Cardiac Variations in Venous Return Studied
With a New Bristle Flowmeter

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According to present concepts blood returns passively to the heart. This view is based mainly on numerous studies which demonstrate that negative pressure does not develop in the ventricle during diastole (1–6). Little attention has been paid to the possibility that venous blood may be drawn toward the heart by its systolic movement, a concept first advanced by Aristotle (7). Burton-Opitz recorded flow in the jugular vein and calculated from his curves that flow is accelerated during systole (7). His instrument (Hürthle Stromuhr) had, however, such large effective mass and low frequency response that his curves are distorted by overshooting and therefore invalid. Hamilton (8) concluded from human cardiopneumograms that atrial inflow could be almost as large as the ventricular stroke volume, whereas Blair and Wedd (9) deduced from similar inferential evidence that Hamilton’s conclusion was quantitatively debatable. Böhme (1) observed with x-ray cineradiography a systolic acceleration of oil drops in the venae cavae of cats and dogs, but his findings could not be quantitated in terms of volume flow. Holzloehner and Schönerstedt (10) recorded volume flow in the jugular vein of dogs with a high-fidelity instrument (stream-bristle) which indicated systolic and diastolic flow accelerations. However, their records suffer from unaccountable artifacts and time inaccuracies due to the lack of simultaneous pressure registration and to the interposition of a long venous segment between the place of measurement and the heart.

Obviously, accurate information is still lacking concerning venous return during different periods of the cardiac cycle. The present investigation attempts to fill this gap in our knowledge.

The solution of this question depends mainly upon the development of an instrument which measures direct flow at the entrance of the atria. The use of an apparatus which requires long tubing is excluded because it interferes with the natural collapse of the veins. A new type of electrically recording bristle flowmeter was, therefore, constructed which permits in situ measurement of volume inflow into the heart under physiological conditions (II).

APPARATUS

Figure 1 is a schematic diagram of the bristle flowmeter previously described (12). A number of details, upon which the successful operation of the instrument depends, were not previously described and are now reported. The flowing blood deviates a small ‘bristle’ (B) which is suitably inserted into the blood stream. The bristle is firmly attached to the plate pin (P). The movement of the plate pin inside the tube changes the number of electrons flowing from the cathode to the anode, thereby giving rise to an electrical signal. This signal is amplified and drives a high frequency mirror oscillograph.

The brass flow cannula consists of three parts (X, Y, Z), assembled by threaded joints. The transducer tube, mounted in a socket (X), has a metal coating which must be firmly grounded by friction contact with the socket. Since the metal jacket of the tube does not permit soldering, electrical continuity from the plate pin to ground is established by soldering the ground wire to socket X. Seepage of blood to the electrical leads at the base of the transducer is prevented by imbedding the

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tube with heat-resisting cement into the socket. 

A 32-mm-long segment of watchspring (Elgin 6094) was used as a 'bristle.' Section Y of the flowmeter cannula gives the bristle sufficient lever length for adequate deviation. Furthermore, interposition of a stationary blood column in section Y between the streaming blood and the transducer prevents temperature fluctuations of the vacuum tube which would alter the cathode’s electron emission. Provision for pressure recording, removal of air bubbles and flushing is accomplished by means of a side tube attached to section Y. Section Z is inserted and fastened into the superior vena cava. Prior to an experiment section Z is adjusted in its position and fixed to Y by a universal joint in order to furnish maximal sensitivity, for the plate responds maximally to deviation in one plane and minimally in the plane 90° to it.

Zero drift or base line instability, the most serious disadvantage of flow recording by use of electronic devices, is greatly reduced by use of: a) a battery supply of filament and anode current for the transducer, and b) a 2

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2A mixture of 1/4 Permatex gasket cement and 1/4 Walsco Radio Cement thinned with General Cement Radio service solvent.

Keithley Chopper Amplifier in which zero drift is less than 30 µV for a 100-V signal/hr. With this arrangement, zero drift remained so small that it could not be detected over the period of experimental observation. The average flow recording lasted about 30 seconds with the photographic paper moving at a speed of 70 mm/sec. Zero flow, recorded at the beginning and the end of each record by interrupting caval flow for 1 second, was unchanged when measured in relation to a fixed base-line.

The bristle flowmeter is a velocity recorder which permits determination of volume flow when the cross sectional area of the blood vessel is fixed, e.g., by inserting the cannula head Z into the vein. The flowmeter is calibrated with steady flows in situ by inserting one rigid tube into the severed central and another into the peripheral end of the caval vein. No difference was found between in situ calibrations and those done outside the body (both measurements were made with the animal's own blood).

The flowmeter does not produce turbulence up to flow rates of 1200 cc/min. At higher flows, turbulence is apparently caused by the lumen of the vertical part of the cannula (Y). Fibrin deposits were never observed since cannula and bristle were siliconized and the animals heparinized.

The mechanical frequency response of the transducer tube is 12,000 cps. This frequency is reduced to 125 cps by attaching the bristle. Figure 2 is a record of its frequency response visualized on a cathode ray oscillograph. From this high fidelity it must be deduced that the instrument permits recording of rapid flow changes without distortion.

The metallic anode plate of the 5734 tube acts as a fulcrum for the plate pin and bristle. According to RCA specifications the mechanically safe range of plate pin deviation extends to 30 arc minutes. The output of the transducer at this degree of deviation is 40 v. The maximal voltage of the signal obtained by the bristle deviation from the in situ blood stream was 1 v, corresponding to less than 1 arc minute, since plate pin deviation and voltage output are a linear function. Such deviation is well within the mechanically safe range. An additional check on the mechanical stability of the plate pin fulcrum
is furnished by the accurate reproducibility of the zero flow calibration at the beginning and the end of each flow record.

The bristle flowmeter's resistance to flow is very small. The superior vena cava is slightly stretched to fit over the cannula lips of part Z. A central vein is ordinarily in a partially collapsed state with an oval cross sectional area. Resistance is, therefore, not increased by expanding the vein at the place of cannulation to a round cross sectional area. The Z part of the inserted cannula is only 1 cm long and does not interfere with the normal venous collapse mechanism since the collapse changes are freely transmitted from the central to the peripheral part of the vein across the cannula.

PROCEDURE

Acute experiments were undertaken in 10 dogs ranging from 15-22 kg in weight; the animals were anesthetized with 3 mg/kg morphine sulfate subcutaneously and 15 mg/kg pentobarbital intravenously. In six experiments, the chest remained open during recording, and in four it was closed after the flowmeter insertion. In addition to superior caval flow the following pressures were recorded with modified Gregg manometers: aortic, superior vena caval or right atrial, endotracheal, external jugular, and in one experiment also right intraventricular.

RESULTS

Flow patterns like pressure contours varied from one experiment to another. Therefore, numerous flow curves were reviewed and a typical one selected which incorporated those features common to all curves.

Figure 3 shows the flow changes in the superior vena cava during the cardiac cycle when the chest is open. Vertical lines were placed on the record to correlate flow with the different phases of the cardiac cycle.

At the beginning of isometric ventricular contraction (point 1 in fig. 3) superior caval flow was already in the process of moderate acceleration which started with atrial muscle relaxation. This flow augmentation was briefly halted, as indicated by a small notch in the flow tracing between 1 and 2, and is apparently caused by the closure and bulging of the tricuspid valves. The greatest acceleration occurred during the phase of rapid ventricular ejection (2-3). Then flow began to decelerate during the phase of reduced ventricular ejection (3-4).

During the brief protodiastolic interval (4-5) and during isometric relaxation flow continued to decelerate. With the opening of the tricuspid valves at 6 the rate of deceleration was retarded and this continued during the rapid inflow phase of the ventricle (6-7). During the short phase of diastasis (7-8) flow accelerated moderately and at the beginning of atrial contraction (8) the flow rate dropped again, reaching zero at the peak of atrial systole (8-9).

In summary, the flow curve shows two main summits, the higher systolic summit indicating the existence of a strong force which accelerates venous return.

Various maneuvers were undertaken to study the forces which affect the return flow of blood during different parts of the cardiac cycle.

Factors Influencing Flow During Atrial Contraction. Atrial systole obviously has a two-fold effect. It forces blood into the ventricle, as commonly known, but at the same time it retards the onflowing blood from the vena cava. Lung inflation in the open chest is generally believed to have no effect on phasic flow. This proved not to be the case as illustrated by figure 4. From left to right the tracing shows that the flow rate dropped to zero during atrial systole when the lungs were deflated just prior to the first cardiac.
cycle. During mild positive pressure lung inflation, venous return decreased progressively until a reversal of flow developed which equaled 180 cc/min. in rate prior to the fourth cardiac cycle. The cause is found in the reduction of the pressure gradient between the jugular and superior caval veins. Pressures were measured at the peak of atrial systole in both veins, making allowance for the retrograde pressure transmission delay of 80 msec. While the pressure gradient between the jugular and caval vein amounted to 41 mm of water before lung inflation, it dropped to a minimum of 35 mm of water prior to the fourth cardiac cycle when the flow reversal was greatest. The dammed up blood led to a rise of jugular pressure and, due to continuous flow from the head, the pressure gradient increased again during the latter part of lung inflation to 39 mm of water. Thus, flow reversal during the fifth cardiac cycle was not so great in spite of greater lung inflation. With lung deflation the dammed up blood immediately augmented forward caval flow and blood entered the atrium even at the peak of atrial systole at a rate of 210 cc/min.

In summary, venous return during positive pressure lung inflation in the open chest is diminished by the operation of two factors, first a reduction of the phasic forward flow toward the right atrium and secondly by the development of reverse flow during atrial systole.

Factors Influencing Flow During Ventricular Contraction. The amount of blood entering the right atrium from the superior cava can be determined by measuring the area under the flow curve. It was found that venous return is greater during ventricular systole when the tricuspid valves are closed and no blood can enter the ventricle (2.35 cc volume inflow from 1-4 in fig. 3) than during ventricular diastole when the tricuspid valves are open and atrium and ventricle form a common cavity (2.1 cc from 4-1 in fig. 3). Since the duration of ventricular systole is relatively less affected by the heart rate than that of diastole, it was of interest to see if the ratio of 'systolic' and 'diastolic' inflow into the atrium is altered by varying the duration of diastole.

Figure 5 illustrates the effect of different heart rates on venous return in the open chest. Segments A and B are taken from a continuous record, 10 seconds apart. Segment A depicts two consecutive heart beats, the first one with a long lasting diastole and the second one with a shorter duration of diastole. The diastole was further shortened in segment B by stimulating the atrium 3 times/sec. The measurements from this record are presented in table I. It is noted that the duration of systole changed only a little with the speeding up of the heart rate and that venous return during systole was barely affected. With a heart rate of 83/min. a volume of 2.8 cc entered the atrium from the superior cava (first beat in segment A), and with tachycardia of 177 beats/min., 2.5 cc (last beat in segment B). However, the respective diastolic inflow amounted to 3.7 cc in bradycardia and only 0.6 cc with tachycardia. Total superior caval inflow remained nearly the same at all heart rates (about 9 cc/sec.). Merely the ratio of systolic to diastolic inflow changed.

These data show that proportionally more blood entered the atrium during ventricular systole than during diastole as the heart rate increased. At slow heart rates, about half the venous blood returned to the atrium during ventricular systole, but with tachycardia, four-fifths.

The record of figure 5 demonstrates also that the entire amount of blood which entered
the atrium during systole must have left the atrium and moved into the ventricle during the very brief diastolic period in tachycardia. This is evident from three facts: a) atrial pressure declined, which indicates that blood was not backed up in the venous tree and the right heart did not fail to move the onrushing blood, b) flow reversal in the superior cava did not increase with tachycardia, and c) total superior caval flow per unit time did not decrease with the faster heart rate.

**Factors Influencing Flow During Ventricular Diastole.** Records 3, 4, and 5 show the relative unimportance of ventricular diastole in promoting venous return. These records were taken in open chest preparations. However, in the closed chest negative intrathoracic pressure may enter as an additional factor promoting venous return. It has been frequently postulated that negative intrathoracic pressure exerts direct suction on the atrial and ventricular wall thereby expanding the heart's cavities passively and in turn permitting greater inflow from the venae cavae.

Two approaches were made to study this question, one in closed chest and the other in open chest experiments. Figure 6 shows a record of a closed chest experiment in which diastolic flow was unusually large after an infusion of 200 cc of blood in order to make the effect of thoracic aspiration more noticeable in the flow curves. Intrathoracic pressure conditions during the first and second cardiac cycle corresponded to those of an expiratory pause; the endotracheal pressure was atmospheric and the pressure in the superior cava below zero. Under this condition, right atrial volume inflow from the superior cava amounted to 2.33 cc during systole and 4.8 cc during diastole (first cardiac cycle). The effect of increased thoracic aspiration on diastolic inflow was tested by mechanical lowering of endotracheal pressure from zero to $-130$ mm of water at the beginning of the third cardiac cycle, marked by an arrow. Inflow increased during diastole from 4.8-7.4 cc (fourth cardiac cycle), and also during systole from 2.33-3.4 cc.

The greater augmentation of caval flow during ventricular diastole than during systole is suggestive evidence that the atrioventricular cavity is aided directly in its dilation by greater thoracic aspiration. This evidence is, however, not conclusive because atrial inflow during ventricular systole is also augmented, though not so much. The reason for the overall flow increase with greater thoracic aspiration is the depletion of the extrathoracic veins into the thoracic veins, a process which has been described formerly (12).

Controlled open chest experiments were conducted in order to eliminate the flow increase which occurs as a result of the depletion of the peripheral venous reservoirs,
which in turn is due to the increased thoracic aspiration. After opening the pericardium, a suction cup was directly applied to the right ventricular wall to simulate 'thoracic aspiration.' Figure 7 presents two segments of a continuous record taken 3 seconds apart. In the control (segment A) superior caval flow acceleration during systole exceeded that occurring during diastole. In segment B a suction cup of 4 cm in diameter exerting −200 mm of water pressure was applied to the right ventricular wall. It is noted that diastolic inflow (marked D) became greatly augmented compared to the reduced systolic flow (marked S). Similar records were obtained when both ventricles were enclosed in a cardiometer and 150 mm of water suction was created. The flow reversal in record B of figure 7 (marked

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X) is apparently caused by an artifact as revealed by the vibrations in the superior caval pressure tracing. This disturbance may have originated from an interference of venous movement by the suction cup.

**DISCUSSION**

These experiments elucidate the role which the heart plays in promoting venous return. Of particular importance is the quantitative finding from direct blood flow measurements that flow toward the heart is greatly accelerated during ventricular contraction. This presents the heretofore missing conclusive evidence in support of the concept that the contraction of the ventricle itself propels blood toward the heart.

The mechanism which could cause the acceleration of venous blood during ventricular systole has been frequently described in the past (1, 8–10). According to anatomical, physiological and X-ray studies, the heart’s apex must be considered the punctum fixum which remains relatively stationary during ventricular contraction, while the atrioventricular junction is the punctum mobile which descends toward the apex. The result of the ventricular contraction would then be

The simultaneous occurrence of the two events, the ventricular systole, and the marked venous flow acceleration leave little doubt that it is the contraction of the ventricular muscle itself which is the responsible force for drawing the blood toward the heart. This raises the time-honored question of the existence of a ‘vis a fronte.’ According to present concepts blood returns to the heart only passively by means of the ‘vis a tergo.’ The data of the experiments presented in this communication make, however, a good case for the existence of a very substantial vis a fronte, if one agrees on a definition of the vis a fronte as a force exerted by the heart itself to attract blood toward it. It is the frontal force of the contracting myocardium which attracts the venous blood by the descent of the atrioventricular junction. The strength of this force can be estimated by the degree of acceleration imparted to the venous blood column. The maximal acceleration of superior caval blood during ventricular systole amounted to 700 cc/sec/sec. This acceleration is of similar magnitude as that of blood in the pulmonary artery (1500/cc/sec/sec., 13).

The function of ventricular contraction is, therefore, a two-fold one. It consists not only of the ejection of blood from the ventricle, but also of the ‘injection’ of blood from the veins into the right atrium. It could be demonstrated by direct flow measurements that this mechanism is of particular importance during tachycardia. While the organism is in the normal state of rest and bradycardia prevails, a large portion of the venous inflow into the atrium occurs during ventricular diastasis. This portion decreases when the heart beats faster. During tachycardia 80% of the blood may enter the atrium during ventricular systole. Obviously, only the

**TABLE I. EFFECT OF HEART RATE ON VENOUS RETURN DURING SYSTOLE AND DIASTOLE IN A REPRESENTATIVE EXPERIMENT**
remarkable acceleration of blood by the ventricular contractions could lead to adequate atrial filling during this period. Without the systolic acceleration the heart may be expected to ‘beat itself empty’ with tachycardia.

The fundamental importance of this finding should be emphasized. It demonstrates that the heart possesses a self-regulatory mechanism to keep its venous return constant by shifting the proportion of its atrial filling from a predominantly passive inflow during the long ventricular diastasis of bradycardia to a largely active inflow with tachycardia due to the rapidly repeated systolic attraction of the venous blood. This mechanism must, therefore, be looked upon as an innate safety device of the heart by which it assures instantaneously its own atrial filling with sudden heart rate acceleration, such as may occur during stress or anticipation of stress. Knowledge of this mechanism makes it understandable that cardiac output can be maintained fairly well at fast heart rates, because each myocardial contraction attracts almost the same amount of blood from the veins into the atrium which the ventricle ejects into the arteries (fig. 5).

The question now arises how the large amount of blood which enters the atrium during ventricular systole and which is momentarily blocked there by the closed tricuspid valves is then transferred into the ventricle during the brief diastole of tachycardia. It must be assumed that the acceleration imparted to the venous blood column by the ventricular contraction aids the rapid ventricular filling during early diastole. The greatly accelerated blood, which still moves at high flow rates even after some deceleration, obviously continues its flow directly through the atrium into the ventricle with the opening of the tricuspid valves, since the blood does not back up in the veins (fig. 5).

Particularly of clinical interest are the beat-to-beat measurements of venous return showing that mild positive pressure lung inflation in the open chest not only reduces forward flow of blood in the central veins toward the heart, but even reverses the direction of flow during a brief phase of the cardiac cycle. This finding emphasizes that positive pressure lung inflation should be cautiously applied during open thoracotomy surgery in order to minimize embarrassment of venous return.

Frequently it has been postulated that one of the factors aiding venous return may be the effect of negative intrathoracic pressure which could dilate directly the atrioventricular
cavity during diastole for greater inflow and accommodation of blood. It has now been experimentally established that this is actually the case. Attention should be called to the fact that this mechanism comes into play concomitantly with another one which increases total venous return during all parts of the cardiac cycle by emptying the extrathoracic venous reservoirs into the thoracic veins with greater thoracic aspiration (12). Both mechanisms supplement one another in aiding the return flow of blood to the heart. This direct dilating action of negative intrathoracic pressure on the atrioventricular cavity explains also the finding of Rushmer et al. (14) who showed with x-ray cinematography that the diastolic volume of the ventricle and auricle is larger in closed chest animals than in thoracotomized ones.

It may be thought that measurement of flow in only one caval vein might not be indicative of blood inflow from the other. However, there is good evidence from other experiments in which superior and inferior caval flow was recorded simultaneously, that flow in both vessels is simultaneously accelerated and decelerated (15). In this connection it is of interest to note that coronary sinus outflow is also augmented during ventricular contraction due to a compressing effect of the myocardium on the coronary venous tree (16). These findings would indicate that myocardial action promotes venous return simultaneously by different mechanisms in the central as well as coronary veins.

**SUMMARY**

Cardiac factors influencing venous return have been studied in acute dog experiments with a new bristle flowmeter of high fidelity. Directly measured blood flow in the superior vena cava is greatly accelerated during ventricular systole when the tricuspid valves are closed. Simultaneously recorded blood pressure and flow curves indicate that this acceleration is caused by the contraction of the myocardium which expands the atrium through a piston-like descent of the atrioventricular junction. Thus, each ventricular contraction ejects not only blood into the arteries but also draws blood from the veins into the atrium.

Venous return is only slightly, if at all, accelerated during ventricular diastole. Conclusive experimental evidence is furnished supporting the view that venous return is augmented during ventricular diastole by the direct sucking action of increased negative intrathoracic pressure on the walls of the atrioventricular cavity. During atrial contraction flow in the central vein is briefly stopped or even reversed. In the open chest this flow reversal is accentuated by positive pressure lung inflation.

The total amount of superior cava flow per unit time remains constant at different heart rates. With bradycardia the larger portion of blood enters the atrium during ventricular diastole, whereas with tachycardia 80% of the atrial inflow occurs during ventricular systole. Thus, the heart possesses a self-regulatory mechanism by shifting its atrial filling from a largely passive inflow during the long diastole at slow heart rates to an active systolic attraction of venous blood with tachycardia. It is emphasized that through this fundamental mechanism the heart assures its own venous return upon which the maintenance of cardiac output depends.

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