Velocity of Blood Flow in Normal Human Venae Cavae

By Lewis Wexler, M.D., Derek H. Bergel, M.B., Ph.D., Ivor T. Gabe, M.D., Geoffrey S. Makin, Ch.M., and Christopher J. Mills, B.Sc.

ABSTRACT
The velocity of flow and pressure in the venae cavae of four normal conscious subjects was studied. Velocity was measured with a catheter-tip electromagnetic transducer. The effects of respiration, Valsalva and Müller maneuvers, coughing, and exercise were studied. Caval blood velocities during breath holding showed marked cardiac pulsations, being maximal at the time of ventricular systole and minimal or reversed at atrial systole. Peak velocities during ventricular systole ranged from 30 to 45 cm/sec in the inferior, and from 10 to 35 cm/sec in the superior, vena cava. A second diastolic forward flow velocity ranged from 36 to 76% of the systolic peak. During inspiration, velocity transiently increased. Reduction of flow velocity in abdominal breathing and the Müller maneuver is consistent with the formation of a local area of inferior vena caval collapse at the diaphragm. During the Valsalva maneuver, abrupt reduction in caval flow was seen that persisted throughout the strain. There was immediate overshoot when the strain was released. Coughing produced a reduction of flow velocity with backflow in the superior vena cava. In leg exercise, inferior caval flow velocity rose immediately, and it remained high during recovery. Marked respiratory velocity variations with inspiratory increases occurred during and after exercise.

ADDITIONAL KEY WORDS
- catheter-tip electromagnetic velocity transducer
- exercise
- deep breathing
- collapsible tubes
- coughing
- Valsalva maneuver
- Müller maneuver

To understand the mechanisms involved in the return of blood to the heart accurate measurements of vena caval blood flow are necessary. While data have been obtained from animal experiments involving, at least, surgical exposure of the vessels in order to place flowmeters, recordings have not previously been obtained from conscious, cooperative human subjects.

Brecher (1) has shown that caval blood flow in the dog has two forward flow phases, and Tafur and Guntheroth (2) confirmed this finding with an ultrasonic flow transducer. They believed that the concept that the flow of venous blood into the heart is affected by events within the heart itself explains the pulsatile nature of caval blood flow. Evidence that left atrial filling is also influenced by intracardiac activity has also been provided (3, 4).

Respiratory effects on venous return are known to exist, although their nature and mechanisms remain uncertain (1, 4-7). It is generally assumed that inspiration increases, and expiration decreases, venous return. The changes in caval blood flow during and following exercise and due to various respiratory efforts such as the Valsalva and Müller maneuvers, deep thoracic or abdominal breathing, and coughing are less well known and poorly understood.

The present report summarizes the results of our investigations of vena caval blood flow in healthy unsedated human volunteers.
Methods

APPARATUS

The instrument used for the measurement of blood velocity was a catheter-tip electromagnetic transducer that has been described elsewhere (8). It measures blood velocity past its tip with equal sensitivity in either direction. The frequency response (relative amplitude 95% at 15 Hz with 4-msec delay) is adequate for the present purpose, and the transducer may be calibrated in vivo. To convert the recordings into volume flow rates the cross-sectional area of the vessel must be known and some assumption made about the form of the velocity profile. Pressure was measured through a no. 1 nylon catheter incorporated into the velocity transducer with its orifice at the extreme end of the transducer. When used with the Statham P23Gb pressure transducer, a resonant frequency greater than 45 Hz and relative damping of about 0.2 could be achieved. Before each experiment, the manometer frequency response of the saline-filled transducer-catheter system was obtained by the pressure-transient method (9), and the apparatus was flushed free of entrapped air until the above resonant frequency was attained.

In two subjects, right atrial pressure was measured with a PE60 radiopaque catheter, 105 cm long (Portex), introduced into an antecubital vein by the Seldinger method and positioned under radiological control. In the other two subjects, intrathoracic pressure was estimated with a balloon-tipped esophageal catheter.

The two pressures, velocity of venous flow, and the electrocardiogram (precordial leads) were recorded with a four-channel direct-writing oscillograph (Devices M4). At intervals, high speed records were also taken on an ultraviolet galvanometer recorder (S.E. Laboratories, type 2005). Before each experiment the phonocardiogram and ECG were simultaneously recorded by a phonocardiographic amplifier and photographic recorder (Cambridge Instrument Co.).

Pressure calibrations from a 20-cm column of water were obtained frequently during each session. Zero pressure level was adjusted to lie 5 cm below the level of the sternal angle. To calibrate the velocity transducer a standard electrical signal was applied to its input. This had previously been related to flow velocities measured in vitro, and the sensitivity determined in vitro has been shown to match that obtained in vivo (8). Zero flow velocity signal was obtained with the velocity transducer in saline before and after the experiment, and this was checked against the signal which lay within an arm vein close to the point of insertion.

PROCEDURE

Recordings were obtained on four healthy, unanesthetized male volunteers aged 29 to 39 (Table 1). Three of the volunteers (L.W., D.B., I.G.) are co-authors of this report and the fourth (J.M.) is a respiratory physiologist. Experiments began within an hour of a light meal and lasted up to 3 hours, during which the subject lay quietly on a radiographic table. A small incision was made over an antecubital vein and the tip of the velocity transducer was inserted into it. At this time, a series of routine safety checks was carried out to ensure the absence of current leakage between the transducer coil windings and the other leads. At the same time, the zero flow velocity level was determined. The transducer was then passed under fluoroscopic control through the right atrium and into the inferior vena cava (IVC) and positioned so that its tip lay at the level of the 11th and 12th thoracic intervertebral disc. This corresponds to a position in the intrahepatic portion of the IVC between the entry points of the hepatic and renal veins.

A standard sequence of measurements was then carried out with intervals of 1 to 5 minutes between each: control records during quiet breathing and apnea, and with intervals of 1 to 5 minutes between each: control records during quiet breathing and apnea at midinspiratory level, deep
CAVAL VELOCITY IN MAN

Simultaneous records of ECG, right atrial pressure, inferior vena caval (IVC) pressure and velocity. An upward deflection indicates velocity of flow toward the heart. Forward flow velocity waves are seen during ventricular systole and diastole. The pattern is similar to that in the SVC. Note the change in time scale during the record. The vertical line to the right of the record has been drawn in to show the timing of the R-wave peak.

Abdominal and thoracic respiration, Valsalva (to a mouth pressure of about 40 mm Hg for 20 to 30 seconds) and Müller maneuvers, and coughing.

At this point, the velocity transducer was withdrawn into the superior vena cava (SVC) with its tip lying between the entry point of the azygos vein and the right atrium. The whole sequence was then repeated. Following this, the transducer was replaced in the IVC and records were taken before, during, and after a period of vigorous pedaling on a bicycle ergometer mounted at the end of the table.

Results

The most striking observation was that the velocity of venous flow is remarkably pulsatile. Figures 1 and 2 show typical records taken from the SVC and IVC, respectively, during apnea. The velocity record from the SVC (Fig. 1) is inverted because a positive flow velocity is given for flow passing from the tip of the catheter toward the shaft and consequently centripetal flow velocity (i.e., toward the heart) will be recorded as negative flow in the SVC.

The caval flow velocity waves (Figs. 1 and 2) are similar to the right atrial pressure waves in their general features, but they are in antiphase. That is, a rise in right atrial pressure generally accompanies a reduction in centripetal flow velocity and vice versa. The pattern of flow velocity was constant in all four subjects. The values of resting peak systolic, peak diastolic, and mean velocities are given in Table 1; systole and diastole refer to the ventricular cycle.

### Table 1: Caval Flow Velocities in Rest and Exercise

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Peak systolic velocity in IVC* (cm/sec)</th>
<th>Peak diastolic velocity in IVC* (cm/sec)</th>
<th>Diastolic peak/systolic peak (IVC)</th>
<th>IVC mean velocity (cm/sec)</th>
<th>Peak systolic velocity in SVC* (cm/sec)</th>
<th>Peak diastolic velocity in SVC* (cm/sec)</th>
<th>Diastolic peak/systolic peak (SVC)</th>
<th>SVC mean velocity (cm/sec)</th>
<th>Peak velocity during exercise (IVC)** (cm/sec)</th>
<th>Peak velocity during exercise (SVC)</th>
<th>/peak control velocity (IVC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.B.</td>
<td>35</td>
<td>183</td>
<td>80</td>
<td>45.3</td>
<td>34.4</td>
<td>69%</td>
<td>20.0</td>
<td>16.4</td>
<td>12.4</td>
<td>76%</td>
<td>7.9</td>
<td>105.7</td>
<td>15.0</td>
<td><strong>106%</strong></td>
</tr>
<tr>
<td>I.G.</td>
<td>39</td>
<td>185</td>
<td>85</td>
<td>44.3</td>
<td>20.0</td>
<td>60%</td>
<td>11.8</td>
<td>32.5</td>
<td>15.0</td>
<td>54%</td>
<td>9.6</td>
<td>87.8</td>
<td>18.9</td>
<td><strong>107%</strong></td>
</tr>
<tr>
<td>L.W.</td>
<td>29</td>
<td>174</td>
<td>78</td>
<td>31.5</td>
<td>19.9</td>
<td>76%</td>
<td>11.8</td>
<td>11.4</td>
<td>6.7</td>
<td>40%</td>
<td>6.5</td>
<td>57.0</td>
<td>11.2</td>
<td><strong>55%</strong></td>
</tr>
<tr>
<td>J.M.</td>
<td>30</td>
<td>169</td>
<td>67</td>
<td>35.8</td>
<td>13.0</td>
<td>76%</td>
<td>16.1</td>
<td>18.9</td>
<td>6.7</td>
<td>46%</td>
<td>6.5</td>
<td>101.2</td>
<td>11.2</td>
<td><strong>51%</strong></td>
</tr>
</tbody>
</table>

IVC = inferior, and SVC = superior, vena cava.

*Average of 10 cardiac cycles during shallow breathing or apnea.
**Exercise at 130 to 160 watt-seconds for 150 seconds (D.B.), 90 seconds (L.W.), and 165 seconds (J.M.).
When compared with the ECG and the previously recorded phonocardiogram, the relation of the venous flow velocity waves to cardiac events can be better appreciated. Figure 3 is a composite diagram constructed to show the average pattern of the flow of blood in the cavae in relation to right heart pressures. Also shown diagrammatically are the ECG and the phonocardiogram to serve as timing references for the velocity changes. Incorporated into the figure are typical recordings of pulmonary artery pressure and velocity, together with the time integrals of inflow and outflow (accumulated volume). In each cardiac cycle there were two main oscillations of the velocity of flow in the veins. At the time of atrial contraction, centripetal flow velocity in both venae cavae was sharply reduced and flow was halted or even reversed momentarily. At the end of atrial systole, flow velocity began to rise toward its mean level. A sudden acceleration of centripetal velocity of flow occurred with the onset of ventricular contraction, and the greatest velocities of flow occurred during ventricular systole. The velocity then declined, but a second increase in the velocity of forward flow occurred in early ventricular diastole. For the remainder of the cardiac cycle caval blood velocity declined slowly, to be sharply reduced again with the onset of the next atrial systole. With alterations in heart rate, slight modifications of this general pattern were seen. At very slow rates, a small third centripetal wave of flow in mid-diastole was sometimes seen. At rapid rates, diastole shortened and the early diastolic forward flow velocity wave became much reduced in size or even disappeared as velocity declined sharply from its peak at ventricular systole to its minimal value in atrial systole.

**EFFECTS OF RESPIRATION**

During quiet breathing, inspiration tended to increase, and expiration to decrease, the velocity of venous flow. This effect was also especially obvious after exercise (Fig. 4), when very large changes in velocity of flow were recorded. On these changes cardiac oscillations were superimposed.

The effects of forced breathing on the pattern of the velocity of flow in the IVC differed greatly in abdominal and thoracic respiration. During thoracic inspiration (Fig. 5), intra-abdominal and intrathoracic pressures fell together. At this time, velocity of flow toward the heart increased transiently, falling...
Caval Velocity in Man

353

Atrial Pressure cm. H2O IV C. Velocity cm/sec.

EXERCISE

CONTROL

FIGURE 4

ECG, right atrial pressure, and IVC flow velocity in a normal subject before, during, and after leg exercise. At the start of exercise there is an initial rapid rise and fall in velocity as the subject takes a deep inspiration and tightens the abdominal muscles. This is followed by a gradual elevation of mean velocity as venous return increases. Immediately following the period of exercise (bottom, left) large respiratory velocity variations are seen. The maximum velocities occur during inspiration. With the tachycardia and shortened diastolic period the second diastolic flow wave disappears. At 45 seconds (bottom, center) and 2 minutes later (bottom, right) similar respiratory patterns persist, although the mean flow velocity gradually falls.

Again during expiration. In general, the mean velocity over one respiratory cycle was greater than that during suspended respiration.

During abdominal inspiration, although the pressure gradient from IVC to right atrium increased, the velocity of IVC flow fell rather abruptly to around zero (Fig. 6). The IVC pressure trace showed sudden swings with each breath, which might be attributed to the movement of the diaphragm past the point of pressure measurement. When right atrial pressure was at its lowest, the velocity of IVC flow was almost zero and the normal cardiac pulsations were not seen. With the onset of expiration, IVC pressures rose abruptly and velocity increased and showed normal cardiac pulsations once more. Again, a breath appeared to result in an increase in average flow velocity into the chest.

In the SVC, forced thoracic respirations produced a marked but transient increase in flow velocity. Abdominal breathing produced no alteration of flow velocity during inspiration; there was a slight and transient increase in the velocity of flow at the moment of expiration.

Valsalva and Müller Maneuvers

During a Valsalva maneuver (Fig. 7),...
ECG, right atrial pressure, IVC pressure, and IVC flow velocity to show the effect of deep thoracic respirations. With each inspiration, atrial and caval pressures fall, causing a gradient favorable to atrial filling, and IVC flow velocity rises. Mean flow velocity falls to slightly less than control levels with expiration. The net result is an increase in venous return. Note that at peak inspiration, when IVC pressure is at its lowest, large velocity oscillations are sometimes seen, possibly indicating changes in vein caliber.

ECG, right atrial pressure, IVC pressure, and IVC flow velocity during forced abdominal respiration. With each breath a sudden swing in pressure is seen, possibly due to the catheter tip being incorporated into the intrathoracic IVC as the diaphragm descends. At the time of lowest right atrial pressure the venous flow velocity is zero and the normal cardiac oscillations are not seen.
Caval velocity in man

The velocity of IVC flow fell abruptly to near zero and remained at that level for the duration of the straining phase. On occasion, there was a tendency for the velocity to increase somewhat at the end of the period of strain, though not to control levels. Immediately after release of the strain, there was a brief period of increased velocity with exaggerated cardiac pulsations lasting for a few seconds. A significant feature of velocity late in the Valsalva maneuver was the persistence of small oscillations with a changed relationship to cardiac events. The maximal velocity of flow then occurred just before the inscription of the QRS complex, when velocity was normally at a minimum (Fig. 2). Similar variations were seen during forced inspiration against a closed glottis (Müller maneuver) (Fig. 8). After an initial large increase, the mean velocity was slightly higher than the resting value, and the timing of oscillations was reversed. However, toward the end of this rather difficult maneuver, the IVC pressure tended to fall until a point was reached when velocity of flow increased greatly and showed normal pulsations about an increased mean. Changes in the velocity of flow with both maneuvers were less marked in the SVC. The mean velocity was reduced during the Valsalva maneuver and the phasic pattern of flow was altered. In one subject, the amplitude of the oscillations in flow velocity and the mean velocity were reduced during the Müller maneuver, but the timing of the pulsations was normal. In another subject, a fall in velocity was accompanied by a loss of the normal phasic oscillations. Intrathoracic pressure fell to a greater extent in the second subject than the first.

ECG, right atrial pressure, IVC pressure, and IVC velocity during a Valsalva maneuver (40 mm Hg mouth pressure for 20 seconds). There is an abrupt fall in venous flow at the onset of the maneuver, and the timing of the cardiac oscillations is reversed. Flow returns immediately after the pressure is released. Note the rather large respiratory flow changes before straining, presumably due to overbreathing at this time. The vertical line has been drawn in to show the time of the R-wave peak.
FIGURE 8
Changes in pressures and IVC velocity during a Müller maneuver. There is a short increase in velocity at the beginning of the effort, but thereafter it continues at a level slightly greater than normal, and the timing of the velocity oscillations is reversed. Toward the end of the maneuver, the pressure in the IVC falls, and that in the right atrium rises. At this time the normal flow oscillations are seen again. At the end of the period, mean flow is increased and falls as the mean IVC pressure falls. The vertical line indicates the timing of the R-wave peak.

EFFECTS OF COUGHING
The effects of coughing are rather difficult to describe because neither the timing nor the force of the cough was controlled. In the SVC, a single cough produced a definite transient reduction in velocity of flow with unequivocal backflow; variations in velocity were the inverse of the intrathoracic pressure changes. In the IVC, the pattern was less clear; the velocity was reduced at the time of the cough, though apparently not reversed. During a series of several coughs separated by intervals of 2 to 3 seconds, velocity of flow between coughs was reduced and cardiac pulsations were absent.

EXERCISE
Velocity of flow was determined in the IVC only. Velocity increased immediately after exercise began and continued throughout the period of exertion. The amplitude of the velocity waves associated with cardiac events diminished slightly, and toward the end of the period, very marked changes in the velocity of flow appeared and were synchronous with respiration (Fig. 4). At each inspiration IVC pressure rose and velocity of flow increased. The peak velocities of flow reached in these subjects during exercise and the percentage increase over control values are given in Table 1. When exercise stopped, the raised velocity of flow was maintained for several minutes, as were the exaggerated changes with respiration. The bicycle used was not calibrated, but by comparison with an electrically damped ergometer the level of work performed in these studies was about 130 to 160 watt-sec.

OTHER OBSERVATIONS
Several observations of the velocity of superior caval flow made in other subjects are relevant to the above findings. In one patient with tricuspid insufficiency, the usual
rise in the velocity of forward flow during ventricular systole was replaced by a marked backflow. In another patient with atrial fibrillation, forward acceleration was seen during ventricular systole but was not preceded by the diminished velocity of flow that would accompany a normal atrial contraction. We have also found that records taken from the lower IVC in several patients show relatively steady flow with diminished or absent pulsations.

Discussion

We did not think it justifiable in these studies to perform angiography to determine the size of the vessels studied, and therefore we cannot convert our measurements of blood velocity into volume flow rates. However, it appears that under resting conditions only small (about 2%) changes in caval diameter are seen in phase with pressure (2). Thus, the waveform of caval blood flow should be the same as that for blood velocity.

On general grounds, it would be expected that the velocity profile in the venae cavae would not be parabolic. The presence of tributaries and the catheter itself will tend to flatten the velocity profile. In addition, the velocity profile in pulsatile flow at heart rate frequency and in tubes of this size should be essentially flat (9). If the dimensions of the veins in these subjects are similar to those measured from cross sections in preserved cadavers (IVC, 3.83 cm²; SVC, 3.02 cm² [10]), then from the average values for mean velocity of flow in Table 1, the total venous return would be 5.6 liters/min, which is a reasonable figure.

Mills and Shillingford have shown (8) that the velocity transducer used here responds linearly to velocity of flow past its tip and that its frequency response is quite adequate for present purposes. The transducer itself has a cross-sectional area of 7 mm², and thus when placed in a vessel 3 cm in diameter, it will occupy 13% of the cross-sectional area. They also showed that the output of the transducer is much less sensitive to the relation between the vessel axis and the transducer axis than might have been expected from a simple cosine relationship. The probable explanation for this is that flow lines tend to follow the wall of the transducer.

The waveforms of velocity of flow recorded here are, as far as we know, the first ones to be recorded from the normal human venae cavae. The patterns of flow agree very closely with those previously reported for dogs (1, 2, 11). The main features of the flow waves are the acceleration of velocity of flow toward the heart during ventricular systole and the marked slowing during atrial systole. That these pulsations are the result of events within the heart is supported by findings in the two patients mentioned above whom we have studied during diagnostic catheterization.

Flow in veins, as in other collapsible tubes, depends both on the pressure gradient along the vessel and on the transmural pressure difference (12-14). When the downstream intravascular pressure is less than the extravascular pressure, a localized segment of the vessel at the downstream end undergoes partial collapse. Flow will then be governed by the difference between the upstream intravascular and the extravascular pressures. Alterations in downstream intravascular pressures will not affect flow. As the extravascular pressure is raised, the length of the partially collapsed segment increases toward the upstream end. When the upstream pressure is exceeded, flow ceases altogether and the vessel is collapsed for its entire length (12, 15). Thus, in such a system, an increase in flow will result when the upstream pressure is raised, but lowering the downstream pressure can only increase flow so long as downstream pressure remains greater than extravascular pressure.

We believe that this simple model describes the superior vena cava. For the inferior vena cava, however, it is necessary to consider a collapsible vessel surrounded by two extravascular chambers separated by the diaphragm. In this model, which has been studied by Doppman et al. (15), collapse occurs...
at the downstream end of either chamber, depending on local transmural pressure differences. (It is often assumed that the collapsible tube is not distensible and is either open or closed. This is unlikely to be strictly true for veins, but the simplification will not alter the general conclusions.) Such behavior is best illustrated in these studies by the results of the Müller maneuver.

The Müller maneuver raised intraperitoneal pressure and lowered intrathoracic pressure, producing a gradient favorable to flow toward the chest. In our studies, however, the increase in velocity of IVC flow was transitory and was followed by return to a level only slightly above control. Holt (12) postulated that the vena cava would collapse during a Müller maneuver, and we assume that lowered pressure in the IVC led to a localized collapse of the vessel just below the diaphragm. Such a collapse has been demonstrated radiologically in ascites, a condition in which the pressure relationships are similar to the Müller maneuver (15). The pressures beyond such a constriction will not influence the flow (14), and in fact, cardiac oscillations were recorded, but their timing in relation to cardiac events was reversed, suggesting that the mechanism responsible for them had altered. Later on in the maneuver, the pressure gradient between the right atrium and the IVC decreased and yet flow increased until, as the gradient became quite small, it reached a maximum and the normal cardiac pulsations reappeared. This indicates that constriction had ceased and flow was once more governed by normal intravascular pressure gradients.

It might be expected that the flow in the SVC during a Müller maneuver would be changed less than that in the IVC. The extravascular pressure is unlikely to have risen around the extrathoracic tributaries of the SVC, the pressure drop at the thoracic inlet should be less than that in the IVC at the diaphragm, and localized constriction might not develop. In fact, the response was variable depending on the magnitude of fall in intrathoracic pressure. In one subject who had a marked drop in pressure in the SVC, flow velocity did fall considerably and normal phasic oscillations were lost. This suggests a collapse of the veins just outside of the thorax. When intrathoracic pressure fell to a lesser extent, there was only a transient increase in flow toward zero with gradual return to slightly above the previous mean. A possible explanation of these findings is the transient development of localized constriction at the junction of the extra- and intrathoracic portions of the vena cava followed by an increase in cross-sectional area of the SVC that balanced the increase in flow. Flow velocity would, therefore, change very little and would not reflect any change in actual flow that might occur.

The changes during abdominal breathing appear to be similar to but less marked than those produced by the Müller maneuver. Those produced by thoracic breathing appeared less complex and can be explained by a moderate decrease in intrathoracic pressure, not great enough to cause collapse of the IVC below the diaphragm. Thus moderate suction within the chest increased flow velocity, but excessive suction caused venous collapse and reduced flow (12). During the Valsalva maneuver, both intrathoracic and intraperitoneal pressures were greatly elevated over those in the IVC and right atrium. IVC flow almost immediately ceased. Abdominal cavagrams taken by Doppman (16) during the Valsalva maneuver did not show a localized constriction or significant collapse of the cava, but rather, there was slowing of the caval stream with diversion of flow into the paralumbarazygos collateral system which drains via the SVC. He regards the thoracic and abdominal cavities as a single uniform pressure chamber into which blood flow is impeded during the Valsalva maneuver. Since the paralumbarazygos venous system is not collapsible, it can be considered to lie outside the pressure chamber. It is apparent from clinical observation that the communicating venous channels outside the pressure chamber dilate...
markedly as the pressure within them rises. At the site where these dilated vessels join the cavae, a point of relative narrowing, but not true constriction, can be demonstrated (17).

In the present experiments, the mean velocity of flow in the SVC was reduced by about half, but it tended to rise to normal levels toward the end of the maneuver. This might be explained by the relatively small compliance of the SVC and its intrathoracic tributaries, which led to a relatively rapid rise in intravascular pressure as inflow continued; this would dilate the SVC and increase flow. In the records of velocity of flow from both the SVC and IVC during the Valsalva maneuver, the normal oscillations were altered with respect to the cardiac cycle. Under these conditions the driving pressure for flow is the difference between upstream intravascular and the extravascular pressures, so that the influence of right atrial pressure waves on the flow pattern would not be expected.

It is appropriate to say finally that the findings in this study confirm the work of Brecher (1), who, using different methods, defined the general features of caval flow. These features have been shown here to be present in normal conscious man, who is able to perform many maneuvers better than animals.

Acknowledgments

We wish to thank Dr. D. Mendel of St. Thomas's Hospital for the right heart pressure recordings used in Figure 3. We should also like to acknowledge the expert technical assistance provided by Mr. P. Burgess, Miss S. Marrington and Miss H. Grove.

References

Velocity of Blood Flow in Normal Human Venae Cavae
LEWIS WEXLER, DEREK H. BERGEL, IVOR T. GABE, GEOFFREY S. MAKIN and CHRISTOPHER J. MILLS

Circ Res. 1968;23:349-359
doi: 10.1161/01.RES.23.3.349
Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1968 American Heart Association, Inc. All rights reserved.
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:
http://circres.ahajournals.org/content/23/3/349

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org//subscriptions/