Dynamics of the Orifices of the Venae Cavae
Studied by Cineangioangiography

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Closure of the caval orifices of the right atrium and dynamics of the venae cavae have been studied by an analysis of high-speed cineangioangiographic records. The influence of respirogenic and cardiogenic factors on venae cavae dynamics was demonstrated. Findings indicated that mechanical and hemodynamic factors interact in directing the atrial blood forward and in preventing its reflux into the venae cavae. Relative narrowing of the caval orifices was demonstrated during atrial systole. The degree of narrowing, however, was insufficient to produce a mechanical closure and thus to prevent caval refluxes when the pressure gradients were altered. The findings are illustrated and discussed.

THE anatomical studies of Keith demonstrated the myocardial structures of the right atrial walls and their function in closing off the venous orifices during atrial systole. Keith further postulated that under the stress of back pressure, as in the diseased heart, these myocardial bands become weak, thus producing the incompetence of the caval orifices.

The existence of such a mechanism remained controversial and lately has been denied. However, recent angioangiographic studies seem to have lent considerable support to the Keith postulates.

Hedman, Lind, and Wegelius, utilizing rapid (6 to 12 exposures per second) serial angioangiography demonstrated the systolic hesitation of contrast material at the superior caval orifice, with free entry into the right atrium during diastole. In a patient with atrial left-to-right shunt they also found, by demonstrating abnormal caval refluxes occurring during atrial systole, that the mechanism closing the inferior vena cava breaks down under stress. Campeti et al. were able to show by cineangioangiography that a similar incompetence develops at the orifice of the coronary sinus. A reflux into the coronary sinus and its tributary veins was observed during atrial systole in 88 per cent of patients with right heart hypertension.

Kjellberg and Olsson, utilizing serial angioangiography, demonstrated a narrowing of the caval and pulmonic venous orifices in both dogs and human subjects during atrial systole. They described a sphincter within the right atrial silhouette and concluded that "a sphincter mechanism is present at the junction of the caval and pulmonary veins with the auricles."

This paper is presented as a further contribution to the problem of dynamics of the caval orifices and of the venae cavae in man.

MATERIAL AND METHODS

Cineangioangiographic examinations, at speeds of 15 and 30 frames per second, were made in 150 patients, undergoing investigation of congenital heart diseases, by a method previously described. These were studied in continuous motion as well as by single-frame projection with an analytic projector. Photographic prints and frame-by-frame tracings of the great vessels and heart chamber silhouettes offered simultaneous comparison of all phases of the mechanical cardiac cycle. Timing of the events within each cardiac cycle was made possible by Campeti's cinecardiometry, a method that produces curves depicting the dynamics of the various portions of the heart cham-
Figure 1. (Legend on opposite page)
OF electrocardiographic bers and great vessels. In many cases synchronous electrocardiographic tracings were correlated with the cineangiograms.

**Radiologic Findings**

Narrowing of the intrapericardial portion of the superior vena cava was frequently seen, as has been described by others, but most often during the *rapid ventricular filling* phase (fig. 1, no. 8). During *atrial systole* the size of the vessel remained unchanged or increased (fig. 1, nos. 2 and 10) according to the flow coming from its tributary veins and to the respiratory phase. In a few cases the sudden peripheral angiographic injection of contrast medium increased the flow and altered the normal hemodynamics.

Hedman, Lind, and Wegelius published an angiogram in the right anterior oblique position illustrating "narrowing of the superior vena cava simultaneously with atrial systole." However, we could identify in their serialogram not the morphology of the atrial systole but that of the rapid ventricular filling or of the diastasis, because atrium, atrial appendage, and subaortic sinus still appeared distended while the right ventricle was partially filled. According to our findings (fig. 2), these atrial structures decrease in size during rapid ventricular filling and diastasis (early and mid-diastole) but are not altered in shape. In fact, during these 2 phases the elastic atrial walls recoil uniformly. They contract only during atrial systole. Then the characteristic silhouettes of the atrial appendage and of the subaortic sinus suddenly decrease, deform, and cannot be identified (fig. 1, nos. 2 and 10).

The caval flow decreases or stops at the level of the atrium when the latter is filled (fig. 1, no. 7) immediately before the opening of the atrioventricular valves. A definite arrest of the flow also occurs during atrial systole and the following phase of isometric contraction (fig. 1, no. 10). The flow starts again to enter the atrial cavity at the beginning of the rapid ejection phase (fig. 1, nos. 3 and 11).

During the period of no flow, the superior caval orifice is well opacified by contrast medium and appears funnel-shaped, with the stem directed distally and the funnel mouth at the atrial vestibulum. During atrial systole the mouth of the superior caval orifice is well marked by the decreased angiographic density of the atrial cavity (fig. 1, no. 10 and fig. 3, C and D). The marked dilution of the contrast medium by nonopaque blood coming from the inferior vena cava and from the coronary sinus is responsible for the decreased density. This borderline of the superior caval orifice in the left anterior oblique position is almost rectilinear or slightly concave downward and oblique to the external wall from the right position of atrio caval junction (fig. 4, nos. 7 and 8) near the aortic angle. In the right anterior oblique position,

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*Fig. 1.* Frames sequence from a right anterior oblique cineangiogram recorded at 15 frames per second in a case of pulmonary valvular stenosis. These frames have been correlated with the cineangiometric tracings shown in figure 6. The pictures show a complete cardiac cycle: 2, atrial systole; ventricular systole: 3, isometric contraction; 4, 5, ejection; 6, end of systole; diastole: 7, A-V valves opening, and 8, rapid ventricular filling; 9, diastasis; 10, atrial systole. Atrial filling starts at the beginning of the ventricular systole and lasts until the opening of the A-V valves (3 to 7). The tricuspid valve ring appears outlined (4, 5, 6, 10, 11). The fused pulmonary valves, distended during ventricular ejection, and the hypertrophic outflow tract of the right ventricle form a mushroom-shaped silhouette (4, 5, 6, 11, 12). During atrial systole (2), at the end of the expiration (fig. 6), contrast medium refluxes into the inferior vena cava. This vessel and the atrial vestibulum between the 2 caval orifices appear distended, while the superior vena cava appears narrowed. This reflux is reversed at the beginning of ventricular systole (3). During inspiration (fig. 6) nonopaque blood from the inferior vena cava enters the right atrium and outlines the superior caval orifice, which is shaped like a clarinet mouth-piece (10, 11). During atrial systole, atrio caval junction appears narrowed (2, 10) according to the respiration phase (fig. 6).
this borderline is slightly concave toward the right and oblique from the atriocaval junction down to the angle formed by the venae cavae. In this projection the silhouette of the superior caval orifice is shaped like a clarinet mouthpiece (fig. 1, no. 10, fig. 2, right, and fig. 3, C and D). According to the anatomy of the right atrium, these borderlines follow the course of the tenia terminalis contracted during atrial systole. In the extreme left anterior oblique or in lateral projections this radiologic morphology of the superior caval orifice could not be identified in our cases. However, we could demonstrate that the superior atriocaval junction was slightly narrowed during atrial systole. According to Keith, this narrowing is due to the contraction of the right tenia terminalis, which passes through the anterior margin of the superior caval orifice, and of the septal band, which lies within the septal walls of the same orifice. "When the right tenia terminalis contracts it descends within the right auricle like the blade of a falcion." The anterior margin of the orifice is brought downward and inward until it approaches the septal walls of the right atrium, just in front of the aortic angle. We could not demonstrate in our cases the deep sphincterial contraction closing the superior caval orifice described by Keith. We think, thus, that the contraction of the right tenia terminalis and of the septal band may produce a change of the cross section of the superior caval orifice from a circle to an ellipse and a moderate decrease of the cross sectional area.

ANALYSIS OF THE CINEANGIOCARDIOGRAPHIC FINDINGS AND DISCUSSION

We analyzed the variations of the superior caval diameters on our cineangiograms by a graphic method. We realized that the superior vena cava is a valveless and collapsible vessel, subjected to the pressure variations of the mediastinum. The cross section of its mediastinal and intrapericardial portions probably varies from a circle to an ellipse according to the phase of respiration and to the phase of the mechanical cardiac cycle. Although these cross sectional variations might have affected the shape of our tracings, the intimate relationship between these tracings and the phases of the mechanical and electrical cardiac cycle.
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Fig. 3. Sequence showing right ventricular filling, from 15 frames per second right anterior oblique cineangiogram recorded in a normal subject 15 years old. A. End of ventricular systole. The right atrium is filled and the silhouette of the subeustachian sinus is distended. The A-V ring is outlined. B. (0.066 second later). The A-V valves are opened. Atrium and superior vena cava appear decreased in size. The ventricle is partially filled. Separate streams of opaque and nonopaque blood enter the right atrium respectively from superior and inferior vena cava (arrows). C. (0.066 second later). The ventricle is filled. The flow from the venae cavae is almost arrested. The caval orifices are outlined by the angiographic density of the right atrium. D. (0.066 second later). End of the atrial systole. The ventricle is completely distended, the atrium is contracted, and the diameters of the intrapericardial portion of the superior vena cava are increased.

was always evident, even in the cases in which the intravenous injection of contrast medium altered the caval flow.

To represent the dynamics of different parts of the superior vena cava,11 3 diameters were studied: the first was traced on the extrapericardial portion immediately below the azygos vein; the second was traced at the midpoint between the azygos vein and the atrium, on the intrapericardial portion; and the third, also intrapericardial, was traced on the atrial inlet of the vein. At this level the terminal half inch of the vessel is surrounded by loops of muscular fibers. According to Keith1 the convexities of these loops are thrown round the medial or left margin of the vessel and they terminate at the right or lateral margin near the lateral fornix of the vein.

The diameter of the atrial inlet of the superior vena cava varies synchronously and proportionately with atrial diameters and with the atrial silhouette area (fig. 5, tracing 3). The diameter increases suddenly during the ejection phase (tracing 3, a-b) corresponding to the onset of the QRS complex. At the end of the ejection phase (S-T segment) the tracing forms a small plateau (b-c) and then it continues to increase gradually (c-d) until the atrioventricular valves open (end of T wave). Afterwards the curve decreases to its lowest value (d-e) within the interval in which the 3 phases of the ventricular filling occur (rapid ventricular filling, diastasis, and atrial systole). On the tracing it is impossible to identify the atrial systole, in which one expects to find an indication of the contraction of the myocardial fibers surrounding the atrial inlet of the vein.

The diameter of the intrapericardial portion of the superior vena cava (fig. 5, tracing 2), traced midpoint between the azygos vein and the atrium, presents the same characteristic variations described for the atrial inlet diameter, although of less amplitude.

The extrapericardial caval diameter (fig. 5, tracing 1), traced immediately below the azygos vein, varies in a different pattern from that of the intrapericardial portion of the vessel. It decreases constantly after the ejection phase, corresponding to the S-T segment and the small plateau already described on
Fig. 4. Sequence from a left anterior oblique cineangiogram, recorded at 15 frames per second in a case of pure pulmonary valvular stenosis. 1. End of the atrial systole; the right atrium is contracted; the intrapericardial portion of the superior vena cava and the atrio caval junction are narrowed; the ventricle is filled and the distended outflow tract outlines the pulmonary valves still closed. 2 to 6. Ventricular systole and simultaneous atrial filling; the caliber of the superior vena cava increases and reaches the maximum before the opening of the A-V valves (6). The nonopaque blood coming from the inferior vena cava produces filling defects (4, 5, 6, 7, 8) that outline the superior caval orifice. The fused pulmonary valves (2, 3, 4) appear distended. 7 to 9. Diastole: with the opening of the A-V valves (7) the ventricle fills and the superior caval flow, almost arrested at the end of the systole (6), starts again to enter the right atrium (7). At the same time the caliber of the superior vena cava decreases. During atrial systole (1, 9) the distal portion of this vessel

(Continued on next page)
the tracing of the intrapericardial diameters (fig. 5, tracing 3, b-e). Then the diameter increases until the opening of the atroventricular valves (end of the T wave). At this point it decreases again, and the amount of decrease depends on the respiration phase and the flow of the contrast medium. Atrial and ventricular systoles are sometimes marked on the tracings by peaks of small amplitude (fig. 6), which are slightly delayed with respect to their electric manifestations (fig. 5).

This graphic analysis of the variations of the superior vena caval diameters demonstrates that the intrapericardial and extrapericardial portions of this vessel are influenced differently by respirogenic and cardiogenic factors.\(^\text{12}\) Respirogenic factors have more influence and thus affect all portions of the vessel as well as the right atrium. During inspiration all of the superior caval diameters and the area of the right atrial silhouette increase in both systole and diastole. The reverse occurs in expiration (fig. 6). The cardiogenic factors affect directly the intrapericardial portion of the vein but influence indirectly the extrapericardial portion (figs. 5 and 6).

Recent studies\(^\text{12}\) have demonstrated that the descent of the atroventricular junction during ventricular systole enlarges the great venous reservoir, namely the atrium and venae cavae. The piston-like downward movement of the atroventricular junction draws blood of the central veins into the right atrium. However, the acceleration of the venous flow during ventricular systole can be decreased by opening of the pericardium or by removing the support of the heart in other ways.\(^\text{12}\) We therefore think of the cyclic variation of the intrapericardial pressure as an influential factor along with the cardiogenic one. This also could explain the fact that the intrapericardial portion of superior vena cava, which does not contain myocardial fibers, varies in a manner (fig. 5, tracing 2; fig. 6, tracing 3) similar to the area of the right atrium and to the diameter of the atrio caval junction.

In many cineangiograms, normal and pathologic, in which the contrast material was injected through the vein of the arm, the reflux of medium into the inferior vena cava was observed during the atrial systole. Although the intrapericardial portion of the superior vena cava was narrowed, the intrathoracic portion of the inferior vena cava was filled and widely distended. The atrial vestibulum between the 2 caval orificies was not contracted and was clearly distinguishable from the shadows of the atrial structures (fig. 1, no. 2). Gross observation and cineangiometric analysis\(^\text{10}\) (fig. 6) correlated with serial photographic enlargements (fig. 1, nos. 2 and 10) demonstrated that the reflux into the inferior vena cava during atrial systole occurred only in expiration. In other cineangiograms, in which the contrast medium was injected peripherally through the inferior vena cava, the small reflux into the superior vena cava, during atrial systole, was observed only during inspiration. This finding leads us to believe that the intrathoracic portion of the inferior vena cava is not influenced by the cardiogenic factors to the same extent as is the superior vena cava. The fact that the medium reflexes into the superior or inferior vena cava, according to the cyclic respiratory changes of the pressure gradient between these 2 vessels,\(^\text{12}\) proves that the 2 venae cavae are in communication during the atrial systole through the atrial vestibulum (fig. 1, no. 2) and through the narrowed but not closed caval orifices of the right atrium. The "closure of the caval orifices" or, in other
words, the forward progression of the blood during atrial systole depends upon the pressure gradient between the venae cavae and right atrium and between the right atrium and ventricle.

Concerning the mechanics of the caval orifices in preventing the regurgitation of the blood into the venae cavae during atrial systole, we cannot agree with Keith and with Kjellberg and Olsson. The fact that the caval orifices are surrounded by myocardial bands that contract during atrial systole, as does all of the atrial myocardium, is not a matter of discussion. The question is about the degree of narrowing. Direct radiographic signs of the contraction of these bands in cases of right atrial hypertrophy, and indirect signs produced by the characteristic stop of contrast

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**Fig. 5.** Caliber variation of the superior vena cava recorded by cinecardiometric method from a 30 frames per second left anterior oblique cineangiocardio gram. Caliber changes are expressed as a percentage of the arithmetical average (base line = 100 per cent) computed by measuring at the same point the diameters in 80 consecutive frames. The tracings are correlated with electrocardiograms recorded synchronously with the cineangiocardio gram, with the time in seconds and with the frames. This figure demonstrates that the extrapericardial portion of the superior vena cava (tracing 1: diameter traced immediately below the azygos vein) varies differently from the intrapericardial portion of the vessel (tracing 2: midpoint between azygos vein and right atrium; tracing 3: atro caval junction).

**Fig. 6.** Right heart dynamics recorded from the same cineangiocardio gram as in figure 1. These tracings obtained by cinecardiometric method as the tracings in figure 5, are correlated with the time in seconds and with the frames. The numbers 1 to 12 on the small scale at the bottom correspond to the pictures in figure 1. These tracings show the influence of the respiratory phases (1: diaphragm movements) and of the cardiac cycle (5, 6: respectively areas of right atrial and ventricular silhouettes) on the extrapericardial portion of the superior vena cava (2: diameter traced immediately below the azygos vein) and on its intrapericardial portion (3, 4: diameters traced respectively at midpoint between azygos vein and atrium and at level of the atrio caval junction).
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medium, have been observed as reported in the foregoing. However, the deep contraction which could be interpreted as a sphincterial closure of the caval orifices was not found. Nevertheless, the mechanical and hemodynamic theories of the "closure" of these orifices are not mutually exclusive.

It has been calculated\textsuperscript{12} from a modified formula of Poiseuille's Law\textsuperscript{13},\textsuperscript{14} that "volume flow through partially collapsed veins obeys approximately the laws of flow through tubes of an elliptic cross-sectional area." The flow through these tubes decreases in proportion to the minor diameter of the cross section, when the pressure and the cross-sectional perimeter are kept constant. Under the same experimental conditions, a decrease of 50 percent in the minor diameter reduces the flow to one third of that through the same tube but of circular cross section. By analogy, we believe that relative reduction of the cross-sectional area of the caval orifices and a concomitant change of the cross-sectional shape from circular to elliptic produces a zone of great resistance to the flow. This would prevent the caval reflux during atrial systole, or minimize it when the pressure gradient between right atrium and venae cavae is altered. The fact that the peripheral injection of contrast medium\textsuperscript{15} produces a small increase in atrial pressure (1 or 2 mm. Hg) and often a reflux into the contralateral vena cava, even in normal cases, suggests that this reflux may occur also under physiologic conditions as a mechanism to prevent right atrial overfilling. The flow through superior or inferior vena cava is subject to increase suddenly in many of the daily physiologic acts that produce abrupt changes of the intrathoracic and intra-abdominal pressure. The venae cavae, then, by reciprocal shunting, act as a pressure-balancing blood reservoir for the right atrium. This should allow a gradual adjustment of the cardiac output to a sudden variation of the venous return.

CONCLUSION AND SUMMARY

The physiopathology of the "closure" of the venous orifices of the human right atrium has been studied in normal and pathologic cases by an analysis of high-speed cineangiocardio graphic records.

Theory of myocardial sphincter-like closure and theory of functional hemodynamic closure could not be proved entirely. Mechanical and hemodynamic factors interact in closing the caval orifices during atrial systole. That caval orifices narrow during atrial systole but do not close can be demonstrated by the fact that contrast material refluxes into the contralateral vena cava during atrial systole, when, for physiopathologic causes, the gradient of pressure between the venae cavae and the atrium is altered. It is assumed by the authors that along with this relative incompetence there exists an atrial mechanism to prevent a sudden overflow from the superior or inferior parts of the body and to allow gradual adjustment of the cardiac output to the venous return.

The influence of respirogenic and cardiogenic factors on the venae cavae dynamics is demonstrated.

ACKNOWLEDGMENT

The authors wish to acknowledge the generous help of Dr. Lee B. Lusted in the preparation of the manuscript.

SUMMARIO IN INTERLINGUA

Le physiopathologia del "clauditura" del orificios venose del atrio dextere in humanos eseva studiate in casos normal e pathologic per le analyse de registrationes cineangiocardio graphic a alte rapiditate.

Le theoria de un clauditura per action sphincteroide del myocardio e le theoria de un clauditura hemodynamic functional non poteva esser provate completely. Factores mechanic e hemodynamic interage in effectuar le clauditura del orificios caval du rante le systole atrial. Que le orificios caval se restringe durante le systole atrial sed non se claude poteva esser demonstrate per le fac to que substantia de contrasto retrofllue a in le vena cave contralateral durante le systole atrial quandno, ab causas physiopathologic, le differentia de pression inter le venas cave e le atrio se altera. Le autores postula que—a
parte le incompetentia relative—il existe un mechanismo atrial que preveni un subite surfluxo ab le partes superior o inferior del corpore e que permitte un adjustment gradual del rendimento cardiaco al retorno venoso.

Es demonstrate le influencia de factores respirogenic e cardiogenic super le dynamica del venas cave.

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THE TORCH BEARERS

ALFRED NOYES

English poet, 1880-

From the Epilogue

The records grow

Unceasingly, and each new grain of truth
Is packed, like radium, with whole worlds of light.

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Circulation. 1959;19:55-64
doi: 10.1161/01.CIR.19.1.55
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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