Pulse Wave Reflection: Can It Explain the Differences Between Systemic and Pulmonary Pressure and Flow Waves?

A Study in Dogs

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SUMMARY. We have studied the effect of changes in pulse wave reflection on the configurations of pressure and flow in systemic and pulmonary circulation. Electromagnetic flow transducers, atrial catheters, and pacing leads were implanted in 10 dogs. In four animals, the flow transducer was placed on the pulmonary artery, in another four on the ascending aorta, and in two additional dogs on both vessels. One week later, ascending aortic and/or pulmonary artery flow and pressure (catheter tip manometer) were measured under general anesthesia (Nembutal, 30 mg/kg, iv). When the pulmonary circulation was studied (six dogs), measurements were made before and during serotonin infusion (0.5-0.75 mg/min). When the systemic circulation was studied (six dogs), measurements were made before and during nitroprusside infusion (50-200 µg/min). To quantify the arterial load, we calculated pulmonary and systemic input impedances. To estimate the amount of reflection, we used a reflection index which we defined as the amplitude ratio of reflected and forward wave. Nitroprusside decreased total peripheral resistance, increased total arterial compliance, and decreased the reflection index; similarity between aortic pressure and flow wave shapes increased, and they looked more like their pulmonary counterparts. Serotonin increased pulmonary vascular resistance, decreased pulmonary arterial compliance, and increased the reflection index. Resemblance of pressure and flow waves decreased. The differences in wave shapes can thus be explained by the amount of reflection: the less reflection the more pressure and flow resemble each other. (Circ Res 51: 479-485, 1982)

The configurations of pressure and flow in pulmonary artery and ascending aorta are determined by the interaction of ventricles and pulmonary or systemic input impedance, respectively. The characteristics of the ventricles differ (Rushmer, 1964; Elzinga et al., 1980). Although a comparative study has not been done, the literature indicates that pulmonary and systemic input impedances are also different (Milnor et al., 1966; Noble et al., 1967; Reuben et al., 1971; Westerhof et al., 1973; Pouleur et al., 1978; Hopkins et al., 1979, 1980). Consequently, resemblance of pulmonary and aortic flow waves is small; the same is true for pulmonary and aortic pressure waves (Fig. 1). Figure 1 also shows that in the pulmonary circulation, pressure and flow waves look alike, but that this is not so in the systemic bed.

Rushmer (1964) has pointed out that the differences between aortic and pulmonary flow waves tend to disappear when left ventricular function deteriorates or when the right ventricle faces an abnormally high load for a long time.

Measured pressure and flow waves consist of forward and reflected waves (Westerhof et al., 1972; van den Bos et al., 1976). The forward wave (equal in shape for pressure and flow, since characteristic impedance is real and frequency independent; see McDonald, 1974) runs into the system and is reflected from distributed sites. Under all physiological and a large variety of pathological conditions, the reflected pressure wave is in phase with the forward wave: addition of forward and reflected wave results in a measured wave which is larger than the forward wave. Consequently, more the forward wave is reflected, the less measured flow resembles measured pressure. The differences in wave shapes in the two systems and the observation that pulmonary pressure and flow waves look more alike than their systemic counterparts could be partly explained if reflection in the pulmonary bed were less. In the circulation under control conditions, the major source of reflection is the periphery, which was described by von Kries as early as 1890. Changes in peripheral resistance could thus alter the amount of reflection.

We have studied systemic and pulmonary flow and
pressure waves in anesthetized dogs. We decreased systemic and increased pulmonary resistance, since we would expect that the wave shapes in both beds might then become more alike. Similarly, vasodilation in the systemic circulation might increase resemblance of pressure and flow waves, and vasoconstriction in the pulmonary bed might decrease the normally existing resemblance.

Methods

We have used 10 male, mongrel dogs (20.5-43.0 kg). One week before the experiments transducers, catheters (left atrium) and pacemaker wires (right atrium and ventricle) were implanted under general anesthesia and sterile conditions, through a left thoracotomy in the 4th intercostal space. In four dogs, the electromagnetic flow transducer (constructed in our workshop) was put on the ascending aorta (group A)—in another four, on the pulmonary artery (group B)—while, in two additional animals, we have put flow transducers on both vessels (group C).

In the actual experiments, the dogs were anesthetized with Nembutal (30 mg/kg, iv), intubated, and connected to a respirator (Pulmomat Drager; mixture of 20% O\textsubscript{2} and 80% N\textsubscript{2}O). Nembutal was added in the course of the experiment if necessary. We produced total atrioventricular block (Randall et al., 1981).

Catheter tip manometers (Millar Mikro-TIP PL 350, 6F or 7F) were introduced into the aorta (group A) from a femoral artery or into the pulmonary artery (group B) from a femoral or jugular vein. In the two dogs in group C, tip manometers were put into aorta as well as pulmonary artery. The tips of the catheters were positioned at the upper rim of the flow transducer. Because of catheter "whip," this was not always possible in the pulmonary artery. When flow and pressure had to be analyzed for input impedance, the two waves were synchronized by the computer program (superposition of back flow and pressure incisura).

Pacing catheters were put into the right atrium and ventricle if necessary. The heart was paced at a series of rates by a pacemaker triggered from the right atrial ECG. This signal, converted to a TTL pulse, was first passed through a digitimer (Devices D 4030). This arrangement provided synchronization of atrial and ventricular contractions. The digitimer could be programmed to trigger the pacemaker at every first, second, third, or fourth atrial ECG, etc., until the ventricles beat at their own rate. For heart rates higher than the atrial rate, we either used two pacemakers triggered from the same source or we paced the ventricles only.

We have recorded on paper (Elema EMT 81) and analog tape (SE 7000): ECG II, left atrial pressure (Statham 23 dB) in all dogs; ascending aortic pressure and flow (Transflow systems 601) in group A and C; pulmonary artery pressure and flow in group B and C.

Protocol

Group A

After control recordings had been made, systemic resistance was decreased with nitroprusside (50-200 \mu g/min).

Group B

After control recordings had been made pulmonary resistance was increased with serotonin (0.5-0.75 mg/min).

Group C

In these dogs, the procedure was performed in sequence, i.e., serotonin was given followed by nitroprusside or vice versa.

When a steady state had been reached during the infusions, recordings were again made. Recordings during control and intervention were made at a series of heart rates ranging from the intrinsic ventricular rate to the maximum rate at which the heart could be paced (about 3 Hz) without causing mechanical alternans. At each heart rate in groups B and C, the respirator was disconnected for about 20 seconds.

Calculations and Analyses

Pressure and flow signals of series of heart beats (from 5 to 15) in the steady state at each pacing rate were passed through a filter (3 dB point at 80 Hz; –12 dB/octave). Beats were either sampled from periods when the respirator had been disconnected or from a complete respiratory cycle. The data was then converted from analog to digital at a rate of 200/sec. We performed Fourier analyses on individual beats. When the amplitude of pressure harmonics was less than 1% and of flow harmonics less than 2% of the first harmonic, these harmonics were considered to be contaminated by noise and discarded. Impedance was calculated by dividing pressure and flow moduli and subtracting their phase angles. The impedance for each heart rate was calculated by averaging the results of individual beats.
performed this procedure for several heart rates to improve resolution of the impedance plot. A computer program subsequently placed the moduli and phase data in classes of frequencies (the class interval was equal to the lowest determined frequency) and determined the mean of each class. A line drawn through these means gives the average impedance.

Systemic peripheral resistance was calculated as the ratio of mean aortic pressure and cardiac output. Characteristic impedance was defined as the average of the impedance moduli above 4 Hz. For pulmonary peripheral resistance, mean left atrial pressure was subtracted from mean pulmonary artery pressure, and this difference was divided by cardiac output.

Total arterial compliance was calculated by fitting the three-element windkessel (Westerhof et al., 1971) to the impedance by means of a least squares method.

Forward and backward (= reflected) waves were calculated from measured flow and pressure in the pulmonary artery and ascending aorta using the characteristic impedance of these two arteries (Westerhof et al., 1978).

To quantitate reflections, we used a reflection index determined frequency) and determined the mean of each class. A line drawn through these means gives the average impedance.

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FIGURE 3. Average input impedances in aorta (A) and pulmonary bed (B) for the six dogs before (drawn line) and during nitroprusside or serotonin, respectively (dashed line). Units as in Figure 2.

considerable variability between dogs. Figure 4 shows the average systemic and pulmonary impedance and their standard errors for all animals.

The changes in the impedance during nitroprusside and serotonin mainly concern the mean term (R_s in Table 1) and very low frequencies. Nitroprusside decreased the moduli and phase angles of low frequency harmonics so that flow and pressure were less out of phase. During serotonin infusion, the moduli and phase angles of low frequency harmonics increased: flow and pressure were less in phase.

From the input impedance, we have also calculated characteristic impedance and total arterial compliance (Table 2). Nitroprusside had a tendency to increase slightly the systemic characteristic impedance. Systemic compliance increased considerably (213%, range 69-420) with nitroprusside. During serotonin infusion, pulmonary compliance fell by 66% (range 42-86).

We have also calculated the forward and reflected wave for one heart beat before and during nitroprusside and serotonin infusion (Fig. 5). After nitroprusside, the reflected wave decreases; the incisura in the pressure pulse moves to a lower level and pressure begins to resemble flow. In the pulmonary circulation before serotonin, the reflected wave is small and the measured pressure and flow wave look alike. After serotonin, the amplitude of the reflected wave has increased considerably and the similarity between flow and pressure becomes less. The increased reflection causes the incisura in the pressure pulse to move to a higher level. Figure 6 gives the reflection index in the systemic and pulmonary bed before and during the interventions. Nitroprusside significantly decreases the index in the systemic bed, while serotonin significantly increases it in the pulmonary circulation.

Discussion

The arterial bed can be described by resistance, compliance and inertance (Westerhof et al., 1977). Changes in any of these parameters alter the characteristics of the system so that pressure and flow will also change. Generation of pressure and flow waves in a set of tubes causes reflection of these waves (Westerhof et al., 1978). The amount of reflection is expressed in the reflection coefficient, also a property of the system. Consequently, changes in the parameters that describe the system influence the reflection coefficient. Since the configurations of pressure and flow are to a major extent determined by the amount of reflection any change in the parameters will alter the wave shapes.
Reflections occur at distributed sites, but the periphery supposedly plays a major role. Welkowitz (1977) argues, on the basis of his analog model of the arterial tree, that normally little reflection occurs in the peripheral bed. Westerhof et al. (1972) clearly show that changes in peripheral resistance alter the reflection coefficient. We have therefore used such changes to alter pulsewave reflection: serotonin in the pulmonary and nitroprusside in the systemic circulation. Our general hemodynamic results with these drugs are comparable to those in the literature (Reuben et al., 1971; Gundel et al., 1981). Our study shows that differences in the amount of reflection can explain the dissimilarity in the pattern of flow and pressure in systemic and pulmonary circulation: the less reflection the greater the resemblance of flow and pressure waves.

When we infused serotonin, the amount of reflection in the pulmonary bed increased significantly. A larger reflected wave must now be added to the forward pressure wave and subtracted from the forward flow wave. Thus, flow and pressure wave lose similarity. On the other hand, when we infused nitro-
prusside, the amount of reflection in the systemic circulation decreased significantly. A smaller reflected wave has to be added to the forward pressure and subtracted from the forward flow wave. Pressure and flow wave become more alike.

We have expressed the amount of reflection as the reflection index (Fig. 6): the ratio between the maximum amplitude of reflected and forward wave. Usually, reflection is quantitatively expressed by means of the reflection coefficient (Westerhof et al., 1972). This is a complex parameter in the mathematical sense with a modulus and phase which are both functions of frequency and therefore not easy to interpret. Our reflection index, on the other hand, gives one number for each heart beat. Even though this number is not easily obtained (one has to calculate forward and reflected waves), it is easy to understand. Calculation of this index clearly illustrates the changes in the amount of reflection occurring with serotonin and nitroprusside.

Under control conditions, the amount of reflection is less in the pulmonary than in the systemic circulation. Serotonin increases pulmonary reflection (as was predicted by Pollack et al., 1968), while nitroprusside decreases systemic reflection. The amount of reflection in the systemic bed is now comparable to that in the pulmonary circulation during control. The same holds for the constricted pulmonary bed and the systemic circulation under control conditions. In both situations, the pulmonary pressure begins to resemble aortic pressure and the pulmonary flow begins to resemble aortic flow. We should realize, however, that the resemblance is restricted to wave shapes. That the wave shapes are not exactly alike is in part due to the differences in pump function of right and left heart; configurations of flow and pressure are determined by the characteristics of the ventricle as well as the arterial impedance.

Peripheral resistance is only part of the load faced by the heart. Milnor (1975) has indicated that this load can best be described by the input impedance. Calculation of input impedance is only permitted for linear systems. Although the linear behavior of the entire arterial tree has been challenged (Milnor and Nichols, 1975; Milnor and Bertram, 1978), several reports show that linearity is a reasonable assumption (Dick et al., 1966; Noble et al., 1967; Li et al., 1981). We have measured the input impedances before and during the interventions (Fig. 4). These impedances mainly differ in their mean term (= peripheral resistance) and the characteristic impedance. The mean term is considerably higher and the characteristic impedance slightly higher in the systemic bed. The changes in the impedance caused by the drugs mainly concerned the mean term and the low frequency range. This can best be seen in Figure 7 which shows the data of Figure 4 replotted on an expanded frequency axis. These changes in the low frequency ranges indicate a change of compliance. When we calculated arterial compliance, nitroprusside increased this parameter while serotonin decreased it (Table 2). This need not be a direct effect of the drugs since compliance is also dependent on pressure (Bergel, 1961). The large changes in compliance (Table 2) causes small changes in the impedance plot (Figs. 4 and 7). Moreover, major changes in general hemodynamics caused by serotonin and nitroprusside were only reflected in the impedance plot by changes in the mean term and the lowest frequencies.

Gundel et al. (1981) have expressed doubts about the use of the impedance concept for clinical work. They suggest that the concept is appropriate principally for physiological studies—Impedance on the one hand and the combination of peripheral resistance and compliance on the other are both ways to describe the arterial system. However, in our opinion the combination of peripheral resistance and compliance is easier to understand and therefore more practical as a description of the load to the heart.

We wish to acknowledge Dr. J.C.J. van Rysen for his help during the experiments, C. Pieksma for typing the manuscript, A. van der Vos for his help with the computer programming and J. Meijer and H. Verbeek for the illustrations.

Dr. O.S. Randall was on sabbatical leave from the University of Michigan, Department of Cardiology, Ann Arbor, Michigan.

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Received November 23, 1981; accepted for publication July 22, 1982.

References


Rushmer RF (1964) Initial ventricular impulse: A potential key to cardiac evaluation. Circulation 29: 268-283


INDEX TERMS: Pulmonary artery • Aorta • Reflections • Impedance
Pulse wave reflection: can it explain the differences between systemic and pulmonary pressure and flow waves? A study in dogs.
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Circ Res. 1982;51:479-485
doi: 10.1161/01.RES.51.4.479

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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