Velocity Profiles in the Main Pulmonary Artery of Dogs and Man, Measured with a Thin-Film Resistance Anemometer

By S. R. Reuben, M.B., J. P. Swadling, and G. de J. Lee, M.A., M.D.

ABSTRACT

Instantaneous velocity of pulmonary artery blood was measured using a thin-film resistance anemometer mounted on a hypodermic needle. Studies were performed, at thoracotomy, in five dogs and five patients. Instantaneous blood velocity was recorded at several sites across the transverse axis of the main pulmonary artery to determine the shape of the velocity profile.

We found the velocity profile, in both man and dog, to be approximately flat. In dog, the mean velocity, normalized to that at the center line, ranged from 1.18 (SD ± 0.03) to 0.81 (SD ± 0.27). In man, mean velocity of pulmonary artery blood, normalized in a similar fashion, ranged from 1.08 (SD ± 0.03) to 0.84 (SD ± 0.07), while normalized peak systolic velocity ranged from 1.04 (SD ± 0.07) to 0.95 (SD ± 0.16). The results, in dogs, suggest that there is little asymmetry of the velocity profile and therefore these have been taken as evidence that the use of cuff electromagnetic flowmeters on the pulmonary artery of dogs is not subject to significant inaccuracies associated with a nonsymmetrical flow profile. The finding of a relatively flat velocity profile in patients, moreover, will make it easier for catheter-tip flowmeters to measure bulk velocity in the pulmonary artery.

ADDITIONAL KEY WORDS

axi-symmetrical Fourier analysis electromagnetic impedance pulmonary hypertension

Recently Schultz and his colleagues (1) described a thin-film resistance anemometer designed to measure instantaneous fluid velocity. The thin-film bead, when mounted on a hypodermic needle, was used by these workers to map the distribution of blood velocity across the transverse axis of the aorta in both dogs and man. They subsequently extended their work by measuring the velocity of aortic blood flow with a catheter-tip, thin-film resistance anemometer during routine cardiac catheterization. The results of this work have been reported in greater detail by Tunstall Pedoe (2).

Several groups of workers have stressed the need for caution in the interpretation of flow measurements using cuff electromagnetic flowmeters. Wyatt (3), Goldman (4) and Bergel and Cesnner (5) have particularly emphasized the need for the blood flow in a vessel to have an axi-symmetrical velocity profile, before the volume flow rate in that vessel can be related to the output of the electromagnetic flowmeter.

However, at present the shape of the velocity profile in the pulmonary artery is not known. This knowledge would be valuable in evaluating the use of cuff electromagnetic flow transducers on the pulmonary arteries of dogs and of catheter-tip, velocity-measuring devices in the pulmonary arteries of patients. We therefore measured the shape of the velocity profile in the main pulmonary artery of both dogs and man by applying Schultz's thin-film techniques.

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Methods

1. VELOCITY-MEASURING TRANSDUCER

The thin film consisted of a glass bead onto which were painted several coats of a suspension of platinum and silver particles in an organic fluid and furnace baked at 640°C (6). In operation the film formed one arm of a self-balancing Wheatstone bridge (Fig. 1) and was normally maintained at approximately 5°C above blood temperature by setting $R_2$ at $1.01 \times R_1$. The transfer of heat from the film to the flowing blood cooled the film, lowered its resistance, and initiated an output from the bridge to the high gain feedback power amplifier, which restored the bridge to balance. The frequency response of the whole system was limited by the gain and bandwidth of the feedback amplifier and was flat to approximately 200 cps, since the thermal diffusion time of the film was of the order of $10^{-2}$ seconds (6). The bead was mounted on the bevelled surface of a hypodermic needle, whose end was bent into a right angle (Fig. 2). The transducer leads were soldered to the film and brought out through the lumen of the needle.

The thin-film probe was calibrated before and after each study, over a wide range of velocities, in a tank of distilled water rotated on a gramophone turn-table. Details of the linearity and dynamic calibrations have been discussed by Schultz (2), who showed that there was no significant difference in the calibration of the probe in blood or in water. After introduction of the needle into the pulmonary artery, the resistance of the film, at central blood temperature, was determined, and $R_2$ (Fig. 1) was then

![Feedback bridge to achieve operation at constant resistance of thin-film probe.](image1)

![Needle velocity probe and half-cuff support.](image2)
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set at 1.01 times this resistance to achieve an overheat of 5°C. The zero velocity signal was obtained by bringing the thin-film element into contact with the back wall of the vessel. Heat transfer, due to conduction and convection, occurs at zero velocity. When the thin-film element is in contact with the posterior wall, heat transfer occurs only by conduction and it is believed that there is no significant difference between the thermal conductivity of blood and that of the vessel wall (Schultz, personal communication). Hence the thin-film signal during diastole, when velocity is zero, is identical to the posterior wall signal. Since the heat transfer characteristics of the probe were nonlinear, the recorded signal was corrected with a commercially available linearizer.1

2. DOG STUDIES

Five mongrel dogs, weighing between 15 and 22 kg, were anesthetized with intravenous chloralose-sulphate (56 and 500 mg/kg) followed by intramuscular morphine sulphate (1 mg/kg). A carotid artery and jugular vein were cannulated and the animals ventilated through a cuffed endotracheal tube connected to a positive-pressure respiratory pump.2 End-expiratory pressure was kept at 5 cm H2O. The minute volume was adjusted according to the results of serial arterial blood gas estimations.

At thoracotomy, the thin-film needle was introduced into the pulmonary artery through a purse-string suture in the anterior wall of the vessel, approximately 2 to 2.5 cm distal to the pulmonary valve. The probe was supported by a perspex half-cuff applied to the vessel, and by a centimeter scale extending vertically above the cuff (Fig. 2). The pulmonary artery was traversed by pushing the probe across the transverse axis of the vessel in 5-mm steps. At each step between 20 and 40 velocity pulses were recorded, along with the electrocardiogram.

3. CLINICAL STUDIES

Five patients undergoing left thoracotomy for either pneumonectomy, lobectomy or ductus ligation were studied. The nature and purpose of the procedure was explained to all the patients, who readily gave consent to the study. Two patients had bronchial carcinomata; the third a lung abscess; the fourth patient had a chronic tuberculous cavity, and the fifth patient had a patent ductus arteriosus. The patients received pethidine 75 mg and atropine 0.6 mg and then anesthesia was induced with thiopentone (250 to 500 mg, intravenously). A Robertshaw double-

humm endobronchial tube was introduced and the patients ventilated by a positive-pressure respiratory pump, with an end-expiratory pressure of 15 to 20 cm H2O. Anesthesia was maintained by ventilation with a 50% nitrous oxide: 50% oxygen gas mixture. A left thoracotomy was performed. The probe was inserted into the pulmonary artery through a purse-string suture in the anterior wall of the vessel. As in the animal studies, it was supported by a half-cuff and the vessel was traversed in steps, the velocity being recorded at five stations across the axis. The traverses were performed while the lungs were inflated and the direction of the needle was checked by the position of the flag on the shaft (Fig. 2).

4. ANALYSIS

The pulsatile velocity data and electrocardiogram were recorded on magnetic tape,3 at a speed of 78 inches/sec. The tape was later replayed through an integrator4 to obtain the mean velocity at each station of the traverse. Both this and the instantaneous velocity was recorded on a 12-channel photographic recorder,5 at a paper speed of 100 mm/sec.

The average mean velocity (V) at each station was calculated for 20 to 40 recorded beats. This was normalized by dividing it by the mean velocity for the center of the vessel (V0). Similarly, each radial location was normalized by dividing it by the radius of the vessel, the center position being designated zero. The instantaneous diameter of the vessel was obtained from the difference between the anterior and posterior wall readings on the centimeter scale of the supporting collar (Fig. 2), after allowing 1.0 mm for the thickness of the needle. In addition, the peak systolic velocity (V) at each radial location was normalized in relation to the center-line peak systolic velocity (V0). This was useful because there were greater respiratory variations in the patients' data than in the dogs.

Results

Figure 3 and Table 1 show the combined results for the five studies on dogs; the velocity profile is approximately flat, both for the mean velocities (V/V0) and the peak systolic velocities (V/V0), which were found to be close to the center-line velocity at all stations measured.

Figure 4 shows a traverse of the pulmonary

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1Dia Electronik, Denmark.
2Palmer Pump Ltd., London.
4Ampex Corp., USA.
5Devices Sales Ltd., England.
artery in one patient who was having a pneumonectomy for carcinoma of the bronchus. This figure demonstrates some reduction in peak systolic velocity towards the posterior wall of the pulmonary artery. Table 2 and Figure 5 show the combined results from the five patients, demonstrating that the velocity profile, in the main pulmonary artery of patients, is approximately flat. However, there is some skewness, particularly of mean velocity (Fig. 5) with the lower velocities towards the posterior (dorsal) wall.

**Discussion**

Although velocity was only measured in one radial axis, we believe that these studies in dogs provide direct evidence of a virtually flat velocity profile in the main pulmonary artery...
An actual trace of the pulmonary artery velocity in a patient with bronchial carcinoma. Velocities were recorded at each station with identical instrumental gain. The vessel was traversed anteroposteriorly.

Velocity distribution across the transverse axis of the pulmonary artery in five patients. The velocities were normalized with respect to the center-line velocity (see text, section 4). $U/U_{CL}$ represents normalized mean velocity; $V/U_{CL}$ represents normalized peak systolic velocity. The data points represent the mean ± 1 se of the combined clinical results. Abbreviations as in Figure 3.
and suggest that there is relatively little asymmetry. This finding supports the use of cuff electromagnetic flowmeters in dogs for measuring pulsatile volumetric flow rate. More confidence can thus be placed on correlations between flowmeter output and volumetric flow rate, and also on calibration of such flowmeters in vivo (7).

In the clinical studies, the pulmonary artery velocity profile was approximately flat, with some skewness of the profile so that lower velocities occurred towards the posterior wall of the pulmonary artery. This deviation does not appear significant and the results suggest that catheter-tip measurements of blood velocity, obtained during cardiac catheterization, may be representative of the cross-sectional blood velocity in the human pulmonary artery.

Measurements of instantaneous pulmonary arterial velocity, simultaneously with the measurement of pressure, in patients with valvular heart disease, with a thin-film resistance anemometer mounted on a standard cardiac catheter may extend our knowledge of ventricular performance in such diseases, by allowing us to calculate total ventricular stroke work (8). Also, by performing Fourier analysis of simultaneously recorded velocity and pressure, the pulmonary arterial input impedance may be calculated (9).

An illustration of the value of such calculations has been recently presented by Milnor and his colleagues (10). They determined the pulmonary arterial impedance in patients after computing pulmonary artery flow by the differential pressure method (11). They showed that the impedance pattern found in these patients resembled that found in dogs (7) and claimed that such impedance determinations could yield valuable information about the state of the pulmonary vascular bed in the pulmonary hypertension of mitral valve disease.

Catheter-tip velocity and pressure measurements in valvular heart disease is, therefore, an alternative and perhaps simpler method for the determination of vascular impedance than the differential pressure method.
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TABLE 2

Pulmonary Artery Velocity Profiles in Man

<table>
<thead>
<tr>
<th>Station</th>
<th>Anterior wall</th>
<th>Center line</th>
<th>Posterior wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>U/U CL</td>
<td>1.00</td>
<td>1.00</td>
<td>0.96</td>
</tr>
<tr>
<td>SD</td>
<td>0.004</td>
<td>0.029</td>
<td>0.044</td>
</tr>
<tr>
<td>V/V CL</td>
<td>0.070</td>
<td>0.033</td>
<td>0.065</td>
</tr>
</tbody>
</table>

Abbreviations: U/U CL = normalized mean velocity; V/V CL = normalized peak systolic velocity.

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References


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