after isoproterenol infusion ($P < 0.01$). This change in pulmonary valvar gradient is proportional to the increase in the PA flow velocity.

The side pressure measures only the potential energy, and the end pressure measures both the potential and kinetic energies. In the right ventricle, only the potential energy is recorded where the kinetic energy is practically nonexistent. The difference between the RV (or end PA) pressure and side PA pressure is proportional to velocity of flow in the pulmonary artery and is due to partial transformation of fluid energy into kinetic energy. Thus, our study helps to explain the pressure gradient across the pulmonary valve in large left-to-right shunt lesions. These studies also raise questions as to the validity of interpretations of the gradients produced in the RV outflow tract after isoproterenol or exercise.

Additional Indexing Words:
Hydraulic (fluid) energy Isoproterenol End pressure Side pressure
Intracardiac left-to-right shunt lesions

PULMONARY outflow tract peak systolic pressure gradients have been well documented in large left-to-right shunts resulting from atrial septal defects,[1–5] ventricular septal defects,[1–3] and atrioventricular canal.[2] These gradients were generally felt to be at the pulmonary valvar level,[1, 2, 4, 5] although one author[3] found a gradual change of pressure from the pulmonary artery (PA) to the right ventricle (RV). The transpulmonary valvar gradient has been attributed to "relative" or "functional" pulmonic stenosis.[2, 4, 5] These authors stated that in large intracardiac left-to-right shunts there is dilatation of the right ventricle and the pulmonary artery without dilatation of the pulmonary valvar ring and they postulated that the normal size pulmonary valve annulus separates the enlarged RV and PA and therefore is relatively or functionally stenotic. We postulate that differences in the expression of the total fluid (hydraulic) energy are responsible for the transpulmonary valvar gradient. The purpose of this paper is to present experimental data to support this hypothesis and to discuss the role of kinetic energy in the production of pulmonary valvar gradients.

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Materials and Methods

Eleven mongrel dogs weighing between 16 and 25 kg were anesthetized with a mixture of sodium pentobarbital and sodium thiamylal and were placed on a positive pressure, volume regulated, piston pump Harvard respirator. The chest was opened through a left lateral thoracic incision in the 4th intercostal space. A polyethylene catheter was placed in the left atrium through the left atrial appendage for monitoring blood gases and appropriate adjustments in ventilation were made to achieve the physiologic range. When necessary, sodium bicarbonate was administered to maintain normal pH. The dogs were then heparinized. A catheter-tip flow probe (Biotronex Model BL-9070) was introduced into the main PA through a stab incision in the RV as was a side-hole catheter of the NIH type. The flow measuring system is located in the distal 2 cm of a 100 cm woven Dacron No. 7F catheter. This consists of a thin-wall epoxy cylinder through which the blood flows, an electromagnetic induction transducer consisting of a single solenoid coil located adjacent to the flow-through cylinder, and signal-sensing electrodes consisting of two parallel platinum wires placed on the inner surface of the epoxy cylinder. The epoxy cylinder is 7.5 mm in length with an internal diameter of 3 mm and an external diameter of 3.5 mm at the tip and causes a uniform flow of blood past the signal electrode. This catheter-tip flow transducer measures volume flow when wedged into a small vessel and measures velocity of flow when placed into a large vessel. An end-hole catheter was placed in the PA in the direction of the stream and another against the direction of the stream. A third end-hole catheter was placed in the RV. After several preliminary experiments, the catheter in the direction of the stream and the RV catheter were replaced with B-D Longdwel (end-hole) teflon catheters (#17). Similarly, the end-hole catheter against the stream was replaced with a #17 needle introduced via the superior aspect of the pulmonary artery (fig. 1). These changes produced quality recordings (with less catheter flipping) without affecting the aim of the study.

The pressures and flow were recorded on a Honeywell oscillographic recorder with a paper speed of 100 mm/sec. P23 De or P23 Bb Statham transducers were used for the pressures and Biotronex flow meter for the flow velocity signal. The transducers were balanced and calibrated prior to each experiment. Static calibration of the catheter-tip flow probe signal was done *in vitro* at various flow rates in a straight tube using blood and was found to be linear. Dynamic calibration of the catheter-tip flow probe was obtained by comparing with a previously calibrated perivascular flow probe measurement in a pump driven closed circuit. A good match in contour was noted at various flow levels covering the range in our experiments. Zero level of flow velocity is frequently difficult to determine but the moduli of harmonics are considered free from the effect of zero drifting if the signal is recorded in a steady state.

The side pressure (side-hole catheter), the downstream pressure (end-hole catheter in the direction of the stream), the end pressure (end-hole catheter against the direction of the stream) (fig. 1) and the flow velocity in PA and the RV pressure were recorded in the control state and after increasing the velocity of the pulmonary arterial flow by administration of isoproterenol (0.4 to 0.8 mg in 50 cc of 5 g/100 ml dextrose by rapid intravenous infusion) in 11 dogs. The respirator was interrupted in expiration during the pressure and flow recording. Similar data were recorded in three dogs prior to and after administration of atropine (1 mg I V) or after creation of arteriovenous (bilateral femoral artery to vein) shunt. Peak systolic and diastolic pressures of all four types of pressure recordings, mean left atrial pressure, peak flow velocity and heart rate were measured in the control state and after the given stimulus. The measurements were obtained from a single beat selected from among five or more beats of stable recording. The PA circumference was measured after isoproterenol was discontinued and the pressure in the PA at the time of recording was between control and experimental pressure levels. The pulmonary arterial cross-sectional area was calculated from the pulmonary arterial circumference. From this and the peak flow velocity, the volume flow (peak) in the PA was calculated.

Figure 1

Catheter locations as used in the experiments. Catheter-tip flow probe is not shown in the figure but was located at the level of the other catheters. PA = pulmonary artery; RV = Right ventricle.

Results

Before the results are presented, it might be worthwhile to review what each of the pressures recorded represents. The side pressure measures only the potential energy, the downstream pressure measures potential energy minus some of the kinetic energy and the end pressure measures both the potential and kinetic energies. In the right ventricle only the potential energy is recorded; the kinetic energy is largely nonexistent.

Isoproterenol increased the heart rate from a mean control rate of 155 (range, 92–212) to 214 (range, 120–280) per minute. The average control

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Table 1

Effect of Isoproterenol, Atropine, and Arteriovenous Shunt on the Various Pressures and Flows

	Control						Isoproterenol					
Expt. no.	RV press. (mm Hg)*	EPA (mm Hg)*	SPA (mm Hg)*	DPA (mm Hg)*	PA flow velocity (cm/sec)	PA vol. flow (cc/sec)	RV press. (mm Hg)*	EPA (mm Hg)*	SPA (mm Hg)*	DPA (mm Hg)*	PA flow velocity (cm/sec)	PA vol. flow (cc/sec)
1	38/2	36/8	26/8	24/8	58.0	124.7	70/8	70/18	37/13	47/14	92.8	199.5
2	32/4	32/6	23/6	24/8	75.0	–†	58/4	56/12	38/10	32/10	100.0	–†
3	42/8	40/15	32/14	35/15	75.0	105.0	55/6	55/13	36/11	38/11	92.8	129.9
4	20/3	20/7	16/7	19/7	116.0	249.4	36/2	34/8	23/6	21/8	157.5	338.6
5	27/1	28/14	17/9	23/11	31.3	25.5	55/1	54/6	26/8	27/12	75.4	61.5
6	22/9	19/5	19/2	12/5	27.9	28.7	28/10	28/5	18/4	14/5	44.1	45.4
7	23/4	21/9	16/5	15/5	23.2	21.3	38/4	38/10	20/5	23/8	46.4	42.7
8	32/2	31/9	18/8	21/11	55.1	77.1	57/1	55/10	33/12	34/16	81.2	113.7
9	25/1	23/12	16/8	18/10	62.6	134.6	38/6	38/8	22/8	23/7	83.5	179.5
10	22/2	22/7	17/4	–‡	18.0	25.2	35/3	34/12	23/7	–	33.1	46.3
11	23/2	23/6	11/4	–‡	37.7	47.9	34/1	34/5	18/3	–	55.5	72.5
			Control						Atropine			
9	23/1	23/6	16/6	17/7	52.5	112.2	40/5	34/12	22/10	23/11	92.8	199.5
10	19/2	20/8	14/4	–‡	16.2	22.7	24/3	25/9	16/5	–	20.0	28.0
			Control						A–V Shunt			
11	19/1	22/7	14/3	–‡	13.0	18.2	27/4	26/10	18/6	–	19.7	27.6

*All the pressures show peak systolic over end-diastolic.

†The pulmonary arterial volume flow could not be calculated since the pulmonary arterial diameter was not measured.

‡The downstream pressure was not recorded in these two experiments.

Abbreviations: DPA = downstream pulmonary arterial pressure; EPA = end pulmonary arterial pressure; PA = pulmonary artery; RV = right ventricle; SPA = side pulmonary arterial pressure; Vol. = volume.

left atrial mean pressure of 4.3 mm Hg (range, 1-10) decreased to 3 mm Hg (range, 1–8) after administration of isoproterenol. The raw data are recorded in table 1. The data were analyzed by the paired *t*-test except when otherwise stated and the results of statistical analysis are listed in table 2. The RV (27.8 ± 2.2) and end PA (26.6 ± 2.1) systolic pressures were nearly equal ($P > 0.05$) and were higher than the side (19.2 ± 1.7) and downstream (21.1 ± 2.2) PA pressure ($P < 0.01$) in the control state. In the experimental state, the RV (45.6 ± 4.1) and end PA (45.1 ± 4.0) systolic pressures were again not significantly different ($P > 0.05$). The RV and side PA (26.5 ± 2.4) pressures were, however, significantly different ($P < 0.01$). The side PA and downstream PA pressures were not significantly different ($P > 0.05$) both in the control and in the experimental state. The peak velocity of the pulmonary arterial flow (63.3 ± 12.2) increased to a significant degree (97.9 ± 16.6) ($P < 0.01$) after administration of isoproterenol. The mean control peak systolic pressure gradient across the pulmonic valve (RV–side PA pressure difference) was 8.8 mm Hg with a range of 3 to 14 mm Hg. After isoproterenol, the mean systolic gradient across the pulmonic valve had increased to 19.1 with a range of 10 to 33 mm Hg. This change is statistically significant with P value < 0.01. When analysis of variance was performed, the transpulmonary valvar gradient remained statistically significant. The results of the effects of atropine in two dogs and femoral arteriovenous shunt in one dog are similar (table 1) but were not statistically analyzed because of the small number of experiments.

Table 2

Statistical Data of the Various Parameters

Parameter	*P* Value
RV and end PA pressure	
Control	>0.05
After isoproterenol	>0.05
RV and side PA pressure	
Control	<0.01
After isoproterenol	<0.01
Side PA and downstream PA pressure	
Control	>0.05
After isoproterenol	>0.05
PA flow velocity before and after isoproterenol	<0.01
RV and side PA pressure difference (transpulmonary valvar gradient) before and after isoproterenol	<0.01

Abbreviations: PA = pulmonary artery; RV = right ventricle.

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Both with isoproterenol and atropine, the RV and end PA pressure increased to a greater degree than the side and downstream pressure and similar results were noted in one dog in which bilateral femoral arteriovenous shunts were created.

Figures 2 to 4 show the effects of isoproterenol, atropine, and arteriovenous shunt on the various pressures and the peak flow velocity in the PA. The tracings from two representative experiments are illustrated in figures 5 and 6. The effect of these stimuli on the pulmonary valvar pressure gradients is graphically represented in fig. 7. The next illustration (fig. 8) plots the change in the pressure gradient against change in the velocity of the PA flow following administration of isoproterenol or atropine, or after creating an arteriovenous shunt. In general, the difference between the RV (or end PA) pressure and side PA pressure is proportional to the increase in velocity of the flow in the PA. When volume flow in the PA is used instead of flow velocity to plot this relationship (fig. 9), there is a wider scatter suggesting that the change in flow velocity rather than change in the volume flow is the factor determining the change in pulmonary valvar gradient.

Figure 2

Experiment No. 2. Effect of isoproterenol on the various pressures in the PA and the RV and the flow velocity in the PA. As shown in this and the following two figures the right ventricular and end PA pressure increased to a greater degree (along with the PA flow velocity) than the downstream or side PA pressure.

Figure 3

Experiment No. 9. Effect of isoproterenol and atropine on the PA and RV pressures and PA flow velocity.

Discussion

Large intracardiac left-to-right shunts have been known to produce pulmonary valvar systolic pressure gradients demonstrated at cardiac catheterization.[1–5] This has been generally attributed to "relative" or "functional" pulmonic stenosis,[2, 4–6] in that the normal-size, undilated pulmonary valve ring separates the dilated RV and PA. Shephard[3] attributed it to the changes in the elasticity in the walls of the pulmonary artery associated with postvalvar dilatation. Theoretical considerations

Figure 4

Experiment No. 10. The response of the pressures in the PA and RV and the PA flow to isoproterenol, atropine and arteriovenous shunt.

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Figure 5

Experiment No. 4. Left panel shows control pressures and flow and the right panel shows the effect of isoproterenol. Note the differences in standardization of the flow signal. In this and the following figure the tracings are lightly touched with pencil for a better reproduction. Both the experiments illustrate a greater increase in the RV and end PA pressure when compared with side and downstream pressure (only in fig. 5) in response to increase in PA flow velocity (and therefore kinetic energy) induced by administration of isoproterenol.

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Figure 6

Experiment No. 11. Effect of isoproterenol on the PA pressures and flow and the RV pressure.

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Figure 7

Effect of isoproterenol, atropine, or arteriovenous shunt on the pulmonary valve peak systolic pressure gradient (RV and side PA pressure difference).

suggest that this gradient is due rather to the differences in the expression of hydraulic energy:

Hydraulic (Fluid) Energy
= Pressure (Potential) Energy
+ Kinetic Energy
+ Gravitational Potential Energy

$$W = P + \tfrac{1}{2}\rho v^2 + \rho g h$$

Where W = fluid or hydraulic energy, P = pressure or potential energy, ρ = density of the fluid, v = velocity of the flow, g = gravitational force and h = height above the reference level.

Figure 8

The change in the peak systolic pulmonary valve gradient (RV and side PA pressure difference) is plotted against the change in the flow velocity difference in PA induced by isoproterenol, atropine, or arteriovenous shunt. This graph illustrates that these two values are proportional in that the greater the flow velocity difference, the greater is the change in pulmonary valvar pressure gradient. Closed circles = isoproterenol; closed triangles = atropine; closed square = arteriovenous shunt.

Figure 9

To fig. 8 are added the relationship of change in pulmonary valvar pressure gradient and volume flow difference in the pulmonary artery produced by the three agents. There is a wider scatter of the volume flow data compared to the flow velocity data suggesting that the flow velocity is the factor determining the pulmonary valvar pressure gradient. Closed signs = flow velocity difference; open signs = volume flow difference; cloesd and open circles = isoproterenol; closed and open triangles = atropine; closed and open squares = arteriovenous shunt.

Since the gravitational energy is equal in the RV and PA in the supine position, it may be ignored. In addition, the kinetic energy forms a greater proportion of total energy in the right side of the heart than in the left side of the heart[7, 8] and therefore, the kinetic energy component of the total fluid energy is more important in the right side of the heart. The ordinarily used catheters (end-hole or side-hole) measure only the potential energy in the PA and do not record the kinetic energy. Recorded RV pressure measures the potential energy where the kinetic energy is practically nonexistent and hence represents the total energy. Different types of catheters were used in our experiments to record the total energy, to separate its component potential and kinetic energies, and to show that the pulmonary valvar gradients are due to failure to record the kinetic energy in the PA. In the above presented experiments, isoproterenol (and atropine and A–V shunt in a few dogs) was used to increase the flow velocity (and therefore the kinetic energy). Our studies showed that when the total energy is recorded in the RV by the pressure energy and in the PA by an end pressure, which records both the pressure and kinetic energies, there is no energy gradient between these two values. If, however, the side pressure is recorded (in the PA), which records only the pressure energy, there is an

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apparent pressure gradient between the RV and PA. This gradient becomes greater as the pulmonary arterial flow velocity (kinetic energy) is increased, but if instead of the side pressure, an end pressure is recorded, there is no pressure gradient. Therefore, the pressure gradient between the RV and the side PA is due to partial transformation of the fluid energy into kinetic energy. Consequently, the pressure or potential energy gradient between these two is only artifactual in that there is no energy gradient and the ordinarily placed catheters fail to record the kinetic portion of the fluid energy. Thus this study helps to explain the pressure gradient across the pulmonary valve in large left-to-right shunt lesions.

Although these experiments are probably adequate to point out the role of kinetic energy in the production of transpulmonary valvar gradients in large left-to-right shunts, there are several factors that were not taken into consideration. The more important among these are 1) variation in the diameter of the pulmonary artery during the cardiac cycle, 2) lack of uniform flow throughout the cross-section of the pulmonary artery due to laminar and parabolic flow, and finally 3) presence of turbulent flow that might produce some loss of energy, possibly accentuated by the introduction of several catheters.

In an attempt to study the effect of pulmonary insufficiency, Ellison et al.[9] performed pulmonary valvectomy in ten dogs. The results of cardiac catheterization performed at varying intervals after surgery revealed an average gradient of 15 mm Hg (range 2–40 mm Hg) across the pulmonic valve. These dogs at autopsy showed no evidence of organic pulmonic stenosis. They attributed this gradient to increased amount RV ejection and increased velocity of flow (not measured) which results in turbulent flow with consequent loss of energy. These authors have correctly pointed out the cause of pulmonary valvar gradients to the energy kinetics but have attributed it to loss of energy. Our studies show that this gradient is due to partial transformation of fluid energy into kinetic energy rather than to loss of energy.

The exact location in the RV outflow tract at which this transformation of energy and the resulting pressure gradient occurs was not evaluated in this study. However, most of the clinical studies documenting the pressure gradient secondary to increased flow,[1, 2, 4–6] and our own observations in infants and children in whom careful pullback pressure recordings across the RV outflow tract were performed, suggest that this pressure gradient occurs at the pulmonary valvar level. Studies by Shephard,[3] and occasional cases studied in our own laboratory, showed that this gradient can also occur in the RV infundibulum.

On a theoretical basis, the downstream pressure should be lower than side pressure since some of the kinetic energy is supposed to be lost in the downstream pressure. But these two pressures did not significantly differ. This might be due to malpositioning of the downstream pressure-recording catheter in such a way that only the side pressure was recorded. The increase in the heart rate and decrease in the mean left atrial pressure are consistent with the chronotropic and ionotropic effects of isoproterenol.[10] Pulmonary arterial (side) pressure increased with isoproterenol in our experiments. Isoproterenol has been shown to produce pulmonary vasodilatation[11, 12] and to increase pulmonary blood flow.[11] The combination of these effects usually produces a fall in pulmonary pressure[13] but can also result in an increase in the pressure,[11] as in our experiments. The latter may be due to a greater increase in pulmonary flow than can be accommodated by the decrease in the pulmonary vascular resistance without an increase in PA pressure.

A wide range of PA flow velocities is recorded. especially during the control state (table 1), and this "inter-dog" variation in velocity is not associated with proportional pulmonary valvar pressure gradient. The differences in the flow velocities may be due to: 1) differences in the size of the PA, 2) different state or condition of the dog at the time of the experiment, and 3) the degree of angulation of the flow probe. It is also possible that the flow probe may not consistently measure the velocity of the flow in absolute terms but only in relative terms. However, the "intra-dog" variation in flow velocity (induced by the various stimuli) produced proportional increase in the pulmonary valve gradient. These observations support the above drawn conclusions on the role of kinetic energy in the pulmonary valvar pressure gradients.

The usefulness of isoproterenol in the evaluation of dynamic obstructions in the left ventricular outflow tract is well established.[14–16] Isoproterenol has also been recommended to be used in the evaluation of congenital heart disease in general[17, 18] and the dynamic forms of right ventricular outflow obstruction in particular.[19] As mentioned earlier, kinetic or flow energy forms a greater proportion of the total energy in the pulmonary

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circuit in contrast to the systemic circuit.[7, 8] (The pressure in the PA is one-sixth that in the aorta but the kinetic energy is very similar in magnitude in both the vessels.) If increase in flow velocity is produced by exercise or by isoproterenol, the importance of kinetic energy becomes even greater in the right side of the heart. Because of these considerations and because the ordinarily used catheters cannot record the kinetic portion of the total fluid energy, the RV outflow tract gradients[19] or pulmonary valvar gradients[17, 18] produced or augmented by isoproterenol cannot accurately indicate active obstruction to the right ventricular outflow tract or the severity of the pulmonic stenosis. If these gradients exist after accounting for the kinetic energy effect, one can draw conclusions as suggested.[17–19]

Measurement of the difference in the RV and side PA pressure, on routine cardiac catheterization, may be useful, at least theoretically, in calculating the velocity of pulmonary arterial blood flow, provided the pulmonary arterial diameter is known.

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