The Dynamic Function of the Atrial Diastole of the Heart and Motion of Venous Valves in Humans

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Abstract

This study demonstrates dynamic function of atrial diastole for the first time in the history of medicine, following the revelation of ventricular function by Sir William Harvey. This study consists of two parts. First part is the study of humans and the discovery of the rhythmic opening and closing of venous valves in the femoral vein segment during each cardiac cycle under fluoroscopy. Its relationship to the right atrial diastole is discussed. Second part is an experimental model developed in a dog's heart. Experiments are conducted in an erect posture, in which the right atrium is partially and totally isolated physically and/or functionally. The right atrium was found to function as a suction pump, readily demonstrated on graphic curves by voluntary stretching of muscle fibers of the empty atrium, creating considerable negative pressure during diastolic function, and responsible for venous return to the heart. Motive forces in the venous return are common knowledge in present medical literature, but are limited to respiration and skeletal muscle contraction of the extremities. The discovery of the right atrium as a suction force due to stretching of muscle fibers during diastolic function, thereby creating negative pressure, represents a departure from the currently accepted paradigm established by Sir William Harvey (1576–1652), with the heart as an organ functioning solely by contraction of its chambers. This is the first time since then a fundamental new discovery concerning cardiac mechanical function has been made utilizing experimental evidence.

Keywords

- ▶ atrial diastole
- ▶ venous valves
- ▶ femoral vein
- ▶ cardiac cycle
- ▶ diastolic function
- ▶ mechanical function
- ▶ motion
- ▶ phenomenon
- ▶ muscle fibers

For more than 1,300 years, Galen's views (130–201 AD) dominated medical opinion, and entire generations of physicians were unquestioning obedient students of his teaching. That is the reason why the entry of Sir William Harvey (1574–1652 AD) into the medical world becomes such an important event in the history of modern medicine.

Galen observed all ingested nutrition enters the gastrointestinal system, transformed into blood, draining from the intestines to the liver, from which it is distributed to various parts of the body. Hence, he preached the liver is

the main organ of circulation. Harvey's life-long experiments on various animal hearts ultimately resulted in the following discoveries, which remain unchanged to this day. It¹ includes: The heart is the center of circulation; ventricles are the forward pushing pumps and are the only force keeping the blood in motion in the human body. Circulation is a continuous phenomenon and not optional as thought until then, and blood leaves the ventricles and comes back to the atrium, moving in a circular fashion within the body.

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Fig. 1 Present concept of circulation. In the diagram, A represents atrium and V represents ventricle. Ventricular systolic force pushes the blood forward into the arteries. This force is exhausted by the time it reaches the capillaries. Blood is thought to move in the veins back into the atrium by respiration and peripheral muscular contraction. At present, the atrium is considered only a passive receptacle, and the heart is just a forward pushing pump.

Both Galen and Harvey, however, did not understand one of the most important functions of blood to carry oxygen from the lungs to various parts of the body, and carbon dioxide from tissue back to the lungs. Harvey had not discussed any functional role of the atria: the atria, long considered passive, and the collecting chambers of the heart where venous blood returns and pools. Harvey thought the forward pushing force of the ventricles was adequate enough to keep blood in motion within the arteries and veins, and return it to the atria.

Some of Harvey's opinions have now come into question. It has been voiced the force imparted by the ventricles to the ejected blood is not sufficient enough to explain the venous return to the atria. The nature of other forces contributing to venous return has been a matter of heated discussions for centuries. At present, two of the forces in the human body that play an important role in the motion of venous blood have been well established. First, the skeletal muscle contraction, which is primarily the calf muscle pump, and is known as the peripheral heart.² Second, Bollinger, in 1971, clearly established the role of respiration in the circulation of blood.³ The force responsible for venous return to the atria is not been explained. Scientists preceding Harvey, as well as those following him, theorized forces contributing to the motion of blood under the broad headlines of *vis-a-tergo* (a force from back by ventricular motion) and *vis-a-fronte* (a force from front by ventricular diastole). None, however, gave credit to diastolic dilatation of the atrial chambers of the heart.⁴

In reviewing past literature, only Wedemyer's⁵ concept alluded to the idea of auricular dilatation, causing an aspirating effect on venous blood. With no experimental evidence backing this hypothesis, Donders⁵ soon discarded it. In the book *Human Physiology* by Houssay,⁶ it is mentioned that during ventricular contraction, intra-auricular pressure falls,

having a slight aspirating effect on blood from the veins. Houssay states it has little or no functional significance. It always had been thought that systole of the ventricles and atria are due to contraction of muscle fibers, and considered to be active and dynamic, and the diastole was always thought to be the phase of relaxation and adynamic. The present concept of motion of blood in the human body is illustrated in **Fig. 1**. In the diagram, "A" represents atrium and "V" represents ventricle. Ventricular systolic force pushes the blood forward into the arteries. This force is exhausted by the time it reaches the capillaries. Blood is thought to move in the veins back into the atrium by respiration and peripheral muscular contraction. At present, the atrium is considered only a passive receptacle, and the heart is just a forward pushing pump.

Materials, Method, and Results

Study on Humans

In 1987, when performing a descending phlebography⁷ on a patient to check the competency of the venous valves, this author identified a strange phenomenon for the first time, and later observed this in many patients, and recorded it by videotape. The author has performed 200 descending phlebographies. This procedure was performed in the X-ray suite under fluoroscopy on a tilted table.

We positioned a 67-year black male in a recumbent position (flat on his back). Under local anesthesia, we introduced an 18-gauge angio-catheter into the right internal jugular vein, through that, a guidewire 0.032 inches in diameter, and advanced to the area of the right femoral vein. Along the guidewire using Seldinger's technique, we introduced a 6-F size 100-cm-long catheter into the femoral vein. Upon removing the guidewire, contrast was hand injected with a 60-mL syringe in small doses of 10 mL each. This enabled visualization of the venous valves in this region. Recorded motion of the venous valves is performed while the patient is in the recumbent position and at an 80° upright position.

The motion of the valves was in time-phase with the electrocardiogram of the patient. The valves, visualized, were located in the common femoral vein, proximal portion of the superficial femoral vein, profunda femoris vein, and great saphenous vein. The venous valves simultaneously open and close, and were in synchronized rhythm to each cardiac cycle. In a patient with 69 cardiac beats/min, the motion of the venous valves corresponded to each cardiac cycle, including missed beats. The motion of the valves is least obvious while the subject is lying down, and becomes very prominent in the erect position (→ **Fig. 2**) demonstrating a continuous cine recording of this motion. The first frame in the figure is 119 and last is 130, for a total of 12 frames. Each frame is timed, and recorded. Frame 119 begins at 15:52:31 (hours : minutes : seconds), and frame 130 ends at 15:52:34. The duration of run time is close to 3 seconds. In a little less than 3 seconds, the femoral valves open and close 3 times. A constant and rhythmic motion occurred during each cardiac cycle. A review of the literature, unequivocally mentions the

Fig. 2 This is a continuous cine recording or approximately four and one-half frames per second, consisting of a total of 12 frames. Frame no. 119 begins at 15 hours 52 minutes, and the last phase of the 31st second frame no. 130 ends at the middle phase of the 34th second. During the approximate 2.75 seconds, the heart rate is recorded as 69/minute beats three times. During each cardiac cycle, the femoral vein valves open and close as noted in frame nos. 119, 122, 126, and 129. Valves open four times. Frame nos. 121, 124, and 127, the valves close three times.

presence of venous valves was noted first in the work of Charles Estienne in 1539.⁸ In accord with present knowledge, the venous valves are “one-way” doors, and their only function is to keep the blood flowing in one direction—toward the

heart. The discovery reported in this article, the opening and closure of the venous valves in humans, and its relationship to the atrial diastole and systole, and its variation as it relates to recumbent and upright positions, has never been described in past medical literature. Our findings led us to conduct a series of experiments in dogs,

Study on Dogs

Experiments conducted in five Mongrel dogs with an average weight of 55 lbs. Because gravitational force plays a role in the atrial function, dogs were kept in a semi-upright or upright

Fig. 3 The experimental model fixed to the wooden board is kept in an upright position during the experiment.

Mongrel Dog, Weight 55 lb. Under general anaesthesia with endotracheal intubation.

Fig. 4 Experiment I. Continuous and simultaneous recording of three monitors. Upper graph is the EKG (heart rate of the model is 124/min). Femoral arterial pressure is recorded as 120/80 mm Hg. The lower graph is the model's right atrial pressure. The catheter is deliberately pushed from the right atrium to the right ventricle. It is brought back, and affixed to the right atrium. The right atrial diastolic pressure is -2 mm Hg.

Fig. 5 Experiment II. The superior vena cava is ligated, and the atrial venous return is partially cut off. This results in an immediate increase in atrial diastolic negative pressure up to -14 mm Hg.

position. Following intubation, we administered general anesthesia to each dog, and placed each in a horizontal position on a wooden board, rectangular in shape, and sideboards for the upper and lower extremities. Monitoring lines were placed, including a femoral arterial line for the continuous monitoring of arterial pressure with graphic tracing. A catheter was inserted through the jugular vein and into the right atrium, connected to a pressure monitor through a transducer to record the atrial pressure changes during the experiment. An electrocardiographic (EKG) machine was used for continuous EKG monitoring. Experiment I is conducted in all dogs. Experiment II is conducted in the same dog several times recording the same finding. Experiment III is conducted in two dogs. Experiment IV was conducted in two dogs.

Presently established forces contributing to the motion of venous blood (**Fig. 1**) were neutralized utilizing the following method. We paralyzed the dog with muscle relaxants to prevent any twitching of skeletal muscles during this experiment. In doing so, this excludes the muscular contraction aiding the venous flow in the body. A vertical midline incision was made from the xiphoid notch to the pubic tubercle, keeping the thoracic and abdominal cavity open. We then transected the diaphragm with an anterior-posterior incision toward the vertebral column. In doing so, this excludes the changes in the intrathoracic and intra-abdominal cavity pressure from contributing to venous blood flow by respira-

tion. The wooden board with the dog was tilted upward and kept against the wall of the room (**Fig. 3**). This kept the dog in an erect position during the rest of this experiment.

In Experiment I done in all five dogs as a preliminary step, **Fig. 4** shows (a) the electrocardiogram indicating the sinus rhythm with a cardiac rate of 124 minutes; (b) the beginning of the graphic curve recording the pressure in the right atrium, $7/-2$ mm Hg.

A catheter is then advanced to the right ventricle. Of note, is the increased height of the graphic curve and rising pressure to $25/6$ mm Hg. The catheter was then withdrawn from the right atrium, resulting in restoration of the graphic curve and pressure of $7/-2$ mm Hg. This experimental maneuver assured the placement of the catheter in the right atrium during the remainder of the experiment. This maneuver utilized for each of the subsequent experiments, with the catheter fixed in the right atrium.

In Experiment II, done in two dogs (**Fig. 5**), the dog is maintained at a 60° erect position. In this experiment, changes of the right atrial pressure were studied by partial exclusion of the atria, ligating the superior vena cava using umbilical tapes over a Rommel clamp. We recorded the immediate changes. The arterial pressure progressively began to fall within a few seconds, and threatened to reach a non-recordable level if the experiment continued. Meanwhile, the graphic tracing in the right atria demonstrated a remarkable change, by increasing three times in size, with a negative pressure reaching a -14 mm Hg. At this stage, the experiment was discontinued by relaxing the ligature on the superior vena cava due to a rapid fall in arterial pressure.

In Experiment III, done in two dogs (**Fig. 6**), the right atrium was totally excluded by simultaneously ligating both the superior and inferior vena cava utilizing the same technique as described above. The immediate observation, indicated within a few seconds of ligation, the arterial pressure reached a non-recordable level, and the atrial chamber went into fibrillation. When we continued the experiment, the dog went into cardiac arrest. We were unable to repeat this particular experiment on the same dog, due to either cardiac arrest, or the dog remained in atrial fibrillation following the initial experiment. It was impossible to interpret the ongoing events during this particular experiment. The graphic tracing of events and atrial pressure changes were continuously recorded for further in depth analysis and interpretation. We recorded the following observations. When the total venous return to the right atria was suddenly stopped, the immediate response became apparent within the first three beats. When the venous return was stopped the graphic tracing increased to five times its original size, and the atrium produced a negative pressure to -24 mm Hg. This astonishing finding, revealed for the first time, the atrium was capable of producing a remarkable negative pressure depending upon the venous return, indicating it could exert a very strong suction force during diastole. This suction force was of a potential and intermittent nature. However, this negative pressure was unsustainable for a longer period of time in this experimental model due to the sharp fall in blood pressure. Each result indicated cardiac arrhythmia and arrest.

Fig. 6 Experiment III. Both superior and inferior vena cava are simultaneously ligated. The venous return to the right atrium is completely cut off. The empty atrium shows diastolic dilatation. Reaching its peak in the first 2–3 beats immediately following ligation, slowly decreasing to a flat curve within a few seconds. Experiment records the maximum diastolic negative pressure of -24 mm Hg. Diastolic dilatation of the empty atrium establishes for the first time, the diastole is a voluntary dynamic stretching of atrial muscle fibers.

We developed the following experimental model in which the venous return was suddenly, and totally, cut off, but at the same time, cardiac output was maintained by alternate means. This experimental model is explained in Experiment IV (►Fig. 7) done in one dog. This experiment had the dog at a 90° erect position. A glass jar (2,000 mL graduated cylinder; Kimble USA, Rockwood, TN) filled with ringers' lactate solution, and connected to the right atrium of the dog through an 18-French silastic tube. The silastic tube was connected to the right atrium through the inferior vena cava via a venotomy. Once the tube was inserted, the venotomy was closed using a purse string 3–0 silk suture to maintain a watertight opening. The silastic tube was filled to the tip with the ringers' lactate solution, and clamped during insertion into the atrium to prevent an air embolism. The jar of ringers' lactate and the tip of the silastic tube were maintained at the same level during the experiment, by maintaining the jar on a wooden stool of appropriate height. During this experiment, both the superior and inferior vena cava were clamped simultaneously, while the clamp on the silastic tube was relaxed. In ►Fig. 5 the graphic tracing in the right atrium tripled in size, and a recorded pressure of -17 mm Hg. We further observed the continuous passage of ringers' lactate solution from the jar to the right atrium. We were able to continue this experimental

model, without the atria going into fibrillation. The flow of ringers' lactate from the jar to the right atrium continued until the fluid level in the jar dropped to 5 cm below the original level, at which point, the flow from the jar toward the atrium stopped. This established the limitation of the atrial suction force and demonstrated the pressure gradient enabled the fluid to flow against gravitational force. We were able to immediately repeat the experiment in the same dog by raising the jar manually, and returning the level of ringers' lactate to its original level. Repetition of the experiment was unable to continue after a few trials due to a sharp fluid overload and cardiac failure.

Discussion

Findings on human beings: depolarization of the ventricles gives rise to the QRS complex.⁹ This main event demonstrated on the EKG by a blinking light and a beeping sound, occurred at the peak of the "R" wave. Hence, we are able to time the opening of the venous valves to the exact segment in the EKG recording, and thereby associate it with a particular part of the cardiac cycle. We observed the valves begin to open immediately after the blinking light, followed by the QRS complex, or after the ventricular systole and before the

Fig. 7 Experiment IV. Superior and inferior vena cava are ligated. The right atrium is connected to a glass jar filled with ringers' lactate solution through a no. 18 F size silastic tube. The atrial diastole produces a suction force up to -17 mm Hg, suctioning the solution from the jar during each diastolic expansion until the level of solution within the jar reaches 5 cm below the original level.

beginning of the P-waves, before the atrial systole, and coinciding with the atrial diastole.

Findings on dog's experiments: What causes valvular motion? Is it a primary or secondary event? Following our experimental findings, we can interpret atrial diastolic dilatation of muscle fibers is an active phenomenon (Experiment III). It creates negative pressure by suctioning blood from the inferior vena cava into its empty chamber. This triggers the motion of the blood column in the entire venous tree and the venous valves open passively. During systole of the atrium, the chamber collapses onto itself and negative pressure is lost. Thus, the suctioning function ceases. Due to gravitational force, which is of diametrically opposing the atrial diastolic suction force, the entire column of blood in the venous tree tends to flow in the opposite direction (downward toward the feet). As the venous valves are "one-way" doors opening toward the heart, the weight of the column of blood flowing downward closes the valves. Therefore, during each cardiac cycle, venous valve cusps open during atrial diastole and close during systole. Atrial diastolic suction force is related to the volume of venous blood return. Hence, this force can vary during different positions of the body, and is related to the variation of gravitational force. Although the motion of venous valves is present in all positions, it is the least obvious in a supine position, and is at its maximum in the upright

position, since it is in this position that gravitational force is at its peak. It was precisely this reason as to why the experimental models were created in unique upright positions.

A review of the literature shows the maximum diastolic pressure ever recorded in a dog's right atrium is -4 mm Hg. In healthy humans, the minimum right atrial diastolic pressure ever recorded is 0 mm Hg. Guyton explains, very close to the tricuspid valve a pressure of -2 mm Hg can be recorded due to the downward motion of the valves during ventricular systole. This does not provide any functional significance.

In Experiment II, by clamping the superior vena cava, the venous return to the atria is partially cut off and its effect is immediately reflected in the graphic tracing. The height of the graphic curve (anacrotic and dicrotic notches) increase remarkably, and the negative pressure falls to -14 mm Hg. This is a significant change in the negative pressure; seven times lower than the resting pressure recorded in the experimental model in the recumbent position. This can be produced only by an extra dilatation by corresponding dynamic stretching of the atrial muscle fibers during diastole. In turn, this results in a more forcible collapse of the atrium during systole, increasing the positive force to 24 mm Hg. This is noticed only for a few beats, due to the clamping of the inferior vena cava, thereby causing a decrease in venous return, and results in a sudden fall in cardiac output. As a consequence, arterial

pressure begins to fall rapidly, and within a few seconds the experiment had to be stopped.

In Experiment III, the entire venous return to the atrium was cut off by simultaneously ligating the superior and inferior vena cava. Effects of this experiment were interpreted by evaluation of graphic tracing, and recording of the atrial pressure. Within seconds, the arterial pressure reached a level not recordable, and the experimental model's heart went into cardiac arrest following cardiac arrhythmia.

During this experiment, the lowest atrial pressure recorded was -24 mm Hg. Immediately following ligation of both vena cava, the graphic tracing indicated the highest graphic curve during the first 3 beats, progressively falling to a flat line within 10 to 12 beats. The atria reached peak dilatation during diastole during the first three beats, indicating its maximum reaction to the lowest venous return.

Starling's laws of muscle contraction states the law of the heart is thus the same as the law of muscular tissue. Since then, it is a common belief that systole is an active contraction, and diastole is a resting period of the heart.¹⁰ By these experiments, we have established it is not true for atrial muscle fibers, which contract during systole and voluntarily stretch during diastole. An empty right atrium records few beats of maximum diastolic stretching of its muscle fibers. Thus, the reason as to why it produces maximum negative pressure by maximum stretching of its muscle fibers even when the venous return is completely cut off. Proving for the first time, the diastole of the atrium is voluntary, and conscious stretching of its muscle fibers as it reacts according to venous return. It also disproves our age-old thinking that diastolic distension is simply due to the venous filling of the chamber, and if there happens to be no venous return, then the atrial chamber would remain a collapsed bag, similar to that of a collapsed pillow cover when the pillow is removed from within. This experiment also establishes the fact that the atrial chambers react to venous return, and are capable of changing the negative pressure to exert the maximum suction force when venous return fails. The negative pressure reaches its maximum force when the venous return falls to its lowest level. It cannot sustain this action for a long period of time, as this experiment is not compatible with life as total venous return is cut off during the experiment. So we developed the next model. Experiment IV was conducted in dog at 90° erect posture. The venous return is cut off by simultaneously ligating the superior and inferior vena cava at the same time the clamp on the silastic tube is released, the atrial suction force is adequate enough to suck the ringers' lactate solution from the jar. Thus, cardiac output can be maintained for a longer period of time without the immediate fall in arterial pressure. Graphic tracings demonstrate sustained negative pressure exists. The ringers' lactate solution continues to decrease until it reaches a level of 5 cm lower than the original level. The original level is exactly at the level of the tip of the catheter in the atrium. The reason as to why the passage of ringers' lactate from the jar to the atrium stops upon falling to 5 cm below the original level establishes the fact that atrial suction force has limitations.

During this experiment, we were able to maintain the model's arterial pressure for a longer period of time, with steady graphic tracing, when the negative pressure of suction force in the atrium reached a -17 mm Hg. When the ringers' lactate level fell below 5 cm of the original level, the passage of fluid ceased. To continue this experiment, we had to manually raise the entire jar to bring the level of ringers' lactate to the level of the tip of the silastic tube in the atrium. It was possible to repeat this experiment in the same model. However, we could only repeat the experiment a few times, due to the fact that the cardiopulmonary system became overloaded with ringers' lactate in a short period of time.

Conclusion

Atrial chambers have a dynamic function. They are not passive chambers. Collecting tanks of venous blood as presently thought. The atrial chambers are strong suction machines dictating the venous return to the heart. This is the main force in the body creating motion of venous blood in the venous tree. Suction force is exerted intermittently during each diastolic expansion of the atria. This suction force is converted to a continuous pump due to the presence of venous valve cusps. The venous valve cusps can open only in one direction, thereby, maintaining venous blood continuously in motion, in one direction, toward the heart.

The four chambers of the heart have separate functions. Ventricles are forward pushing pumps. The left ventricle propels blood into the systemic arteries, and the right ventricle pushes blood into the pulmonary arteries. The right atrium draws blood toward the heart from systemic veins, while the left atrium similarly draws blood from the pulmonary veins. In systemic venous circulation, contraction of skeletal muscles and quiet respiration, acts as adjunctive forces assisting the motion of venous blood.

In summary, this article reveals, through experimental evidence, the following three discoveries:

1. The human heart is a dual machine, forward pushing ventricles, and backward suctioning atria.
2. The diastolic phenomenon of the heart is due to active stretching of its muscle fibers.
3. Venous valves in humans open and close during each cardiac cycle.¹¹

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