Influence of right atrial pressure pulse on instantaneous vena caval blood flow

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BRAWLEY, ROBERT K., H. NEWLAND OLDHAM, JOHN S. VASKO, R. PETER HENNEY, AND ANDREW G. MORROW. Am. J. Physiol. 211(2): 347-353. 1966.—The patterns of instantaneous blood flow in the superior and inferior vena cavae were determined, and related to right atrial and right ventricular pressure events in 40 dogs. The character of the normal right atrial pressure pulse was changed by producing tricuspid regurgitation or pulmonary stenosis, and the normal sequence of right atrial and right ventricular pressure events was altered by the experimental induction of nodal rhythm, atrial fibrillation, or complete heart block. In all dogs, in the control state and during the various experimental conditions, there was a constant and inverse relationship between the contour of the right atrial pressure pulse and the instantaneous vena caval flow pattern. The lack of a constant relationship between ventricular systole and the acceleration of caval blood during cardiac arrhythmias indicates that the phasic pattern of instantaneous thoracic vena caval flow is determined by right atrial pressure events which predominate over any effects attributable to “ventricular systolic suction.”

venous blood flow; venous return; ventricular systolic and diastolic suction; pressure-flow relationships in the vena cavae

The patterns of instantaneous blood flow in the intact systemic venous circulation have received little attention, although many studies have been directed to determination of the factors that influence the amount of venous blood returned to the heart. Fluctuations in venous pressure associated with contraction of the heart were observed by early investigators (13, 15, 20, 21), and the phasic nature of vena caval flow has been demonstrated in animals by contrast cinefluorography (2, 12, 16) and in man by a differential pressure technique (14). Brecher has made important contributions to this neglected subject and, using a bristle flowmeter, directly determined both the volume and instantaneous velocity of blood flow in the vena cavae (3, 4). From these and other studies, several investigators have concluded that venous return is dependent not only on passive right atrial filling provided by the pressure gradient between the venous reservoir and the right atrium, the force from behind or vis a tergo, but also on an attraction of blood from the veins, the force from the front or vis a fronte, produced by contraction of the heart and inspiration (4, 6, 7). Brecher demonstrated that vena caval flow was markedly accelerated during ventricular systole, and used this evidence to support the concept of “ventricular systolic suction.” Other investigators, however, consider that central venous pressure pulsations decrease rather than augment venous return (8, 9).

In the present studies, instantaneous thoracic vena caval blood flow was measured with an electromagnetic flowmeter, and related to right atrial and right ventricular pressure events in dogs with various experimentally induced hemodynamic alterations. The effects of these abnormal hemodynamic conditions and the relative importance of atrial and ventricular pressure events in determining the dynamics of vena caval flow were assessed.

METHODS

Mongrel dogs weighing 16–20 kg were anesthetized with intravenous chloralose (60 mg/kg) and ventilated with 100% oxygen supplied through a cuffed endotracheal tube by a positive-pressure respirator. Heparin (2 mg/kg) was given intravenously, and a thoracotomy was performed through the right fourth intercostal space. Specially designed intravascular electromagnetic flow transducers (Medicon QH-2070-C; QH-2080-C) were placed in the superior and inferior vena cavae immediately distal to their junctions with the right atrium. A transducer with an internal diameter of 7 mm was used in the superior cava, and one with an internal diameter of 8 mm in the inferior cava. In animals of the size utilized the transducers did not obstruct caval flow; pressures were measured in the cavae immediately proximal and distal to the transducers and no pressure gradient

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was ever demonstrated. The flow transducers were employed in conjunction with a Medicon M-4000 flowmeter. The two transducers were calibrated daily with measured flows of saline, and the two flowmeter amplifiers were adjusted to be equisensitive. Zero flows were determined at the conclusion of each study by occlusion of the venae cavae between the heart and the flow transducers.

Teflon Gensini catheters, size 0F and 15 cm in length, were placed into the right atrium through the ligatedazygous vein, into the right ventricle through a stab wound at the apex, and into the aorta through the femoral artery. The catheters were attached to Statham P23Db pressure transducers. Instantaneous blood flow in the superior and inferior venae cavae, and aortic, right atrial, and right ventricular pressures were simultaneously recorded on a multichannel photographic oscillograph. In order to eliminate respiratory variations in flow and pressure, the respirator was stopped for a brief period when the recordings were made.

Observations were obtained under 1) Control conditions, before and after pericardiotomy, and after the following experimental interventions: 2) Tricuspid regurgitation or pulmonary stenosis: in 10 dogs, during a brief period of venous inflow occlusion, the anterior leaflet of the tricuspid valve was excised. Observations were made acutely in five of these dogs with tricuspid regurgitation, and 12–16 weeks after operation in the other five. Acute pulmonary stenosis was produced in nine dogs by gradual constriction of the main pulmonary artery. Recordings were made at the highest right ventricular pressure that could be obtained without an associated fall in systemic arterial pressure. 3) Atrial or ventricular septal defects: atrial septal defects were created in six dogs by the Blalock-Hanlon procedure (1), and ventricular septal defects were made in four animals with a cork borer which was inserted through a purse-string suture in the outflow tract of the right ventricle (11). 4) Cardiac arrhythmias: in eight dogs, nodal rhythm was produced by crushing the sinoatrial node. Atrial fibrillation was acutely induced in seven dogs by the application of a few crystals of aconitine to the surface of the right atrium

Fig. 1. Continuous recordings of the electrocardiogram, instantaneous flow in the inferior vena cava (IVC), superior vena cava (SVC), and right atrial pressure (RA) made in a normal dog before, during, and after wide incision of the pericardium (arrow). The patterns of instantaneous venous flow are identical before and after pericardiotomy; mean flow in the IVC was 1,150 before and 1,175 ml/min after the pericardium was opened, the mean SVC flows at these times were 400 and 425 ml/min, respectively. The mean right atrial and systemic arterial pressures (not shown) were unchanged. The high-frequency deflections in the RA pressure record are artifacts, the result of motion of the RA catheter during the pericardiomy.

Fig. 2. Simultaneous recordings of the electrocardiogram, instantaneous blood flow in the inferior vena cava (IVC) and superior vena cava (SVC), and right atrial pressure (RA) in a normal dog during control conditions. The caval flow pattern is the one most frequently noted (18 dogs). It is biphasic, and least forward flow occurred at the points designated in the flow records as a and v; these correspond to the peaks of the atrial a and v pressure waves. Forward caval flow rapidly accelerated during atrial relaxation and the y descent, and became maximal at times corresponding to the lowest right atrial pressures. In this animal the e wave in the atrial pressure pulse had no detectable influence on caval flow.

RESULTS

1) Control conditions. In initial studies in three dogs, superior and inferior vena caval flow and right atrial and systemic arterial pressure were recorded continuously before, during, and after wide incision of the pericardium (Fig. 1). In no animal did pericardiotomy cause any detectable change in the patterns of venous flow. Accordingly, observations in subsequent experiments were made with the pericardium open. The patterns of instantaneous superior and inferior vena caval flow were recorded under control conditions in 30 normal dogs with regular sinus rhythm. In them the mean right atrial pressures ranged from –1 to 5 mm Hg and the...
phasic or biphasic flow patterns, small amounts of reverse flow occurred at the peak of the prominent atrial a wave; least flow was observed during atrial relaxation and became maximal at the points of lowest right atrial pressure. In nine normal animals, the atrial v wave was inconspicuous, and in most records, the atrial c wave was of small amplitude and was not associated with any apparent alteration in flow. Occasionally, however, a prominent c wave gave rise to a triphasic flow pattern, as shown in Fig. 4.

The inverse relationship between right atrial pressure and instantaneous vena caval flow is demonstrated in another way in Fig. 5. In this record the flow signals were electronically inverted, and the peaks in the flow pattern represent points of minimum flow. It is apparent that the atrial pressure tracing and the inverted flow pattern have quite similar contours, but the peak deflections are not precisely related in time. In all the tracings the changes in caval flow occurred shortly after the corresponding change in atrial pressure. In vitro studies utilizing the same flow transducers, pressure catheters, and recording system employed in the present investigation of vena caval flow indicated that transmission times for pressure and flow signals differed by less than 10 msec; therefore, the observed delay between corresponding changes in caval blood flow and right atrial pressure is attributed to the time required for transmission of the atrial pressure pulse to the cavae and the complex impedence of the system. Also, the time of peak forward or peak regurgitant flow in the inferior cava was almost always later than that in the superior cava. This difference is probably attributable to the greater volume and inertia of flow in the inferior cava (18).

2) Tricuspid regurgitation or pulmonary stenosis. In the 10 dogs with either acute or chronic tricuspid regurgitation, the mean right atrial pressure was elevated (average 5 mm Hg) and the prominent atrial pressure peaks which occurred during ventricular systole ranged from 8 to 20 mm Hg. Records of right atrial pressure and caval blood flow recorded in one dog with chronic tricuspid regurgitation are reproduced in Fig. 6. In all animals, large volumes of retrograde flow occurred in both the superior and inferior venae cavae, coincident with the highest atrial pressure peak. The flow patterns were similar in the two cavae, but the time of maximum retrograde flow was usually later in the inferior cava. Also, when the c wave was distinctly separated from the v wave, its effect on flow was most readily apparent in the superior cava.

In the nine dogs with acute pulmonary stenosis, the mean right atrial pressure was elevated to an average of 6 mm Hg, and the atrial pressure waves were usually characterized by prominent a waves. The outflow obstruction also resulted in diminished cardiac output and total venous return. Retrograde flow was always recorded at the atrial pressure peak in the superior cava, and often in the inferior cava as well (Fig. 7). With both tricuspid regurgitation and pulmonary stenosis, acceleration and deceleration of forward flow corre-
tion while the tricuspid valve is closed. Typical records of vena caval flow and right atrial and ventricular pressure during nodal rhythm are reproduced in Fig. 8. Significant retrograde flow occurred in both venae cavae in all animals at the peak of the atrial pressure pulse and forward flow was accelerated during atrial relaxation.

Atrial fibrillation resulted in right atrial pressure pulses characterized by irregularly occurring v waves and absent a waves. Again, forward caval flow accelerated rapidly during the v descent and prior to ventricular contraction (Fig. 9). Furthermore, in most cycles ventricular systole corresponded to a period of decreasing rather than increasing forward flow.

Complete heart block resulted in the familiar dissociation of atrial and ventricular pressure events. As shown in Fig. 10, each increase in right atrial pressure caused by atrial contraction was followed by diminished forward caval flow, and flow accelerated sharply during atrial relaxation. This acceleration of forward vena caval flow was of similar magnitude whether or not atrial relaxation was followed by ventricular contraction. When the atrium contracted while the tricuspid valve was closed, resulting in a cannon wave, large volumes of retrograde flow were noted in both cavae.

The primary role of the right atrial pressure events in the determination of vena caval and the lack of a significant effect from ventricular contraction are shown in the records reproduced in Fig. 11. In this dog transient atrioventricular dissociation occurred during the study. During normally conducted beats, seen at the right of the record, forward caval flow accelerated during atrial relaxation, which, of course, occurred immediately before ventricular contraction. In the remainder of the record, however, atrial relaxation is seen to occur at varying times in relation to ventricular contraction, and the influence of each event on caval flow may be assessed independently. In every cycle maximum acceleration of forward flow occurred during the descent of the atrial pressure pulse, and minimum flow coincided with peak atrial pressure. In contrast, no relationship was evident between ventricular contraction and the acceleration of caval flow.

DISCUSSION

The patterns of instantaneous vena caval blood flow that were recorded in the normal dogs revealed one, two, or occasionally three peaks of distinct forward flow during each cardiac cycle. Also any conspicuous peak in the right atrial pressure pulse, whether caused by atrial contraction, ventricular contraction, or passive right atrial filling, was always associated with a decrease in caval blood flow. Similarly, any abrupt decrease in right atrial pressure, regardless of its cause, always corresponded to a period of acceleration of caval flow. Although the flow in both the inferior and superior venae cavae reflected right atrial pressure events, the influence of pressure waves of small magnitude was more
In the studies made after the production of various cardiac arrhythmias the normal sequence of pressure events during the cardiac cycle was altered. Under these circumstances it became possible to assess independently the effects of atrial and ventricular contraction on caval flow. Thus in nodal rhythm contractions of the right atrium and right ventricle occurred simultaneously, and the resultant cannon waves caused large amounts of retrograde flow in both cavae; marked acceleration of caval flow occurred with the fall in pressure during atrial relaxation. Since effective atrial contractions were eliminated during atrial fibrillation, the isolated influence of ventricular contraction on caval blood flow could be determined, and during this arrhythmia the only changes in caval flow were related to the rise and fall of the atrial v wave. In dogs with complete heart block, atrial contraction produced atrial pressure waves which always corresponded to sharp decreases in forward caval blood flow; when the atrium contracted against a closed tricuspid valve, the atrial pressure pulse was augmented and much larger decreases in forward flow, or even retrograde caval flow, occurred.

In this investigation the experimental production of an atrial or ventricular septal defect did not appreciably alter the magnitude or contour of the right atrial pressure tracing, although intracardiac flow was surely changed. Significantly, there was no difference in the caval flow patterns in comparison to those recorded during control conditions in these same dogs.

In all animals studied, whether under control conditions or during various experimental hemodynamic alterations, the pattern of instantaneous caval flow, when related to right atrial and ventricular pressure events, was always found to reflect inversely the contour of the right atrial pressure tracing. No correlation with right ventricular pressure events was apparent, except as ventricular contraction influenced right atrial pressure.

The influence of the ventricle on the pattern of vena caval blood flow has been emphasized by Brecher (4) and Brecher and Galletti (6), whose interpretation of the dynamics of instantaneous blood flow in the venae cavae is that most widely accepted. He stated that ventricular contraction resulted in a downward movement of retrograde flow in both cavae; marked acceleration of arterial blood flow occurred with the fall in pressure during atrial relaxation. Since effective atrial contractions were eliminated during atrial fibrillation, the isolated influence of ventricular contraction on caval blood flow could be determined, and during this arrhythmia the only changes in caval flow were related to the rise and fall of the atrial v wave. In dogs with complete heart block, atrial contraction produced atrial pressure waves which always corresponded to sharp decreases in forward caval blood flow; when the atrium contracted against a closed tricuspid valve, the atrial pressure pulse was augmented and much larger decreases in forward flow, or even retrograde caval flow, occurred.

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atrial muscular contractions barely affect flow in central veins, blood is distinctly accelerated during ventricular systole. (3) The acceleration of venous flow during ventricular systole can be decreased by opening the pericardium or by removing the support of the heart in other ways.” His records also showed that the typical venous flow pattern was biphasic, one peak occurring during ventricular systole and the other during diastole, and that in the closed-chest dog spontaneous inspiration augmented diastolic more than systolic flow. He ascribed the diastolic acceleration to negative intrathoracic pressure (3) or “ventricular diastolic suction” (5). In summary, Brecher concluded that ventricular pressure events were primarily responsible for the acceleration of vena caval flow, and the ventricular mechanism of venous flow acceleration was evidence in support of the concept of cardiac vis a fronte.

In the present study, pericardiotomy was shown to have no effect on either the pattern or magnitude of caval flow. Furthermore, records of instantaneous caval flow, made in normal dogs with open chests, indicated that in some dogs most of the venous return occurred during ventricular diastole, whereas in others it took place during ventricular systole. Occasionally, in the same animal and without obvious cause, the major portion of forward caval flow was noted to shift from ventricular systole to diastole, or conversely. The influence of respiration on the magnitude of venous flow during ventricular systole and diastole has been studied in both open- and closed-chest animals (4,16); in the present study, the effect of respiration was eliminated in order to evaluate more clearly the influences of cardiac contractions on venous flow.

During regular sinus rhythm, ventricular contraction follows atrial relaxation so closely that it is impossible to determine which event is primarily responsible for the acceleration of forward flow seen at this time in the vena caval flow pattern. However, the simultaneous flow and pressure records obtained during nodal rhythm (Fig. 8) or atrioventricular dissociation (Fig. 11), clearly demonstrated that if forward caval flow was accelerated by relationship between the two. In contrast, peak atrial pressure always slightly preceded minimum caval flow, and atrial relaxation resulted in acceleration of caval blood flow in every cardiac cycle.
ventricular systolic suction, this effect was abolished by the occurrence of a large positive atrial pressure deflection during ventricular systole. Brecher found evidence to support the concept of ventricular systolic suction in animals with 2:1 or 3:1 heart block, and stated that forward caval flow was not accelerated unless atrial relaxation was followed by a ventricular contraction. These observations are at complete variance with those made in this study in dogs with complete heart block. In them a distinct acceleration of caval flow was associated with every period of atrial relaxation regardless of whether it was succeeded by a ventricular systole (Fig. 10). Also in contrast to Brecher’s findings, the onset of acceleration of vena caval flow during atrial fibrillation was clearly found to occur before the onset of ventricular systole (Fig. 8). Thus ventricular systole was found to coincide with varying phases of the caval flow pattern depending on the preparation studied. In all records obtained in the present investigations, acceleration of forward vena caval flow corresponded to a decrease in right atrial pressure, and deceleration of caval flow corresponded to an increase in atrial pressure. Right ventricular contraction, on the other hand, influenced vena caval flow only as it altered right atrial pressure.

REFERENCES


