Negative Diastolic Pressure in the Intact Canine Right Ventricle

Evidence of Diastolic Suction

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SUMMARY To determine whether the canine right ventricle (RV) can develop a negative diastolic pressure indicative of suction, RV pressure was measured in 15 dogs, with catheter-tip micromanometers. Six dogs were studied only with the chest closed. In these dogs intrapleural pressure was measured mediially (near the heart) in four and laterally in two. In nine dogs, RV pressure was evaluated with the chest closed and after the chest had been opened. In all dogs, with the chest closed, minimal RV diastolic pressure during expiration was negative, —4.8 ± 0.3 mm Hg. The lowest diastolic pressures occurred during early diastole. Intrapleural pressure during expiration was never this low. Intrapleural pressure measured in four dogs was positive during expiration (1.0 ± 0.6 mm Hg). In the two dogs in which it was measured in the lateral pleural space, it was somewhat negative during expiration (—2.6 and —1.3 mm Hg, respectively). After the chest had been opened (nine dogs) minimal RV pressure during early diastole was negative in six dogs and positive in three (range: —1.8 to 0.8 mm Hg). These results indicate that the negative RV diastolic pressure during expiration did not result from a negative intrathoracic pressure. It appears that the RV during early diastole can create a sucking effect which may contribute to the filling process. Circ Res 49: 108-113, 1981

A NEGATIVE pressure is often measured in the human right ventricle during early diastole (Stein et al., 1980a; Sabbah et al., 1980a), and is generally thought to reflect a negative intrathoracic pressure rather than diastolic suction, although the latter has been suggested (Sabbah et al., 1980a). If ventricular diastolic suction exists, the implication is that ventricular filling in early diastole may be assisted by a process of active ventricular relaxation (Katze, 1930; Rushmer et al., 1953; Brecher, 1956). In general, it is thought that the ventricle is filled only due to the pressure in the atrium during diastole and by atrial contraction. Studies in animals have demonstrated that the left ventricle is capable of generating a negative diastolic pressure under...
certain circumstances which suggests the concept of ventricular diastolic suction (Bloom, 1956; Bloom and Ferris, 1956a; Fowler et al., 1957; Brecher, 1958; Brecher and Kissen, 1958; Fowler et al., 1958). Negative diastolic pressures were shown to occur in the left ventricle of dogs when mitral inflow was impeded (Bloom and Ferris, 1956a; Fowler et al., 1958). Rapid bleeding from the right ventricle has also been shown to result in negative left ventricular pressure during early diastole (Fowler et al., 1958).

In view of these observations, it was suggested that negative intraventricular diastolic pressure occurs only after the heart has been reduced to a critical volume by expulsion of its contents in systole (Bloom and Ferris, 1956b; Brecher and Kissen, 1957). We showed that negative diastolic pressure can occur in the left ventricle of patients with severe mitral stenosis, even in the presence of a normal end-systolic volume (Sabbah et al., 1980b). Others speculated on the occurrence of diastolic suction in the human left ventricle based upon autopsy of a man who died of a gunshot wound that penetrated the right side of the heart (Roberts et al., 1979). Autopsy findings showed the left atrial appendage to be inverted and invaginated into the mitral orifice (Roberts et al., 1979). The authors suggested that only a vacuum effect of the left ventricle during diastole could explain this observation (Roberts et al., 1979).

Whether the right ventricle can develop negative pressure during diastole has received less attention. Diastolic pressures in the right ventricle of the intact dog heart with the chest open were slightly negative (Bloom and Ferris, 1956b) or sometimes positive (Fowler et al., 1958), but became negative or more negative as the inflow (superior and inferior vena cavae, coronary sinus, and azygous veins) was occluded (Bloom and Ferris, 1956a; 1956b; Fowler et al., 1958).

The absence of a negative diastolic pressure in the normal left ventricle under physiological conditions generally has caused a hesitancy to accept the concept of ventricular diastolic suction. The normal right ventricle, on the other hand, has been shown to have negative pressures during early diastole; but whether these pressures reflect negative pleural pressures or ventricular suction is unclear. An evaluation of the cause of right ventricular negative diastolic pressure would assist in clarifying whether a self-filling action of the ventricle is a physiological phenomenon. The purpose of this investigation, therefore, was to evaluate diastolic pressure in the intact canine right ventricle to determine whether diastolic suction may be produced by the right ventricle. Toward this end, a simultaneous assessment of intrathoracic pressure was made, as well as an evaluation of the effects of opening the chest upon right ventricular and right atrial pressures.

Methods

Measurements of right ventricular pressure during diastole were made in 15 mongrel dogs. The dogs weighed between 16 and 30 kg, and were anesthetized with droperidol (1.2 mg/kg) and fentanyl citrate (0.24 mg/kg) [Innovar-Vet (0.6 ml/kg)]. The depth of the anesthesia was maintained throughout the procedure with a continuous intravenous infusion of droperidol (0.7 mg/min), fentanyl citrate (0.01 mg/min), and pentobarbital (0.17 mg/min).

Measurements of Right Ventricular and Intrathoracic Pressure

Simultaneous measurements of intrathoracic and right ventricular pressure were made in six of the 15 dogs. All six dogs were positioned on their backs and allowed to breathe spontaneously. Respiration was measured with a nasal thermistor (Electronics for Medicine). Right ventricular pressure was measured with a number 6-French catheter-tip micromanometer (Millar Instruments). The micromanometer was introduced through a femoral vein cutdown and was positioned in the right ventricle under fluoroscopic control. Intrathoracic pressure was also measured with a number 6-French catheter-tip micromanometer. The micromanometer was introduced into the pleural space through a 20-French Ochsner bladder trocar that was modified by insertion of vacuum seals into the side branch. These seals permitted the introduction of a catheter without the introduction of air into the pleural space. A skin incision approximately 5 mm in length was made at the mid-lateral chest wall along the 4th or 5th intercostal space. A purse-string suture was then placed around the skin incision. The trocar was then advanced into the chest wall and the purse-string suture was tied around it. The trocar was gently pushed until it penetrated into the pleural cavity. The vacuum plunger then was withdrawn and the catheter was passed into the pleural space. Under fluoroscopic control the catheter was positioned over the anterior surface of the heart in four dogs and was positioned in the right lateral pleural space in two dogs. To ensure that the introduction of the trocar into the pleural space did not cause a partial pneumothorax, selective pulmonary arteriograms near the point of entry were performed at the end of the study.

The characteristics of the pressure transducers have been described previously (Sabbah et al., 1980b). Its low mass makes it insensitive to acceleration forces and "catheter whip." Tests in our laboratory showed a baseline drift of less than 1 mm Hg in 3 hours. The zero baseline of the transducer was adjusted to atmospheric pressure at the beginning of each procedure. In view of baseline shifts that may occur with temperature differences between the body and room air, the level of zero pressure was established following withdrawal of the catheter-tip micromanometer from the vein to room air or from the chest cavity to room air.

Simultaneous recordings of right ventricular pressure, intrathoracic pressure, the phases of respiration, and lead II of the electrocardiogram were obtained on a VR-12 photographic recorder (Elec-
tronics for Medicine), at paper speeds of 250 mm/sec, as well as at slower speeds. The frequency response of the recording system was flat to 2500 Hz. However, pressures were filtered at frequencies above 250 Hz.

**Measurements of Right Ventricular and Right Atrial Pressure before and after Thoracotomy**

In nine of 15 dogs, right ventricular pressure was measured before and after the chest had been opened. Simultaneous measurements of right atrial pressure were made in six of these nine dogs. Before the chest was opened, spontaneous respiration was monitored with a nasal thermistor. Catheter-tip micromanometers were positioned in the right ventricle and right atrium. The sensors were made equisensitive prior to insertion. The zero baselines, relative to atmospheric pressure, were rechecked following withdrawal of both micromanometers to room air. After pressure was recorded with the chest closed, the dog was ventilated with a respirator and a mid-sternal thoracotomy was performed without altering the position of the catheters within the right ventricle and right atrium.

In all dogs, right ventricular pressure during diastole was measured at the lowest point that the pressure reached during expiration. Right atrial pressure was measured at the same time. Intrathoracic pressure was measured at maximal inspiration and at end-expiration.

**Results**

In all dogs with the chest closed, right ventricular pressure was negative during the entire respiratory cycle. During expiration, minimal right ventricular pressure occurred in early diastole and was $-4.8 \pm 0.3$ mm Hg (range: $-1.9$ to $-6.8$ mm Hg). Right ventricular pressure during diastole became more negative during inspiration (Fig. 1).

Intrathoracic pressure measured medially (near the heart) in four dogs was positive or approximated atmospheric pressure during a major portion of expiration (Fig. 1). It reached a value of $1.0 \pm 0.6$ mm Hg (range: 0 to 2.8 mm Hg) at end-expiration. It became prominently negative at peak inspiration, $-9.5 \pm 2$ mm Hg. Intrapleural pressure was lower laterally than medially. In two dogs in which intrapleural pressure was measured in the right lateral pleural space, it was negative during expiration ($-2.5$ and $-1.3$ mm Hg, respectively) (Fig. 2). In these two dogs minimal right ventricular pressure during expiration was even lower ($-4.8$ and $-6.5$ mm Hg, respectively).

After the chest had been opened (nine dogs), right ventricular pressure during diastole became less negative in six dogs and positive in three. In these dogs, minimal right ventricular pressure following thoracotomy occurred during early diastole and was $-0.6 \pm 0.3$ mm Hg (range $-1.8$ to 0.8 mm Hg) (Fig. 3).

Right ventricular pressure was always lower than right atrial pressure during early diastole irrespective of whether the chest was open or closed (six of six dogs) (Fig. 3). With the chest closed, minimal right ventricular pressure, $-4.8 \pm 0.3$ mm Hg, was lower than right atrial pressure measured at the same moment during expiration, $-0.7 \pm 0.2$ mm Hg. When the chest was open, minimal right ventricular pressure during early diastole increased to $-0.6 \pm 0.3$ mm Hg, whereas right atrial pressure measured at the same moment increased to $3.6 \pm 0.4$ mm Hg (Fig. 3). Exposure of the heart to atmospheric pressure was associated with an increased minimal right ventricular pressure of 4.2 mm Hg and an increased right atrial pressure of essentially the same amount when measured at the same moment ($4.3$ mm Hg). Although opening the chest affected minimal right ventricular pressure and right atrial pressure during early diastole, it had little or no effect upon right

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**Figure 1** Right ventricular (RV) pressure and medial intrapleural pressure during inspiration (INSP) and expiration (EXP) in a dog with the chest closed. The phases of respiration were recorded with a nasal thermistor.
ventricular systolic pressure. With the chest closed, right ventricular systolic pressure was 22 ± 1 mm Hg and with the chest open it was 23 ± 1 mm Hg (NS) (paired t-test).

**Discussion**

A negative early diastolic pressure in the right ventricle during expiration does not appear to be due to a negative intrathoracic pressure. We observed a negative right ventricular diastolic pressure even though intrapleural pressure, measured near the heart, was positive or approximated atmospheric pressure. We also observed a negative right ventricular pressure during early diastole in dogs with the chest open.

Measurements of intrapleural pressure in this study are consistent with measurements by others. The pressure within the pleural cavity that is most likely to affect the pressure within the heart would be the pressure measured in close proximity to the heart, rather than pressure at some distance from it (Coleridge and Linden, 1954). Pressures in the medial pleural space and mediastinum were shown to be less negative than in the lateral pleural space.

**Figure 2** Right ventricular (RV) pressure and right lateral intrapleural pressure during inspiration (INSP) and expiration (EXP) in a dog with the chest closed.

**Figure 3** Right ventricular (RV) and right atrial (RA) pressure in a dog during expiration. Left: the chest was closed; right: the chest was open.
(Coleridge and Linden, 1954). In a typical anesthetized dog lying supine, the intrapleural pressure near the heart at end-expiration was \(-0.5 \text{ cm H}_2\text{O}\) (Coleridge and Linden, 1954). Illustrations by the same investigators showed that intrapleural pressure near the heart was positive during early and mid-expiration. Records by Fishman (1966) of intrapleural pressure in a human subject at rest showed that intrapleural pressure was positive (approximately \(2 \text{ mm H}_2\text{O}\)) during expiration and became negative (approximately \(-5 \text{ mm H}_2\text{O}\)) only during inspiration. Measurements of intrapericardial pressure were made in closed-chest dogs by Shabetai and associates (1961, 1963, 1965). Illustrated records from these studies indicate that intrapericardial pressure was positive or approximated atmospheric pressure during the early to mid-portion of expiration. In view of these observations, and our observations as well, it appears that there is only a small (if any) negative intrathoracic pressure near the right ventricle during expiration.

Diastolic pressures in the right ventricle of the intact dog heart with the chest open were described by some investigators as slightly negative (Bloom and Ferris, 1956b) and by others as slightly positive (Fowler et al., 1958). The pressures became progressively more negative (\(-5 \text{ mm H}_2\text{O}\)) after ligation of the superior and inferior vena cavae, coronary sinus, and ayzygous veins (Bloom and Ferris, 1956b). Others reported right ventricular pressures during diastole of 0 to 1 mm Hg in the intact dog (Fowler et al., 1958). They also showed negative right ventricular diastolic pressures of \(-1\) to \(-2.6 \text{ mm H}_2\text{O}\) following obstruction of the vena cavae and ayzygous veins (Fowler et al., 1958). We observed a negative pressure in the right ventricle of open-chest dogs during early diastole. This negative pressure, in view of the fact that the chest was open, could not be caused by a negative intrathoracic pressure.

When the chest was open, right ventricular pressure during early diastole was less negative than when the chest was closed. This difference may relate to a change of right atrial pressure. When the dog's chest was open, right atrial pressure increased. A higher head of pressure in the right atrium would assist the inflow of blood into the right ventricle and tend to minimize the development of a negative pressure. The increased right atrial pressure following thoracotomy was equivalent to the increment of diastolic pressure within the right ventricle. This is analogous to observations we made in patients. In patients with pulmonary hypertension and right ventricular failure in whom right atrial pressure was elevated, minimal right ventricular diastolic pressure was positive (Sabbah et al., 1980a). In patients with normal pulmonary arterial pressure or pulmonary hypertension but without failure, right atrial pressure was low and a negative right ventricular pressure during early diastole was observed (Sabbah et al., 1980a).

The mechanism of ventricular suction, particularly whether it is produced by contraction in the myocardial fibers or elastic recoil of fibers, is speculative. One speculative process would be the diastolic contraction of muscle fibers which, owing to their anatomic arrangement, would enlarge the ventricle during diastole (Gausp, 1954). Another speculative process is the development of a force during diastole that acts to lengthen the muscle fibers (Brecher and Galletti, 1966). Processes that may permit diastolic suction also include elastic recoil which would tend to restore ventricular diastolic dimensions (Bloom and Ferris, 1956b; Brecher and Kissen, 1957). Perhaps myocardial fibers of different layers pull against one another and stretch the connections between them (Rushmer et al., 1953). This action may produce potential energy which is wasted as far as systolic ejection is concerned. As the ventricles begin to relax, however, this potential energy would be released and could act to return the ventricular chambers to their original diastolic dimensions. Under these conditions, the inflowing blood would encounter a small resistance to distension, and rapid filling in early diastole would be facilitated (Rushmer et al., 1953). The restoring force also may result from distortion of the intramural structure of the wall and, in part, this force may be derived from compression of the sarcomeres (Tyberg et al., 1970). If individual muscle fibers are made to contract, their sarcomeres will shorten and, with relaxation, the sarcomeres will spring back to their longer length, demonstrating a restoring force (Parsons and Porter, 1966). In this sense, the shortening sarcomere overcomes a resistance of unknown magnitude and retains this energy as elastic recoil upon relaxation (Tyberg et al., 1970).

Negative diastolic pressures were shown in the right ventricle of patients who were not in right ventricular failure (Sabbah et al., 1980a). However, negative diastolic pressure did not occur in the normal left ventricle (Sabbah et al., 1980b). A possible explanation for the difference between right ventricular and left ventricular diastolic pressures may relate to differences between right atrial and left atrial pressures. Normally, right atrial pressure is lower than left atrial pressure (Yang et al., 1972). If the restoration of the ventricles to their diastolic dimensions were due to an active process, the presence of a high head of pressure in the atrium would assist in causing a rapid inflow into the ventricle and therefore tend to eliminate any negative pressure. On the other hand, if the atrial pressure is low, as in the right atrium, a rapid change of ventricular dimensions may surpass the rate of flow available to fill the ventricle and, therefore, create a negative pressure. The same mechanism may explain the less negative right ventricular pressure in open-chest dogs in comparison to closed-chest dogs. In dogs with the chest open, right atrial pressure was higher than when the chest was closed. The higher atrial pressure would minimize any negative dia-
stolic pressure within the ventricle.

Any force that lowers pressure in a chamber toward which flow occurs can be termed suction, whether or not the pressure developed in that region drops below atmospheric pressure (Brecher and Galletti, 1966). Rushmer et al. (1953) stated, however, that the term must be used only when a negative pressure is present. The observations of a negative pressure within the right ventricle during diastole in this study are consistent with both definitions of suction. The absence of a negative pressure during diastole does not necessarily imply the absence of suction, and there is some evidence to support this concept. Observations of left ventricular pressure-diameter relations during early diastole were shown to be incompatible with passive filling (Burch et al., 1952; Sabbah and Stein, 1981). These observations suggested that the left ventricle during early diastole assisted in its own filling even in the absence of a negative left ventricular diastolic pressure.

In conclusion, in dogs with the chest closed, negative right ventricular diastolic pressure did not appear to result from the effects of intrathoracic pressure. Negative pressure in the right ventricle persisted, but to a lesser extent, after the chest had been opened. On this basis, it appears that the dynamics of the right ventricle during early diastole may create a sucking effect which may contribute to the filling process.

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