Critical Review of Recent Work on Ventricular Diastolic Suction

By GERHARD A. BRECHEK, M.D., PH.D.

Unequivocal evidence in favor of the concept that a ventricular diastolic vis a fronte contributes to ventricular filling has been established only in the following conditions:

a. When the ventricle contains an abnormally small residual volume (rat, dog, turtle);

b. When the ventricle contains a more nearly normal, residual volume due to ejection against the resistance of a fluid column (dog, turtle).

The quantitative contribution of the ventricular vis a fronte to ventricular filling at various residual volumes and various levels of cardiac activity is still unknown. The physiological significance of diastolic suction for the return flow of blood can therefore not yet be evaluated.

A new era of interest in the age-old problem of intraventricular diastolic suction began in 1952 with a flurry of publications from various parts of the world. Some of these were more or less conceptual or theoretical, whereas others were predominately experimental studies in which some attempts were made to approach the problem directly. This renewal of interest was preceded by nearly two decades of quiescence (1934 to 1952). During this period it was generally agreed that there was no evidence for the existence of ventricular diastolic suction, since, according to Straub, Wiggers and others, negative ventricular diastolic pressures had never been reliably recorded. This opinion was based on the work of Cotton in 1934, who showed that the apparently observable, negative pressures in the turtle ventricle, reported in 1930 by Katz, resulted from the specific experimental arrangements used. Furthermore, the elegant experiments of von den Velden in 1906 demonstrated that fluid in the dog was not sucked through a cannula from a reservoir situated below heart level into the left ventricular cavity.

According to an extensive review of the older literature by Ebstein and a brief one by Brecher, very few problems in medicine have caused as much controversy as diastolic suction, or involved such strong emotional reactions. This is the case even nowadays, as may be gleaned from an appropriate remark in a recent book review: "Whatever role cardiac suction of venous blood may play in determining circulatory dynamics, no one can deny that mention of this term has proven a most effective method of raising blood pressure in several generations of cardiovascular physiologists." Several factors account for this situation. Precise information about all forces which cause ventricular filling was not obtainable in the past and is still very scanty today. In the absence of reliable information there has been much speculation and wishful thinking about the probable or possible forces involved.

The purpose of this article is to review critically the evidence provided by recent work concerning this topic and to summarize the present status of the problem.

The Postulate

It has been postulated by numerous cardiovascular workers that intraventricular diastolic suction somehow contributes to ventricular filling. Often the same idea is expressed by the use of the terms "active diastole" and "diastolic vis a fronte." This postulate has been based mainly on reasoning from very meager evidence or even no evidence at all. The line of reasoning by the adherents of the "active diastole" concept is approximately as follows: It must be assumed that suction exists, because if it did not it would be hard to explain how the ventricle fills so rapidly during early diastole. In the absence of suction,
it would be even harder to explain ventricular filling in the human being during tachycardia and in exercise. If suction did not exist, it would be also difficult to conceive how the ventricles of small mammals and birds could be filled adequately during the exceedingly short diastolic periods in the presence of fast heart rates, such as 600/min. in the mouse or 800/min. in the hummingbird. All of this may sound logical, but active diastole will remain a postulate until it is established by unequivocal, experimental evidence.

**Terminology and Problem Analysis**

The terms intraventricular "suction," "vis a tergo," and "active diastole" have often been applied loosely and interchangeably with various connotations. It is therefore necessary to define or describe the meaning of these terms. Suction is not something mysteriously different from pressure, but represents a point of view concerning a pressure difference. In order that a fluid flow, a pressure difference (gradient) must exist. If this pressure difference is produced by raising the pressure at a point relative to the rest of the fluid, one speaks of "positive pressure." One sometimes thinks of the fluid as being pushed by this positive pressure. On the other hand, if the pressure difference is set up by lowering the pressure at a point relative to the rest of the fluid, one speaks of negative pressure or suction. One thinks of the fluid in this case as being drawn or pulled by the negative pressure. It is most important to realize, however, that it is in every case the pressure difference which provides the motive force. For the creation of this force, energy must be expended. Applied to the ventricle, this would mean the following: During diastole the ventricle, the atrium and the adjoining veins form a common cavity. If the pressure in the ventricle is lowered by some force below that which would exist in the ventricle in the absence of such force, then one may call this phenomenon suction. The lowering of the pressure in the ventricle would result in flow of blood from the atrium and veins into the ventricular cavity. It is clear, from a physical standpoint that the problem of intraventricular diastolic suction hinges on the question of whether or not, under physiological conditions, such lowering of the pressure occurs, how large it is, how long it lasts and how much flow it could produce.

The forces which cause the movement of blood in the circulation have been divided traditionally into the *vis a tergo* and the *vis a fronte*. The *vis a tergo* is the force from behind, which is imparted to blood to propel it from the ventricles though the systemic or pulmonary circulation back into the ventricles. The *vis a fronte* is the frontally acting force, which draws blood from various parts of the circulatory bed toward the ventricle. Three different frontal forces, known to act in the central venous system and heart, can draw blood toward the ventricle. Each force lowers the pressure in a segment of the system below the pressure which would exist in the absence of the force. The first is thoracic aspiration, by which blood flows from the extra-thoracic into the thoracic veins. The second, systolic ventricular *vis a fronte*, is produced by ventricular contraction, and results in a lowering of atrial pressure during ventricular systole, contributing thereby to atrial filling. The third, diastolic ventricular *vis a fronte*, is the force which concerns ventricular filling directly. It results in a lowering of intraventricular pressure during diastole under certain experimental conditions. This is the frontal force with which this review is concerned.

As concluded from the general usage, active diastole is a physiologic term for conveying the functional concept that during diastole the ventricle itself contributes by some means to its own filling. The word "active" does not necessarily refer to muscle contraction, but may merely refer to the existence of some force or forces in the ventricle during diastole which result in ventricular filling. Active diastole is contrasted to passive diastole. The latter refers to ventricular filling in which the ventricle walls are pushed apart and the
cavity is enlarged solely by a *vis a tergo*, without energy release by the ventricle during diastole. The forces responsible for active diastole may stem from several energy sources. Some authors (Bracht, Magendie, Krehl, Dürk, cited by Villa and Guasp) have suggested that specific muscle fibers contract during diastole and expand the ventricle. Others have suggested that blood, entering at the end of systole into the coronary arteries, caused a slight expansion of the ventricles. Still others have expressed the belief that a deformation of some components of the ventricle during systole furnish energy for an elastic recoil during diastole (see Epstein). The idea that an elastic recoil develops during diastole has been recently brought into focus by detailed discussions of Rushmer, who introduced the valuable concept of "interfascicular tension." (see also Rushmer, Crystal and Wagner).

According to this analysis each of the three terms (suction, active diastole, *vis a fronte*) refers to a different aspect of the same physiological phenomenon. This phenomenon must be explained in terms of a force which is developed in the ventricle during diastole. The deciding issue in this problem area, therefore, is to find out whether or not such a force, called diastolic *vis a fronte*, exists.

Ventricular filling could, however, also take place without a lowering of intraventricular pressure. This would be the case if the *vis a fronte* caused a change in form of the ventricular wall during diastole in the longitudinal direction. The apex of the ventricle would remain almost stationary while the atrioventricular junction would move upwards and would thereby engulf, during diastole, blood that was outside of the ventricular orifice at the end of systole. Such mechanical action of the *vis a fronte* would result in the incorporation of some blood into the ventricle during diastole by a pushing of the ventricular walls, like an elongating cylinder, over part of the atrial content without there necessarily being a decrease in intraventricular pressure. This concept has been advanced many times in the past by anatomists and physiologists.

Looked upon from the physiologic standpoint, the result would be the same whether a diastolic force in the ventricle draws blood in by suction, or engulfs blood by moving the ventricular walls upward over atrial blood. In either case the force may be called a *vis a fronte*.

**DISCUSSION OF THE EVIDENCE**

In an interesting theoretic discourse on cardiac dynamics, Burch, Ray and Cronvitch depicted in 1952 the relationship of left ventricular volume and pressure by plotting the pressure-volume time course in the form of a loop (fig. 7 of reference 1). The values used were taken from intraventricular pressure and cardiometer volume curves of Wiggers and his associates. In this particular case Burch and his co-workers did not use original records of Wiggers, but the rather schematic curves which had been reproduced in Best and Taylor's textbook *The Physiological Basis of Medical Practice*. In describing the loop curve they state on page 509: "During the ejection phase pressure changes slightly, whereas volume increases rapidly. Then there is a rapid reduction in pressure with only slight increase in volume, and finally there is a rapid rise in volume with continued reduction in pressure as the ventricle fills during diastole. Incidentally this latter hemodynamic state indicates a 'suction' action of the ventricle."

**Critique.** This conclusion has often been taken at its face value and referred to in discussions at scientific meetings as evidence in favor of the existence of a diastolic frontal force. Mention of it as supportive inferential evidence is also made in the literature (Bloom and Ferris, Rushmer pp. 84 and 111). It is therefore necessary to analyze the factors which may be responsible for the decline during diastole of the loop tracing to see whether this decline is really evidence for suction. A decline of the tracing indicates that intraventricular pressure falls despite a ventricular volume increase. This phenomenon could be caused by a *vis a fronte* (e.g., an elastic recoil) which would expand the ven-
Recent work on ventricular diastolic suction

Ventricle faster than blood entered it. However, it could also be caused by a decrease in viscosity of the ventricular walls during diastole. If viscosity is relatively high at the beginning of ventricular filling, then ventricular pressure is high in the presence of a small volume. If viscosity decreases during diastole, then pressure falls despite a volume increase. This could be easily visualized in a suitable physical model in which only vis a tergo is employed. It is well known that the viscosity of the ventricular wall decreases during diastole. The rates of the viscosity changes and the factors affecting them are less known. It is therefore possible to explain the decline of the pressure-volume loop in the presence of a vis a tergo alone, merely by changes in ventricular wall viscosity. The decline does not necessarily indicate suction or the existence of a vis a fronte.

On the other hand, it should be pointed out that the presentation of ventricular pressure-volume relations in the loop form, as it was advanced by Burch and his co-workers, is a useful tool for the studies of ventricular hemodynamics. Under suitable experimental conditions, it might even be possible to use it for investigating suction. In this connection more recent work based on measurements of actually recorded pressures and volumes should be mentioned. Katz, Katz and Williams recorded in 1955 pressure and volumes of single left ventricles in dogs with the systemic circulation intact. Inspection of their figures reveals that there is no decline in the diastolic portion of any of the loop tracings. Rushmer has employed the loop tracings extensively in unanesthetized dogs. In a strict sense, his curves are not pressure-volume loops, since he recorded pressure versus ventricular circumference and diameter. However, there is reason to assume that the circumference and diameter changes go hand in hand with volume changes. As pointed out by Rushmer, the loop tracings during the diastole show great variability (figs. 8, 11-13, 15, 16, chapter 6) indicating changes in ventricular distensibility. Some tracings show declining portions. In the light of the foregoing discussion, this finding alone is not conclusive evidence for the existence of a vis a fronte.

In 1954 Villa advanced a new concept to explain the origin of a frontal force. He believes that there is enough evidence presented by former workers, especially by Luciani in 1871, to establish the existence of an active diastolic filling of the ventricle. Villa's paper concerns the possible muscular mechanism which causes the active diastole. He suggests that during muscle relaxation the return of the myosin molecule inside the myofibril from a globular to an elongated state is an active biochemical process attributable to a dephosphorylation. According to him, this process must be accompanied by the production of mechanical energy available for diastolic aspiration. The term decontraction is introduced to indicate an active state of elongation as over against the generally held concept of passive relaxation.

Critique. There is no reported experimental evidence to support this interesting view. The occurrence of biochemical activity in the ventricular muscle during diastole does not make it necessary to assume an energy release in the form of mechanical activity. This paper offers no evidence for the existence of a ventricular diastolic vis a fronte.

To explain the probable occurrence of an active diastole, Cignolini formulated in 1954 the intriguing hypothesis of a dynamic coupling between the heart muscle fiber and its capillary. He based his views on histologic studies of Wearn as reported by Gregg. According to Cignolini, the myocardial fiber contracts during systole and shortens the capillary that is mechanically tied to it. During diastole the capillary fills and elongates the attached muscle fiber. In support of this view Cignolini demonstrated by roentgen kymography the presence of an "i wave" corresponding to an expansion of the coronary bed in early diastole. He mentioned that this correspondence parallels the phasic changes of dimensions of the coronary capillary bed shown by the phasic blood flow studies of Gregg.
Critique. The evidence presented by Gig­
nolini in his kymograms is convincing, in that an "i wave" can be observed. However, in the absence of other information which would demonstrate definitely that the dynamic cou­pling of heart muscle fibers and capillaries produce an active diastole, this work does not contain conclusive evidence in favor of the existence of diastolic suction.

In an extensive monograph on cardiac hemo­dynamics in 1954, Guasp proposed a new version of the old concept that some parts of the heart muscle contract during diastole. According to him, the muscular trabeculae of the inner myocardial layers form an "expansive myocardium." He believes that these structures receive their impulses for contraction last among all parts of the myocardium, and therefore contract in early diastole when the outer myocardial layers are already in the process of relaxation. He illustrates diagrammatically that these inner myocardial layers present a canticular structure in which the openings of an intricate tubular system are directed toward the entricular cavity. The contraction of these structures in early dias­tole is supposed to result in expansion of the ventricle as a whole. He imagines that the blood contained between the trabeculae is projected into the ventricular cavity and that through this action it contributes to the expansion of the ventricular walls. In a more recent monograph on the functional anatomy of the heart, Guasp gave a new description of the arrangement of the ventricular fibers within the ventricular walls, a description differing from the generally accepted one so far as the mechanical action of the various myocardial layers is concerned. He contends that his concept provides an explanation of the way in which the ventricles effect their emptying and expansion through muscular contraction.

Critique. Although interesting and stimula­tive, both of Guasp’s monographs are purely theoretical and rather speculative. They are based entirely on the findings and experimental data of other workers, none of which provides conclusive support for the view that ventricular filling is aided by a contraction of parts of the myocardium during diastole.

Modern experimental approaches to the problem of intraventricular diastolic suction were initiated by Bloom in 1955, when he presented a motion picture of the excised rat heart beating in a beaker of saline. A more detailed description was given by him in 1956. Direct observation and film analysis revealed the following: With each systole, fluid was ejected from the aortic stump thereby propelling the heart rocket-like. During each diastole a flap of atrial wall was drawn into the mitral orifice. Bloom concluded that the heart must have filled each time in order to empty and that it must have done so by developing a negative ventricular pressure. He pointed out that, since the hyd­rostatic pressure in the submerged heart was equal inside and outside the heart, there was no filling pressure gradient between the ventricular orifice and cavity, unless the heart furnished the energy for filling and developed a negative pressure in diastole. He demonstrated such negative pressure later by direct measurements.

Critique. This elegant demonstration fur­nished unequivocal evidence that the excised, mammalian ventricle can develop during diastole a vis a fronte which results in some ventricular filling. It is possible that the diastolic vis a fronte also could have brought about ventricular filling by engulfing fluid that was outside the mitral valves during sys­tole, but was incorporated into the ventricu­lar cavity by an expansion of the ventricular walls in a longitudinal direction as discussed on page 556. However, this experiment does not demonstrate the development of a dias­tolic frontal force in a normal ventricle probably containing a considerable residual vol­ume.

Parenthetically, it should be mentioned that Bloom’s experiment had a historic fore­runner, which has been forgotten although it presented the same conclusive evidence for the existence of a ventricular diastolic vis a fronte as did the excised heart. In 1823 Johnson and in 1836 Chassigne submerged in water
spontaneously beating, excised turtle, dog and cat hearts and described how they ejected fluid during systole and took fluid into the ventricle during diastole.

A different approach to the problem was made by Brecher in 1956. In the intact circulation of a dog, he temporarily occluded the mitral orifice and recorded negative intraventricular diastolic pressures ranging from —13 to —108 mm. of water. For the establishment of an accurate hydrostatic reference zero level, the heart was submerged by filling the chest with saline. Precautions were taken, so that erroneous negative pressures were not produced by instrumental artefacts. The pressures became more negative under the action of epinephrine. Bristle flowmeter recordings showed that, when intraventricular pressures became negative during mitral orifice occlusion, saline or blood flowed into the ventricle through an intraventricular cannula from a reservoir situated below heart level. He concluded that the mammalian ventricle is capable of sucking blood from the atrium into its cavity.

Critique. These experiments furnished direct evidence for the existence of a ventricular diastolic vis a fronte in the intact mammalian heart. However, this evidence is limited to the unusual condition of greatly restricted ventricular volume and ejection against the small load of low aortic pressures, which fell precipitously during the mitral orifice occlusion. One cannot conclude from these experiments that a diastolic frontal force contributed to ventricular filling under normal conditions, when the end systolic ventricular volume is larger than it is under these experimental conditions.

It is historically interesting that Brecher’s experiment was in some respects a modification of von den Velden’s experimental arrangement. The difference is that von den Velden did not restrict the mitral orifice and did not establish a reliable zero pressure reference level. This explains also why von den Velden never observed any suction. Without occlusion of the mitral orifice, there was ample atrial blood available at a positive ventricular filling pressure (V point of atrial pressure) to flow into the ventricle in von den Velden’s experiments. Obviously, with a wide open mitral orifice and pliable atrial walls, a ventricular diastolic vis a fronte would not manifest itself by sucking fluid into the ventricle from a reservoir below the heart level, unless the frontal force were very powerful.

In 1956 Kraner and Ogden described two different experimental arrangements for testing intraventricular diastolic suction in the excised beating turtle ventricle. In the first series of their experiments they ligated a pipet into the ventricle, placed the heart in a plethysmograph filled with Ringer’s solution and connected the plethysmograph to another pipet. The pipets were exactly horizontal, and arranged either at equal level or with the plethysmograph pipet above the cardiac pipet. They stated that “under these conditions fluid consistently flowed into the ventricular cavity during diastole.”

Critique of the First Series of Experiments. An objection might be raised that the systolic acceleration of the fluid column in the cardiac pipet may cause a sucking effect in the ventricular cavity by the inertia of the accelerated fluid column. This well known artefact, produced by oscillating fluid columns in tubes with fluid air interfaces, can simulate intraventricular diastolic suction. As long as this artefact cannot be completely ruled out as a possible explanation for the observed phenomena, this experiment is not conclusive.

For this reason Kraner and Ogden used a second approach. A cannula was bound into the aorta leaving the valves intact and the excised heart (with its atria open) was suspended in a beaker of Ringer’s solution. The aortic tube led into a fluid-filled beaker in order to avoid the possible suction artefact arising from an oscillating fluid column. Under these conditions the ventricle filled and ejected fluid in the absence of a positive filling pressure. It was concluded that the ventricle can “suck” fluid using energy liberated from its walls during diastole.

Critique of the Second Experimental Arrangement.
proach. These experiments furnish conclusive evidence that the nearly empty, thin, walled turtle ventricle can produce a small but measurable diastolic vis a fronte.

In 1956 Bloom and Ferris\textsuperscript{12, 13} pursued Bloom's original work,\textsuperscript{7, 8} further, by making pressure measurements in the excised rat ventricle and intact dog ventricles. In the rat, left ventricular pressures were phasically recorded via a 20 gage needle connected to a Statham strain gage in such a way that the hydrostatic zero level could be established. With the intact atria the intraventricular pressures were continuously negative and rose only slightly toward zero in systole. In this case, the collapsible atrial walls occluded the mitral orifice and prevented saline inflow into the ventricular cavity. With the atria excised, the systolic pressure in the ventricle was positive and became slightly negative in diastole. These pressure changes confirmed Bloom's observation\textsuperscript{7, 8} that the excised heart was capable of drawing fluid into the ventricles during diastole. In the open-chest dog with an intact circulation, right and left intraventricular pressures were measured with a 19 gage needle and Statham strain gage. The lowest diastolic pressures within the ventricles ranged between \(-1\) to \(-3\) mm. Hg when inflow was not obstructed, but were as low as \(-15\) to \(-20\) mm. Hg in the left, and \(-4\) to \(-6\) in the right, ventricle upon obstruction of venous return. According to the authors, these findings suggest that the heart functions as a pump, drawing fluid in during diastole and ejecting fluid in systole. A further observation should be added here, which was communicated personally by Dr. Walter Bloom to the reviewer. The aortic and mitral valves appeared to open and close more or less normally in the excised rat hearts. This indicates that the pressure differences created by the ventricular form changes in systole and diastole are sufficient to operate the valves even when no positive pressures exist either at the atrium or aorta.

Critique. In the rat the intraventricular pressure curves were phasically recorded with sufficient detail for analysis. However, in the dog heart, the records were not taken with a speed high enough to obtain the details of the pulse contours. These details would have been necessary for arriving at any statements as to the negativity of diastolic pressures. Transient artefacts can easily occur in recordings of this type, and may be mistaken for "negative pressures." The literature is full of such curves with artefacts. The observations of the authors concerning the small, negative, intraventricular pressures of \(-1\) to \(-3\) mm. Hg occurring in the normal ventricles should therefore be considered with great caution and in the light of possible recording artefacts. On the other hand, the markedly negative intraventricular pressures, ranging from \(-15\) to \(-20\) mm. Hg during venous return occlusion, are probably well beyond the range of transient artefacts. The case of the negative, intraventricular pressures in the dog heart would have been stronger if detailed pulse contours had been included in the publication. The conclusion of the authors, that the heart functions as a pump, drawing fluid in during diastole, is limited to nearly empty ventricles. No conclusions can be arrived at from these experiments as to the existence of a vis a fronte in normally filled hearts.

All the foregoing experiments suggested that reliably measurable, negative, intraventricular pressures appeared only in ventricles of very small residual volumes. In 1957 Brecher and Kissen\textsuperscript{14, 15} examined therefore, the pressure volume relations in dog ventricles. They found that negative transmural pressures developed consistently in manually expressed viable ventricles. The average amount of fluid added to the right and left ventricles, to raise negative transmural pressures to zero, was 5.1 and 8.2 ml, respectively, in hearts of 126.2 Gm. average weight. They concluded that elastic forces of the ventricular walls tend to restore diastolic dimensions under static conditions and are in part responsible for ventricular suction.

Critique. It should be pointed out that the average ventricular volumes found to produce negative transmural pressures were
very small. They are probably smaller than the residual volume of a dog ventricle when the animal is at rest. Figures of direct volume measurements in normal unanesthetized dogs are not available. The estimates of left ventricular, endsystolic volumes made by Holt38 with an indirect method are about 57 ml. It is therefore questionable if the elastic forces of the ventricular walls, which can be shown to exist under static conditions, develop during diastole in a normal ventricle and act as a frontal force. The evidence presented in the experiments of Brecher and Kissen is conclusive as to the development of negative ventricular diastolic pressures upon static ventricular wall deformation. No conclusions can be derived from their study as to the occurrence of a \textit{vis a fronte} in normal ventricular filling, where dynamic deformations of the ventricular wall may come into play.

In 1957 Fowler, Bloom and Ferris10 examined the variations in intraventricular pressure in excised, beating rat hearts. Pressures were measured by needle and strain gage and the zero reference level was accurately determined by the surface of the submerging fluid. It was found that greater systolic pressure rises were followed by greater negativities of diastolic pressures. Epinephrine and levarterenol increased systolic pressures and negative diastolic pressures. Negative pressures were greatest in early diastole but often lasted until the end of diastole. It was concluded that "the observations are consistent with the concept that negative pressure change due to elastic diastolic recoil is inversely proportional to the ventricular volume following the preceding systole."

\textbf{Critique.} These well designed experiments established accurately the course of the negative diastolic pressure changes in the excised rat heart. The conclusion that the negative pressures are caused by an elastic recoil is probably correct but not definitely established, as long as an active participation of muscular contraction for a ventricular expansion has not been entirely ruled out.

The common denominator of all experiments in which ventricular diastolic suction could be demonstrated has been an abnormally small residual volume. In 1957 and 1958 Brecher and Kissen17-19 studied, therefore, the occurrence of ventricular diastolic suction in ventricles with more nearly normal residual volumes. They submerged dog hearts by filling the chest cavity with blood, extirpated the left atrial walls and maintained normal aortic pressures and circulation by pump perfusion. They found small, systolic, aortic pressure pulses resulting from ventricular ejections, which were enhanced by epinephrine. They concluded that even a ventricle with a more nearly normal end systolic residual volume draws blood into its cavity. The authors advanced the concept that in an intact normal heart a \textit{vis a fronte} can contribute to ventricular filling even if the intraventricular transmural pressure does not fall below zero.

\textbf{Critique.} In this preparation the coronary circulation was perfused by blood at normal aortic pressures, and the possibility arises that Thebesian flow may account for some or all of the ventricular filling which was manifested by the aortic systolic pressure pulses. This was ruled out as a source for ventricular filling, by occluding the mitral orifice. Ventricular ejections disappeared thereupon. Suction by a lowering of intraventricular pressure may not be the only explanation for ventricular filling. As mentioned on p. 556 of this review the ventricle could engulf blood outside the mitral orifice by expanding the walls longitudinally. The ventricle would thereby incorporate some of the blood which filled the chest cavity. This would not involve lowering of diastolic pressures to subatmospheric values, although the intake of blood into the ventricle would still be caused by a \textit{vis a fronte}. The statement of a "more nearly normal end systolic residual volume" is debatable in the absence of any volume measurements. It must be conceded that at the end of systole a greater systolic reserve and residual volume will be maintained when the ventricle ejects against the resistance existing in the aorta at normal arterial pres-
sures that when it ejects into an empty aorta. However, it is uncertain how closely this volume resembles a normal residual volume unless measurements are made. The evidence is conclusive as to the existence of a ventricular diastolic frontal force in the presence of volumes which are larger than those previously observed in empty or excised hearts. However, since neither ventricular volumes nor stroke volumes were determined in these experiments, they furnish no evidence as to the amount of blood contributed by the *vis a fronte* to the filling of the normal ventricle. The concept of ventricular filling by a frontal force, which under dynamic conditions does not necessarily result in negative transmural pressures, is physically sound and can be tested in simple models.

The same problem which concerns diastolic suction in ventricles ejecting against a resistance was approached in 1958 by Kraner in the turtle heart. He used two different experimental arrangements. The first was essentially the same as that described by Kraner and Ogden in their first series (see page 559). By suitably preventing the ejection from the cardiac pipet during systole, and raising stepwise the level of the plethysmograph pipet, he found that the filling by suction of the turtle ventricle became less with increasing residual volumes. Suction stopped when the residual volume of the nearly empty ventricle (beating against a negligible outflow resistance) was increased by 0.55 ml.

**Critique of the first series of experiments.** It is difficult to say whether these experiments furnished evidence that ventricular filling by suction is inversely related to the residual volume. The significance of these results as to suction in normal ventricular filling is uncertain in the absence of absolute residual volume measurements. The objections raised on p. 559 of this review against the use of this method may also apply here.

In the second series of experiments Kraner allowed a submerged, excised turtle ventricle to empty into a vertical, fluid-filled tube which was bound into the right aorta without destroying the aortic valves. Fluid was permitted to overflow during systole at the top of the tube, but was prevented during diastole by a flap valve from seeping back into the ventricle through incompetent aortic valves. Without positive filling pressures, some ventricles ejected against loads of fluid columns up to 20 cm. This corresponds to normal turtle aortic pressures. Outputs up to 3 ml./min., or about 10 per cent of the probable resting cardiac output, were maintained.

**Critique.** These experiments furnish unequivocal evidence that a ventricular diastolic *vis a fronte* can contribute to filling in ventricles which eject against a normal output load. The value of these experiments would have been enhanced by ventricular volume determinations.

Fowler, Couves and Bewick reported in 1958 the effect of ventricular inflow obstruction and acute hypovolemia on diastolic ventricular pressures in open-chest dogs. They recorded intraventricular pressures via a no. 12 gage needle and Statham strain gage on a Sanborn Polysino Electrocardiograph or upon an Electronics for Medicine Research Recorder. The pressure at the tricuspid valve was used as a zero reference level. During mitral inflow obstruction, pressures of −2.5 to −18 mm. Hg were measured in the left ventricle. During right ventricular inflow obstruction, right ventricular pressures fell to −1 to −2.6 mm. Hg. During acute bleeding from the right ventricle the pressures fell in the left ventricle to maximal values ranging from −2.5 to −6.5 mm. Hg. As the authors mention, "This observation suggests that ventricular suction may be important in ventricular filling during state of acute hypovolemia."

**Critique.** The records presented by the
RECENT WORK ON VENTRICULAR DIASTOLIC SUCTION

Authors were taken with a Polyviso which has a flat frequency response up to about 40 C.P.S. In the determination of negative, intraventricular diastolic pressures, one is concerned with minute subatmospheric pressures ranging often in fractions of mm. Hg. It is therefore especially important to circumvent any possible sources of error which might give more negative readings than are actually present. With this in mind, one wonders whether the writer employed especially for this problem had sufficient frequency response. If the frequency response is inadequate the pen may produce an "over shoot" or "under shoot" in the tracings, especially with fast heart rates. An over shoot could simulate artificially large, negative pressures. Although the findings of the authors are consistent with previous findings of others concerning the occurrence of negative transmural pressures in nearly empty ventricles, their observations would have gained greater validity by the use of a more suitable recording device. One can conclude from the evidence presented by the authors that negative intraventricular pressures could be recorded only in ventricle which contained small residual volumes.¹

In 1957 C. J. Wiggers called attention to the limitations on the conclusions which can be reached from the presently available evidence concerning diastolic suction. He stated, "During early moments of ventricular relaxation, elastic stresses created during contraction are released. Thus the effective left ventricular pressure falls, say, from 100 to 10 mm. Hg in approximately 0.08 sec. If blood could enter the ventricular chamber during this phase of diastole, such a rapid drop in pressure would unquestionably constitute a potent aspirating force. However, both the inlet and outlet valves remain closed until this major fall of pressure has taken place. It is not until a succeeding interval of about 0.1 sec. that blood rushes from the atria into the ventricles. An aspirating force could, therefore, aid the positive left atrial pressure in transferring blood only during this short interval. Crucial evidence is still required that the small remnant of elastic recoil still operative at the end of relaxation can create sufficient suction to be of significance in filling the normally-beating heart. Dynamically it must be shown that the concordant declines of atrial and ventricular pressures are due to a more rapid rate of ventricular relaxation than of filling from the atria."

Critique. These words of caution may well prevent unjustified conclusions from the scanty evidence available. However, the statements of Wiggers are not in conflict with the concept that a vis a fronte could aid ventricular filling. According to him, the time available during which suction could act would be approximately 100 ml./sec. This is the critical interval about which so little is known and during which, at least theoretically, a considerable amount of blood could be drawn into the ventricle in addition to being pushed into it by the vis a tergo. It is during this short interval that the highest flow rates of ventricular filling are observed. According to Rushmer and his associates these rates exceed even the rates of ventricular ejection. The comments of Wiggers point out that the problem is still far from a solution.

SUMMARY

At present the following unequivocal experimental evidence is available in favor of
the existence of a ventricular diastolic *vis a fronte*.

1. In the beating mammalian and reptilian heart fluid can be drawn into the ventricular cavity in the absence of a positive ventricular filling pressure, provided the ventricle is nearly empty.

2. In nearly empty ventricles negative transmural (subatmospheric) pressures can develop during diastole.

3. Greater negative ventricular pressures are preceded by stronger systolic contractions.

4. Elastic forces in the walls of quiescent nearly empty ventricles produce negative transmural ventricular pressures.

5. Part of the *vis a fronte* is produced by an elastic recoil of the ventricular walls which tend to assume their diastolic filling state after the end of systole.

6. The amount of fluid drawn into the ventricle is larger during diastoles which follow strong systolic contractions than during diastoles which follow weak systolic contractions.

7. Ventricles which eject their contents against the resistance offered by a fluid column of the height of about normal aortic pressures can draw fluid into their cavities in the absence of a positive filling pressure.

8. The action of epinephrine increases the amount of fluid which can be drawn without positive filling pressure into a ventricle which ejects against the resistance of a fluid column.

9. The contribution of the *vis a fronte* to ventricular filling is approximately 10 per cent of the estimated resting cardiac output in turtle ventricles working against a load which corresponds to that of normal aortic pressures in that species.

No evidence or insufficient evidence is available to support views on the following aspects of ventricular diastolic suction.

1. It is not known how much of the ventricular filling is caused by a diastolic frontal force under normal physiological conditions.

2. No information is available concerning the quantitative relation of ventricular residual volume and amount of diastolic suction under dynamic conditions.

3. There is no evidence that cardiac murmurs are caused by suction.

4. There is no evidence that the ventricles are expanded either by some myocardial contraction, or by an "active de-contraction" or by the filling of the coronary tree.

**Summario in Interlingua**

A iste momento, le sequente inequivoco provas experimental existe in favor del postulato de un *vis a fronte* diastolic ventricular:

1. In le pulsante corde de reptiles e mammalia, fluido pote esser tirate a in le cavitate ventricular in le absentia de un positive pression replenatori ventricular, providite que le ventriculo es quasi vacue.

2. In ventriculos, que es quasi vacue, negative pressiones transmural (subatmospheric) pote disveloppar se durante le diastole.

3. Plus grande pressiones intraventricular negative es precedite per plus forte contractions systolic.

4. Fortias elastic in le parietes de quiescente ventriculos, que es quasi vacue, produce negative pressiones intraventricular transmural.

5. Un parte del *vis a fronte* es producite per un resalto elastic del parietes ventricular que tende a attinger lor stato de replenation diastolic post le fin del systole.

6. Le quantitate de fluido tirate a in le ventriculo es plus grande durante diastoles que seque forte contractions systolic que durante diastoles que seque debile contractions systolic.

7. Ventriculos que ejice lor contento contra le resistentia representate per un columnna de fluido del altor de approximativamente normal pressiones aortic es capace a triar fluido a in lor cavitates in le absentia de un positive pression replenatori.

8. Le action de epinephrina augmenta le quantitate de fluido que pote esser tirate, sin positive pression replenatori, a in un ventriculo capace a ejicer contra le resistentia de un columnna de fluido.

9. Le contribution del *vis a fronte* al replenation ventricular es approximative-mente 10 pro cento del estimate rendimento
cardiac in stato de reposo in ventriculos de tortuca que travalia contra uu carga correspondente a illo de normal pressioues aortic in ille specie.

Nulle supporto o supporto insufficiente existe pro le sequente aspectos del suction diastolic ventricular:
1. II non es cognoscite qual portion del replenation ventricular es causate per le vis a fronte diastolic sub normal conditions physiologic.
2. Nulle information existe relative al relation quantitative inter le residue volumine ventricular e le magnitude de suction diastolic sub conditions dynamic.
3. Nihil supporta le these que murmures cardiac es causate per suction.
4. Nihil supporta le these que le ventriculos es expaudite per alicun genere de contraction myocardial o per un "dis-contraction active" o per le replenation del arbore coronari.

REFERENCES
4. GUASP, F. T.: El ciclo cardiaco, consideraciones criticas sobre la interpretacion clasica y nuevas ideas sobre el mismo. Privately published monograph by Guasp from the Medical Faculty of the University of Salamanca, Madrid, 1954.
Critical Review of Recent Work on Ventricular Diastolic Suction
GERHARD A. BRECHER

Circ Res. 1958;6:554-566
doi: 10.1161/01.RES.6.5.554
Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1958 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circres.ahajournals.org/content/6/5/554

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

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

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