Measurement of Instantaneous Blood Flow Velocity and Pressure in Conscious Man with a Catheter-Tip Velocity Probe

By Ivor T. Gabe, M.D., James H. Gault, M.D., John Ross, Jr., M.D., Dean T. Mason, M.D., Christopher J. Mills, B. Sc., John P. Schillingford, M.D., and Eugene Braunwald, M.D.

SUMMARY
Twenty-three patients were investigated during diagnostic right and left cardiac catheterization with an electromagnetic catheter-tip velocity probe. The catheter contained a pressure lumen for simultaneous measurements of intravascular pressure. Average peak and mean blood velocities were 66 and 11 cm/sec in the ascending aorta, 57 and 10 cm/sec in the pulmonary artery, 28 and 12 cm/sec in the superior vena cava, and 26 and 13 cm/sec in the inferior vena cava. The velocity pattern in the ascending aorta was similar to that obtained by other methods. Positioning of the catheter in the ascending aorta required care; in one patient with aortic stenosis the recorded blood velocity pattern was unsatisfactory. In the pulmonary artery flicking of the catheter often produced artifacts in the records. The effect of deep respiration on blood velocity in the ascending aorta and pulmonary artery was studied. In the ascending aorta the highest velocities and stroke volumes were achieved during late expiration while in the pulmonary artery blood velocity and stroke volume were greatest in inspiration. In nine patients the cardiac outputs calculated from the product of mean velocity and radio logically measured cross-sectional area of the ascending aorta or pulmonary artery were compared with cardiac outputs determined by the indicator-dilution method; the correlation coefficient was 0.73. There were no complications, and the probe proved reliable.

Additional Indexing Words: Cardiac output Pulmonary artery Aorta

While the measurement of intravascular and intracardiac pressures in man can now be easily accomplished, it has not been possible, until recently, to determine

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Summary of Data on 25 Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Hosp. no.</th>
<th>Diagnosis</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>HR</th>
<th>Systemic arterial S/D</th>
<th>Mean in atrium</th>
<th>Pulmonary arterial S/D</th>
<th>Cardiac output (L/min)</th>
</tr>
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<tr>
<td>1. A.R.</td>
<td>07-69-67</td>
<td>AF, MI, MS</td>
<td>F</td>
<td>60</td>
<td>78</td>
<td>160/58</td>
<td>8</td>
<td>40/20</td>
<td>3.9</td>
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<td>2. V.Ha.</td>
<td>07-69-08</td>
<td>AF, AS, MI, MS</td>
<td>F</td>
<td>54</td>
<td>65</td>
<td>130/60</td>
<td>6</td>
<td>28/8</td>
<td>4.1</td>
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<td>3. H.H.</td>
<td>07-62-50</td>
<td>LV+</td>
<td>M</td>
<td>46</td>
<td>60</td>
<td>82/58</td>
<td>3</td>
<td>60/25</td>
<td>4.0</td>
</tr>
<tr>
<td>4. T.F.</td>
<td>07-65-98</td>
<td>Mild IHSS</td>
<td>M</td>
<td>36</td>
<td>50</td>
<td>138/78</td>
<td>6</td>
<td>36/14</td>
<td>4.3</td>
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<td>5. P.G.</td>
<td>07-72-03</td>
<td>AS+</td>
<td>M</td>
<td>59</td>
<td>78</td>
<td>130/75</td>
<td>2</td>
<td>30/8</td>
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<tr>
<td>6. M.M.</td>
<td>06-80-45</td>
<td>Pericarditis; Hodgkins d.</td>
<td>M</td>
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<td>77</td>
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<td>24/7</td>
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<tr>
<td>7. L.P.</td>
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<td>MI, MS</td>
<td>F</td>
<td>44</td>
<td>80</td>
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<td>16</td>
<td>105/40</td>
<td>—</td>
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<td>8. L.W.</td>
<td>05-54-68</td>
<td>AF, MS, MI, AI</td>
<td>M</td>
<td>38</td>
<td>70</td>
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<td>90/44</td>
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<td>48</td>
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<td>22/10</td>
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<td>10. M.B.</td>
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<td>F</td>
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<td>97</td>
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<td>20/8</td>
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<td>11. M.K.</td>
<td>07-73-53</td>
<td>AF, MS, MI</td>
<td>F</td>
<td>32</td>
<td>74</td>
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<td>Isch. ht. d.</td>
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<td>24/10</td>
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<td>55/30</td>
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<tr>
<td>14. J.M.</td>
<td>07-00-38</td>
<td>Pericardial effusion</td>
<td>M</td>
<td>24</td>
<td>111</td>
<td>120/80—18</td>
<td>18</td>
<td>36/20</td>
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<tr>
<td>15. P.T.</td>
<td>07-74-32</td>
<td>AI</td>
<td>M</td>
<td>40</td>
<td>67</td>
<td>140/40</td>
<td>5</td>
<td>34/16</td>
<td>5.3</td>
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<td>17. J.C.</td>
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<td>M</td>
<td>43</td>
<td>66</td>
<td>122/64</td>
<td>3</td>
<td>23/12</td>
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<tr>
<td>19. H.W.</td>
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<td>M</td>
<td>26</td>
<td>76</td>
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<td>58/30</td>
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<td>20. C.D.</td>
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<td>21. S.S.</td>
<td>07-70-72</td>
<td>Idio. edema of legs</td>
<td>F</td>
<td>26</td>
<td>82</td>
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<td>23/8</td>
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<td>22. V.H.</td>
<td>06-18-26</td>
<td>AF, MI, MS, AI</td>
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<td>48</td>
<td>80</td>
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<td>50/18</td>
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<tr>
<td>23. M.S.</td>
<td>07-76-11</td>
<td>Functional chest</td>
<td>F</td>
<td>25</td>
<td>68</td>
<td>150/77</td>
<td>4</td>
<td>21/10</td>
<td>4.0</td>
</tr>
</tbody>
</table>

*Pk = peak velocity; minimum = minimum velocity in cycle.
†Aortic or pulmonary diameter not estimated.
‡US = unsatisfactory record; NR = no record taken at site; NE = vessel not entered.
§Innominate artery.
**Descending aorta at T-8.
††Descending aorta at T-12.
‡‡Descending aorta at L-1.

Abbreviations of diagnoses: AF = atrial fibrillation; AI = aortic insufficiency; AS = aortic stenosis; CS stim = carotid sinus stimulator; IHSS = idiopathic hypertrophic subaortic stenosis; Isch. ht. d. = ischemic heart disease; LV = left ventricle; MI = mitral insufficiency; MS = mitral stenosis; TI = tricuspid insufficiency; ++ = severe.

Intravascular velocity probe, the transducer.

exposure animals, on instantaneous blood velocity practical. The development of an electromagnetic velocity probe, mounted at the end of a cardiac catheter, has now made intravascular measurement of blood flow velocity practical. This probe, which has been described previously,\(^6,7\) has been evaluated in model systems and in dogs and has been used in a study of blood flow velocity in the venae cavae in normal human subjects.\(^8\) This report will describe the technic employed in the measurement of the velocity of blood flow and the resting pattern of instantaneous velocity observed in the human systemic arteries, the pulmonary artery, and the venae cavae. A preliminary report has appeared elsewhere.\(^9\)

\(^{6}\) GABE ET AL.

Table 1

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## Methods

Twenty-three patients (age range, 21 to 60 years) were investigated at the time of diagnostic right or left cardiac catheterization carried out at the National Heart Institute, Bethesda, Maryland. The patients were premedicated with 100 mg of sodium pentobarbital, and they were conscious during the studies; no anticoagulants were given. Ten of the 23 patients had valvular heart disease, 6 had ischemic heart disease, two had pericarditis, one had mild idiopathic hypertrophic subaortic stenosis, one bronchitis, one idiopathic edema, one left ventricular hypertrophy, and one functional chest pain (table 1).

### The Catheter-Tip Electromagnetic Velocity Probe

In brief, this instrument consists of a coil wound within the tip of a nylon tube (O.D. 0.25 cm, equivalent in size to a no. 8 cardiac catheter). The coil is energized by a 1,000 Hz sine wave and the electromotive force induced by the movement of blood through the magnetic field in the neighborhood of the catheter tip is detected at two electrodes near the coil. The output of the associated electronic apparatus is recorded, with other signals, on an 8-channel photographic recorder.

Steady-flow tests in an in vitro system have shown that the output of the probe is a linear function of blood velocity and the sensitivity in vitro is identical to that in vivo. The transducer was calibrated by applying a known electrical signal to the input; the velocity equivalent to this signal had previously been determined in vitro. The frequency response of the velocity probe is determined to a large extent by the output filter used. During the studies reported here the amplitude response was ± 1% to 10 Hz, with a linear phase shift with frequency of 5.94°/Hz (equivalent to a delay of 16.5 msec).

The catheter-tip electromagnetic velocity probe was sterilized by autoclaving at 121 C at 15

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*Now manufactured by S. E. Laboratories, Feltham, Middlesex, England.

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**Table 1**

<table>
<thead>
<tr>
<th>Asc. Ao.</th>
<th>Other artery</th>
<th>Pulmonary arteries</th>
<th>SVC</th>
<th>IVC</th>
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<tr>
<td></td>
<td>Pk* Mean</td>
<td>Pk* Mean</td>
<td>Pk* Minimum* Mean</td>
<td>Pk* Minimum* Mean</td>
</tr>
<tr>
<td>NE 33</td>
<td>7 NE ‡</td>
<td>US ‡</td>
<td>NR ‡</td>
<td>11 0 6</td>
</tr>
<tr>
<td>NE 44</td>
<td>6 §</td>
<td>41 NE</td>
<td>16 -3 5</td>
<td>18 -1 10</td>
</tr>
<tr>
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<td>44 7</td>
<td>38 18 24</td>
<td>30 1 18</td>
</tr>
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<td>38 13</td>
<td>26 0 13</td>
<td>28 -1 13</td>
</tr>
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<td>120 10</td>
<td>29 10 20</td>
<td>18 5 11</td>
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<td>40 8</td>
<td>25 12 15</td>
<td>22 2 6</td>
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<tr>
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<td>65 10</td>
<td>32 0 11</td>
<td>14 -4 7</td>
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<tr>
<td>US 59</td>
<td>9 62 7 † †</td>
<td>41 8</td>
<td>34 16 4</td>
<td>25 1 12</td>
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<tr>
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<td>25 -1 10</td>
<td>22 3 11</td>
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<td>8 62 9 §</td>
<td>45 9</td>
<td>40 15 30</td>
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<tr>
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<td>12 50 8 † †</td>
<td>52 8</td>
<td>31 10 22</td>
<td>24 -1 11</td>
</tr>
<tr>
<td>NE 76</td>
<td>11 55 8 §</td>
<td>US</td>
<td>18 0 8</td>
<td>43 2 1 24</td>
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<tr>
<td>63</td>
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<td>NE</td>
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<tr>
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<td>61 9</td>
<td>26 4 15</td>
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<td>8 48 11 † †</td>
<td>48 11</td>
<td>19 1 10</td>
<td>17 0 8</td>
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pounds per square inch, for 20 min. For ease of manipulation the catheter was bent at about 30°, approximately 3 cm behind the tip, by placing it in a polypropylene former during the autoclaving process. Since the catheter was made of nylon, the curvature introduced in this manner remained constant unless changed during subsequent autoclaving. Before the catheter was inserted into a patient, a series of electrical tests was carried out to ensure that the probe was intact and safe. The criteria satisfied were as follows: (1) the coil resistance was approximately 55 ohms, (2) the resistance between the coil and the ground and detecting electrodes exceeded 100 mho, and (3) with the catheter tip in a beaker of saline, the resistance between the detecting electrodes was approximately 1 kohm. These precautions were taken to avoid the hazards of accidental electrical stimulation of the myocardium.

With the probe tip stationary in saline, the apparatus was adjusted initially so that zero output was recorded. Insertion of the catheter into an antecubital vein or the brachial artery, with no flow near the point of introduction, produced no significant output and this zero velocity signal was subsequently used for reference in recordings made in the venae cavae and in the descending aorta. In the ascending aorta and in the main pulmonary artery the signal in mid and late diastole was taken as zero velocity. The signal from flow to the coronary arteries was within the noise level of the instrument. It was also found possible to obtain valid levels for zero blood flow velocity by wedging the catheter tip in a branch of the pulmonary artery, and these corresponded well with the base line determined by the methods just described.

The catheter-tip velocity probe contains a no. 1 nylon catheter (OD, 0.93 mm; ID, 0.74 mm), the orifice of which lies at the tip of the probe. Pressure measurements were made through this catheter, using a Statham P23Db transducer. The zero pressure reference level was taken to be the mid-chest position. Following the procedure in each patient the frequency response of the catheter-transducer system was estimated by the pressure-transient method.10 Under these conditions the damped resonant frequency was between 28 and 48 Hz with relative damping of 0.19 to 0.23 Hz.

The brachial artery pressure was measured through a Cournand needle, using a Statham P23Db strain gauge. Cardiac output was measured by the indicator-dilution technique, using a cuvette densitometer and indocyanine dye. In nine patients, the cross-sectional area of the midportion of the ascending aorta or main pulmonary artery was determined from biplane cineangio-

graphic measurements of vessel diameter in the frontal and lateral projections, taking an average value between diastolic and systolic measurements. Cardiac output was then estimated from the average aortic or pulmonary arterial blood flow velocity and the cross-sectional area, and this value was compared with the one derived from an indicator-dilution curve obtained immediately prior to the angiogram. Respiratory movements were recorded by measuring the pressure within a compliant air-filled tube encircling the chest.

Results

Measurements of velocity of blood flow were made in the venae cavae in 18 patients, the pulmonary artery in 16, the ascending aorta in 12, the descending aorta in 10, and in other arteries in 14. No measurements were made in the arterial system when there was no indication for diagnostic left heart catheterization. In one patient (C.C.) the catheter was too large to be introduced into the arm veins. In three patients it was found possible to pass the catheter into the descending but not into the ascending aorta.

No complications resulted from the use of this catheter, and there was no evidence of blood coagulation around the catheter.

The pressure measurements and peak and mean blood velocities in the arterial system, pulmonary artery, and venae cavae are given in table 1. Average peak and mean velocities were 66 and 11 cm/sec in the ascending aorta, 57 and 10 cm/sec in the pulmonary artery, 28 and 12 cm/sec in the superior vena cava, and 26 and 13 cm/sec in the inferior vena cava.

Aortic Blood Flow Velocity

An example of a recording obtained from the ascending aorta in patient H. W., with moderate mitral stenosis, is shown in figure 1. The wave form of velocity is similar to that obtained by a conventional electromagnetic probe.4 With the catheter-tip probe in this position the most direct way of determining zero velocity is by assuming that no significant flow occurs in mid-to-late diastole. When the current energizing the magnet was turned off, no significant change from the end-diastolic level was observed. Similar findings were noted throughout this study, but sufficient
Figure 1

Blood velocity and pressure in the ascending aorta (Asc. Ao.) of a patient with mitral stenosis, recorded with the catheter-tip electromagnetic velocity probe. Zero velocity has been taken from the signal level in mid and late diastole. Note that the signal level does not change significantly when the magnet current is switched off.

Figure 2

Blood velocity and pressure in the descending aorta at the levels of T-5, T-7, T-10, L-1, L-3 and in the iliac artery in a patient with ischemic heart disease.

experience to recommend this method is not yet available.

The positioning of the catheter tip in the ascending aorta to produce a consistent wave form always required care. If the catheter tip was placed behind an aortic cusp, the velocity signal was small and variable, and withdrawal of the catheter tip by 3 or 4 cm was associated with an increase in the signal and the appearance of the expected velocity wave form. In one patient with severe aortic stenosis (P. G.) the wave form in the ascending aorta was extremely noisy, the dominant velocity being toward the ventricle.
in systole; it is likely, in this case, that the tip was near the aortic wall and in a stream of eddies set up by the jet emerging from the aortic valve. Although measurements of blood flow velocity were not possible in the ascending aorta in this patient, satisfactory records were obtained in the descending aorta.

Figure 2 shows the velocity wave forms in M. B., a patient with ischemic heart disease, in the descending aorta at levels T-5, T-7, T-10, L-1, and L-3 and in the right iliac artery. The reversed velocity following the systolic phase became more prominent in the lower portion of the aorta, particularly at the level of L-3 (fig. 2E). In the iliac artery this effect was less marked and the mean forward velocity was sufficiently high to prevent actual reversal of flow. The diameter of the iliac artery was not measured, but the reduction in the cross-sectional area produced by the presence of the catheter in this vessel may have been significant. It is unlikely, however, that such a
reduction would have materially changed the velocity pattern.

**Pulmonary Artery Blood Flow Velocity**

In general, the records of blood flow velocity in the pulmonary artery were less satisfactory than in the ascending aorta. This difficulty was related to the motion of the catheter as it passed through the right side of the heart. The tracing reproduced in figure 3, from M. K., a patient with mitral valve disease, shows little such artifact, and the pattern of blood flow velocity exhibits only instrumental noise in diastole. More commonly the tracing in diastole exhibited the fluctuations shown in figure 4, obtained from S. S., a patient with pedal edema but without hemodynamic evidence of heart disease. In three of the 19 patients in whom the catheter probe entered the pulmonary artery, marked longitudinal flicking of the catheter with each cardiac cycle observed on fluoroscopy produced greatly distorted blood flow velocity patterns; in the remaining cases the records were sufficiently stable for meaningful analysis.

**Effect of Deep Respiration**

The effects of deep respiration on the velocity of blood flow in the ascending aorta were determined in five patients. A typical tracing, reproduced in figure 5, obtained from a patient with ischemic heart disease exhibits cyclic variations in peak velocity. The highest velocities (57 cm/sec) were achieved during expiration, while the lowest (40 cm/sec) occurred at the end of inspiration. In the pulmonary artery, the changes differed in their relation to the respiratory cycle, as

![Figure 5](image1)

*Effects of deep respiration on the blood velocity and pressure in the ascending aorta (Asc. Ao.) of a patient with coronary artery disease. In this and in subsequent figures inspiration is indicated by a downward deflection of the pneumogram (Pneum.). The highest peak velocities occur during expiration and the lowest during inspiration.*

![Figure 6](image2)

*Blood velocity and pressure in the pulmonary artery (PA) of a patient with ischemic heart disease. Maximum peak velocity occurs during early inspiration, while brachial artery (BA) pressure rises to a maximum about 2% sec later.*

*Circulation, Volume XL, November 1969*
Figure 7

*Calculated values of relative right ventricular stroke volume, pulmonary artery (PA) systolic and pulse pressures, and brachial artery (BA) systolic and pulse pressures during three deep respirations in a patient with ischemic heart disease.*

illustrated in figure 6. Here the highest velocities (100 cm/sec) were achieved during inspiration and the lowest (50 cm/sec) at the end of expiration.

Since, as indicated below, the area beneath the systolic velocity pulse in the pulmonary artery and ascending aorta may be considered to be approximately proportional to the stroke volumes of the right and left ventricles, relative changes in stroke volume over the course of several respiratory cycles were calculated. Figure 7 shows the estimated changes in right ventricular stroke volume relative to the first beat, the brachial artery systolic and pulse pressures, and the pulmonary artery systolic and pulse pressures in F. T., a patient with ischemic heart disease. The highest stroke volumes occurred during, but prior to the end of inspiration and fell during early expiration. The fluctuations in stroke volume were approximately ± 25% of the end-expiratory value. Small, but similar changes in the pulmonary artery pulse pressure were also observed. Figure 8 illustrates the estimated changes in left ventricular stroke volume and the aortic systolic and pulse pressures over three respiratory cycles in patient C. C., whose tracings are shown in figure 5. The greatest stroke volumes occurred during early expiration, the volumes decreasing during inspiration. The variations in stroke volumes which occurred were ± 20% of the mean over the three respiratory cycles.

**Blood Velocity in the Venae Cavae**

Typical blood velocity patterns observed in the venae cavae are illustrated in figure 9. The phasic changes are more marked in the inferior vena cava. The velocity was near zero at the time of atrial systole, rising to a maximum during ventricular systole, before decreasing, and then rising to a second, smaller peak in diastole. At more rapid heart rates, the second rise in diastole often disappeared (fig. 10).

**Cardiac Output Comparisons**

In the nine patients in whom the diameter of the aorta or pulmonary artery was determined angiographically, the cardiac output was calculated from the product of the mean
BLOOD FLOW VELOCITY AND PRESSURE

Figure 9
Blood velocity and pressure in the superior (SVC) and inferior (IVC) venae cavae of a patient with ischemic heart disease. Phasic velocity changes are more evident in the IVC. Blood velocity is near zero during atrial systole, rises to a maximum during ventricular systole, then falls and rises to a second smaller peak in diastole. Heart rate 65/min.

Discussion
The catheter-tip device used in this study is essentially a velocity-sensitive instrument, responding linearly to the velocity of the conducting fluid in the neighborhood of the electrodes. The voltage developed at the electrodes reflects the average velocity of the sleeve of fluid between the probe and a distance of approximately one probe radius from its surface. In principle then, the device is sensitive to the velocity profile of the blood within a vessel. Thus, if the tip of the probe were at the center of a tube carrying steady flow with a parabolic velocity profile, the velocity that it would sense would be approximately twice the mean velocity. In practice, the presence of the probe in the vessel disturbs the velocity profile of blood flow in a favorable manner, since the probe diameter constitutes a significant fraction of the diameter of the vessel. If the ratio of probe to vessel diameter is 0.1, then the ratio of peak to mean velocity will not be 2:1 but, in the case of established annular flow, 1.57:1, and the velocity of flow near the catheter will be less than the peak velocity. Two other factors will also tend to flatten the velocity measured blood velocity, the cross-sectional area of the vessel, and the heart rate. The results were compared with the cardiac outputs determined by the indicator-dilution method and are shown in figure 11. The correlation coefficient was 0.73 (P = 0.005).

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profile: (1) the pulsatile nature of flow in medium and large vessels, and (2) the fact that the common sites of measurement are well within the inlet length, that is, at a point in the vessel where the full profile has not yet had time to develop. Therefore, there is some theoretical as well as experimental support for the finding in this study that the signal output is relatively insensitive to the position of the catheter tip. However, when the catheter tip is close to the wall of a vessel, the velocity signal diminishes markedly, for electrical as well as hydraulic reasons. Under these circumstances rotation of the catheter usually restores the full velocity wave form.

A more significant problem than the position of the probe within a vessel is introduced by longitudinal motion of the catheter, which is usually the cause of disturbances in recordings of blood velocity in the pulmonary artery. These artifacts do not alter the estimate of the mean velocity provided there is no net change in the position of the catheter within the vessel during a cardiac cycle.

The velocity probe used in this study is similar in principle to that tested by Bond and Barefoot in dogs, although that device did not allow the measurement of pressure at the catheter tip. Two different kinds of catheter flowmeter have been described by Kolin and his colleagues. One of these requires the flow to be directed transversely through an aperture in the catheter, which must be correctly positioned at a branch of the arterial system. The other is intended for the measurement

![Figure 10](image)

Blood velocity and pressure in the superior vena cava (SVC) in a patient with ischemic heart disease. The heart rate is 98/min and, in contrast to the pattern in figure 9 no second forward velocity wave is evident in diastole.

![Figure 11](image)

The cardiac output in nine patients, calculated from the mean velocity of blood in the aorta and pulmonary artery and the radiologically measured vessel area, compared with the cardiac output determined by the indicator-dilution technic. The line of identity is shown. The correlation coefficient is 0.73 (P = 0.005). Thirteen comparisons were made in nine patients.
of flow in the aorta and requires that the end of the catheter be brought to lie, by means of a wire system, across the lumen of the aorta. Both of these designs have been tested in animals, but practical considerations may make their use in man difficult at present.

The pattern of the aortic velocity waves observed in this study is comparable to those recorded in man by cuff-type electromagnetic flow meters\(^4\) and by the pressure gradient method;\(^14-16\) since all records are calibrated in terms of velocity, a direct transformation into volume flow through an artery requires the determination of the vessel diameter and an assumption about the velocity profile. Assuming that the velocity profile is flat, for the reasons given above, the cardiac output was calculated in nine patients from the product of mean velocity and the measured aortic or pulmonary artery cross-sectional area and compared with that estimated by an indicator-dilution curve, recorded either immediately before or after the velocity record.

The comparison (fig. 11) shows a significant correlation between these two sets of measurements, but with considerable scatter about the line of identity. In considering the possible errors involved, it must be noted that if the probe is situated near the arterial wall, the velocity signal will be reduced and the calculated cardiac output will be falsely depressed. Errors are also associated with the radiologic estimation of the cross-sectional area of the artery; it is the diameter of the vessel which is actually measured and any error in this measurement will be magnified, since the cross-sectional area of the vessel is proportional to the square of the diameter. The error introduced by the assumption that the vessel diameter does not change over the course of a cardiac cycle is probably a relatively small one for the aorta. Greenfield and Patel have determined that the change in cross-sectional area in the ascending aorta in 10 patients averaged only 5.5% of control;\(^17\) in the pulmonary artery the change in cross-sectional area is more than twice this value;\(^18\) and we should therefore expect the error in the estimation of the stroke volume of the right ventricle to be greater than that of the left ventricle. Lastly, the technic used for the comparison, the indicator-dilution method, is itself associated with errors, probably of the order of at least ± 10%.

Thus, the use of the electromagnetic velocity probe for the measurement of cardiac output is difficult and in most instances unnecessary, for other methods are simpler. The particular advantage of the velocity probe is that it provides instantaneous values of velocity from which directional beat-to-beat changes in stroke volume may be estimated. If the changes in aortic or pulmonary artery diameter are small, then the stroke volume may be assumed to be proportional to the area beneath each individual velocity pulse, and relative changes in stroke volume can thus be derived without any calibration procedure. If absolute values for flow and stroke volume are needed, they may be calculated by equating the mean signal amplitude (from planimetry or an electrical mean measured from a zero base line) with the cardiac output determined independently by the indicator-dilution or Fick methods. Illustrations of this approach to the measurement of relative stroke volume are shown in figures 7 and 8. The estimated phasic changes in right and left ventricular stroke volumes with deep respiration are very similar to those found by Charlier,\(^19\) who used cuff-type electromagnetic flowmeters on the aorta and pulmonary artery in dogs. It is likely that the later rise in left compared to right ventricular stroke volume results from the delay in transmission of the augmented right ventricular stroke volume through the pulmonary vascular bed and into the left side of the heart.\(^20, 21\)

No complications occurred from the use of the velocity probe in this study. The instrument proved reliable, although in the patient with severe aortic stenosis, no satisfactory record could be obtained in the ascending aorta, and in others motion artifacts were common in the pulmonary artery. On the basis of these and related studies, the probe appears...
to be capable of contributing useful information to the investigation of the cardiovascular system of man.

References
Measurement of Instantaneous Blood Flow Velocity and Pressure in Conscious Man with a Catheter-Tip Velocity Probe

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