Pressure-Diameter Relations during Early Diastole in Dogs

Incompatibility with the Concept of Passive Left Ventricular Filling

HANI N. SABBAH AND PAUL D. STEIN

SUMMARY We studied left ventricular (LV) pressure-diameter relations in 13 open-chest mongrel dogs to explore whether ventricular filling is passive, active, or a combination of both. Left ventricular internal diameter along the minor axis and thickness of the LV free wall were measured with ultrasonic dimension gauges. Pressures in the LV, left atrium (LA), and aorta were measured with catheter-tip micromanometers. Pressure in the LV during the rapid filling phase of diastole decreased from 13 ± 1 mm Hg to 3 ± 0.4 mm Hg (mean ± SE). During this period, LV internal diameter increased from 32.6 ± 1.5 to 36.7 ± 1.5 mm, and this represented 49% of the total change of LV diameter that occurred during diastole. The pressure-diameter relation that we observed during the rapid phase of filling suggests that active enlargement rather than passive distension of the left ventricle occurred. Reduction of pressure within the left ventricle during rapid filling, while the ventricular diameter increased, appears to be due to forces within the LV wall that act to restore the LV to its diastolic dimensions. This allows the LV to draw blood from the atrium by creating a pressure in the LV that is lower than in the LA. On the other hand, during the latter part of diastole, following atrial contraction, the pressure-diameter relation suggests that the left ventricle undergoes passive distention.


WHETHER the left ventricle fills passively or actively assists in its filling has been a subject of investigation for many years. In general, it is thought that the ventricle is filled by a head of pressure in the atrium during early and mid diastole and by atrial contraction in the later part of diastole. In this respect, filling would be considered a passive process. This concept forms the basis for the assessment of ventricular compliance by diastolic pressure-volume relations (Mirsky, 1969; Diamond et al, 1971; Mirsky and Parmley, 1973; and Mirsky, 1976). Another concept is that of active filling, in which the ventricle fills itself by drawing blood from the atrial reservoir. This form of ventricular filling has been characterized as “diastolic suction” (Katz, 1930; Brecher, 1956; Bloom and Ferris, 1956a; Fowler et al., 1958). If ventricular diastolic suction exists, then ventricular filling in early diastole would result from an active process of changing ventricular diastolic dimensions either by elastic recoil (Bloom and Ferris, 1956b) or muscular contraction (Gausp, 1954). Under certain circumstances, the ventricle can develop a negative pressure during early diastole, and this has been the major evidence in favor of non-passive ventricular filling (Katz, 1930; Brecher, 1956; Bloom and Ferris, 1956a; Sabbah et al., 1980). An inability to demonstrate negative intraventricular pressure during diastole under normal physiological conditions has caused the concept of active filling to be largely disregarded.

The purpose of this investigation was to explore the relation between pressure and diameter during diastole in the normal canine left ventricle. The pressure-diameter relation during diastole might help determine whether filling of the ventricle is passive, active, or a combination of both.

Methods

Simultaneous measurements of left ventricular, left atrial, and aortic pressure, as well as measurements of left ventricular internal diameter and wall thickness, were made in 13 mongrel dogs. The dogs weighed between 21 and 26 kg, and were anesthetized with droperidol, 1.2 mg/kg, and fentanyl citrate, 0.24 mg/kg (Innovar-Vet 0.6 ml/kg, iv). 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/min; and pentobarbital, 0.17 mg/min. In each dog, a left thoracotomy was performed and the pericardium was opened.

Left ventricular internal diameter, along its minor axis was measured using ultrasonic crystal dimension gauges (Schuessler) (Fig. 1). The method for insertion of the endocardial ultrasonic crystals was similar to that described by Horwitz et al.
were threaded through a 13-gauge, 4-inch-long stainless steel needle from which the hub had been removed. A stab incision was made through the anterior left ventricular wall between the stitches. The needle, through which the wires of the ultrasonic gauge were threaded, was inserted into the anterior left ventricular wall to position the crystal against the anterior endocardial surface. The wires of one of the ultrasonic crystals were anchored in position by purse-string sutures. We did not find it necessary to occlude the inferior or superior vena cava during the procedure (Horwitz et al., 1968). Two no. 00 control stitches were placed just inferior and perpendicular to the first diagonal branch of the left anterior descending coronary artery. The wires of one of the ultrasonic crystals were passed through the lumen of a 4-cm-long rigid plastic tube with an internal diameter of 1.4 mm. The plastic tube was used to push the crystal through the incision in the anterior wall. The tube was then removed. A gentle pull on the wires positioned the ultrasonic crystal against the anterior endocardial surface of the left ventricle. The crystals were anchored in position by purse-string sutures. We did not find it necessary to occlude the inferior or superior vena cava during the procedure as had been done by others to reduce bleeding during the procedure (Horwitz et al., 1968). The crystals that were used for the measurement of the internal diameter were hemispheric with a diameter of 5 mm and a maximal thickness of 1.5 mm. Both crystals were placed on the endocardial surface with the convex surfaces of the crystals pointing toward each other. The location of the crystals on the endocardial surface was confirmed at autopsy.

Simultaneous changes of thickness of the left ventricular anterior wall were also measured with a pair of ultrasonic crystals (Fig. 1). One crystal was sutured to the epicardial surface; the other was inserted as near as possible to the endocardial surface without perforating into the left ventricular cavity. The subendocardial crystal was introduced at an angle of 45° relative to the epicardial surface to avoid injury of the myocardium between the two crystals. In implanting the deep crystal, a tunnel was created with an 18-gauge needle. The epicardial crystal was sutured in direct alignment with the subendocardial crystal. The epicardial crystal was 1.5 mm thick and 4 mm in diameter; whereas the deep crystal was 1 mm thick and 2 mm in diameter. When in place, the convex surfaces of both crystals pointed toward each other. The location of the subendocardial crystal was confirmed at autopsy.

Aortic, left ventricular, and left atrial pressures were measured with catheter-tip micromanometers (Millar Instruments). The micromanometer used to measure left atrial pressure was introduced directly into the left atrium through an incision in the left atrial appendage. Bleeding was controlled by tying the tip of the atrial appendage around the catheter. Atrial fibrillation was not induced in any of the dogs. The hematocrits in these dogs, after complete instrumentation, ranged from 35 to 46 ml/100 ml.

The pressure sensors had a frequency response that was flat within ±2% at 5 kHz and within ±5% at 10 kHz. The phase lag of this type of sensor is 90° at 35 kHz, which is equivalent to a time delay of approximately 7 μsec. Tests in our laboratory showed a baseline drift of less than 1 mm Hg in 3 hours. The zero baseline of each transducer was adjusted to atmospheric pressure at the beginning of each procedure. All transducers were made equisensitive. Because of baseline shifts that may occur with temperature differences between the body and room air, the level of zero pressure was reestablished after withdrawal of the catheter-tip micromanometers to room air. This level of zero was used in all cases as the true zero level. The equisensitivity of the transducers was checked at the end of each study.

Pressures, wall thickness, and ventricular diameter were recorded on a VR-12 photographic recorder (Electronics for Medicine) at paper speeds of 25 to 250 mm/sec. The frequency response of the recorder was flat to 2500 Hz. Pressures, however, were filtered with a 250 Hz low pass filter. The frequency response of the ultrasonic dimension gauge system was flat from 0 to 60 Hz.

Diastole was defined as the period from that point at which left ventricular pressure fell below left atrial pressure to the point at which left ventricular pressure increased above left atrial pressure. The phase of rapid filling was defined as the portion of diastole during which the diameter increased rapidly. Diastasis was defined as the period of slowly increasing or unchanging diameter during the mid-portion of diastole. Atrial contraction was
defined as the portion following the onset of the left atrial A wave.

Calculations of ventricular diameter and wall thickness were made when left ventricular pressure fell below left atrial pressure (the point of crossover), at the time of minimal left ventricular pressure, at the onset of the left atrial A wave, and at the end of diastole when left ventricular pressure crossed over left atrial pressure. The difference of pressure between the left ventricle and left atrium during diastole was calculated and plotted using an electronic digitizer (Numonics) on line with a 21 MX computer (Hewlett-Packard). The pressure-diameter curves of single beats were constructed from data obtained throughout the entire diastolic period. Left ventricular pressure and left ventricular internal diameter along the minor axis were digitized at 1-mm intervals at paper speeds of 250 mm/sec, which corresponded to intervals of 4 msec.

Instantaneous distensibility of the left ventricle in early diastole was calculated as the ratio of the instantaneous value of left ventricular diameter to instantaneous left ventricular pressure. The ratio of diameter to pressure was plotted as a function of time at intervals of 4 msec, starting at the time of crossover of left ventricular and left atrial pressure, and ending at the time of minimal left ventricular pressure.

Results

The crossover of left ventricular and left atrial pressure occurred at a pressure of 13 ± 1 mm Hg (mean ± se). Left ventricular pressure continued to diminish and reached a minimal value of 3 ± 0.4 mm Hg an average of 66 msec (range, 50–90 msec) after the point of crossover (Fig. 2). This time period represented 29% of diastole (Figs. 2, 3). Past the point of crossover, left atrial pressure decreased more slowly than left ventricular pressure (Figs. 3, 4). The pressure difference developed because left ventricular pressure diminished more rapidly than left atrial pressure.

The internal diameter of the left ventricle increased as pressure within the left ventricle diminished (Figs. 3, 4). The left ventricular internal diameter, measured at the moment of left ventricular and left atrial pressure crossover, was 32.6 ± 1.5 mm. It increased to 36.7 ± 1.5 mm at the time of minimal left ventricular diastolic pressure. This represented an average of 49% (range, 24–82%) of the change of diameter that occurred during diastole.

Left ventricular wall thickness, measured at the moment of pressure crossover, was 13.0 ± 0.5 mm. The wall thinned to 11.4 ± 0.5 mm at the moment of minimal left ventricular pressure (Figs. 2–4). This reduction of wall thickness represented an average of 54% (range, 24–89%) of the thinning that occurred during diastole. The left ventricular wall became thinner, therefore, as pressure within the left ventricle diminished.

During the rapid filling phase of diastole, left ventricular pressure-diameter relations showed a diminishing pressure in the left ventricle associated with a prominently increasing ventricular diameter (Fig. 5).

Left ventricular pressure began to increase shortly after it reached a minimal value, and it increased gradually or was nearly constant during diastasis. During diastasis, left atrial pressure was nearly constant or diminished (Figs. 3, 4). Atrial contraction resulted in an increase of both left atrial and left ventricular pressure. At this time, left atrial pressure increased more than left ventricular pressure. The pressure difference between the chambers increased. During atrial contraction, left ventricular diameter increased from 38.8 ± 1.4 to 41.2 ± 1.4 mm. This accounted for 28% of the total change of diameter during diastole. Wall thickness diminished from 10.5 ± 0.5 to 10.0 ± 0.5 mm during atrial contraction, and this accounted for 17% of the
total reduction of wall thickness (Figs. 3, 4). During atrial contraction, therefore, the further increase of diameter and thinning of the wall of the left ventricle were accompanied by an increased pressure within the left ventricle.

Distensibility of the left ventricle calculated as the ratio of left ventricular internal diameter to left ventricular intracavitary pressure increased in early diastole in all the dogs (Fig. 6). During this period, starting at the time of crossover of left ventricular
and left atrial pressure and ending at the time of minimal left ventricular pressure, the change of distensibility with time was relatively constant in 8 dogs, and it showed an increasing change with time in five dogs (Fig. 6).

**Discussion**

The relation between left ventricular pressure and diameter that we observed during the rapid phase of ventricular filling suggests that left ventricular filling may not be a passive process. During early diastole, we observed a diminishing left ventricular pressure as left ventricular diameter increased. Such a behavior is compatible with self enlargement of the left ventricle, possibly secondary to an active process of myocardial relaxation.

A diminishing left ventricular pressure associated with an increasing ventricular volume also conceivably could occur if the distensibility of the left ventricular wall, during early diastole, markedly increased during that time. This would require a lower stress (ventricular pressure) to achieve a passive enlargement of the ventricle. To explore this possibility we examined the distensibility of the ventricle during early diastole. The minor axis of the left ventricle has been shown to relate directly to left ventricular volume (Rankin et al., 1980). If one defines instantaneous distensibility of the ventricle as the ratio of ventricular diameter (and therefore ventricular volume) to ventricular pressure, then

\[ V = D \cdot P \]  

where \( V \) is instantaneous ventricular volume (ml), \( P \) is instantaneous ventricular pressure (mm Hg), and \( D \) is instantaneous distensibility (ml/mm Hg).

The use of instantaneous pressure-volume relations is compatible with the work of Suga and Sagawa (1974). If all three parameters are time dependent, then by differentiating Equation 1, one would obtain the following relation:

\[ \Delta V = \Delta P \cdot D + \Delta D \cdot P \]  

where \( \Delta V \) is the change of volume (ml) for a given period of time; \( \Delta P \) is the change of pressure (mm Hg) during this time and \( \Delta D \) is the change of distensibility (ml/mm Hg) during this time.

It could happen that \( \Delta D \) is sufficiently large to permit \( \Delta P \) to be negative, yet result in a positive increment of volume. However, when the data from our dogs were substituted in Equation 2, starting at the time of the left ventricular and left atrial pressure crossover and ending at the time of minimal left ventricular pressure, \( \Delta P \cdot D + \Delta D \cdot P \) was always negative (Fig. 6). This suggests that a reduction of pressure would be accompanied by reduction of volume if there were passive distention. However, during the rapid filling phase, ventricular diameter increased while pressure within the ventricle decreased. Therefore, ventricular enlargement does not appear to result from passive filling. A reduction of left ventricular pressure during filling suggests that the ventricle causes its diameter to increase and therefore contributes to its filling. Elastic recoil or muscular contraction within the left ventricular wall during diastole may produce such restoring forces and explain these observations. Such a process could produce thinning of the ventricular wall during diastole. Prominent thinning of the free wall of the left ventricle was observed as the ventricle increased in diameter during rapid filling.

Although there was a pressure difference between the left atrium and left ventricle during rapid filling, it should be considered in perspective with the self-filling action of the left ventricle. The pressure difference occurred due to a more rapid fall of left ventricular pressure than left atrial pressure. This can occur only as a result of the self-enlarging action of the left ventricle. Blood flowed into the left ventricle because of the pressure difference. However, the pressure difference was caused by the reduction of left ventricular pressure below left atrial pressure.

A large number of studies of ventricular compliance based on pressure-volume relations during diastole indicate passive filling of the left ventricle. How are these studies compatible with our observations? The answer seems to relate to the portion...
FIGURE 6  Top: Left ventricular (LV) internal diameter is shown as it changes with time during early diastole in a single beat. \( t_1 \) is the time of crossover of left ventricular and left atrial pressure, and \( t_2 \) is the time of minimal left ventricular pressure. Middle: Left ventricular (LV) pressure is shown as it changes with time during the same cardiac cycle and during the same period of time as above. Bottom: Distensibility is shown as it varies with time during early diastole. Distensibility was calculated as the ratio of diameter (top panel) to pressure (middle panel). From these graphs, data can be obtained to substitute in the equation \( \Delta V = \Delta P \cdot D + \Delta D \cdot P \). From the bottom panel, the change of distensibility over the time \( t_1 \) to \( t_2 \), \( \Delta D = 11 \text{ mm/mm Hg} \), and the average distensibility, \( D = 8 \text{ mm/mm Hg} \). From the middle panel, the change of left ventricular pressure, \( \Delta P = -9 \text{ mm Hg} \) and the average LV pressure, \( P = 6 \text{ mm Hg} \). Therefore, by substitution in the equation it is clear that the change of volume, \( \Delta V \), would be negative. Assuming passive distention, therefore, for a given reduction of pressure a reduction of volume would be predicted. This did not occur.
the static relationship present during dynamic filling (Rankin et al., 1980). Some have calculated a negative stiffness during the early phase of ventricular filling (Kennish et al., 1975). Inertia of the cardiohemic system was excluded as a likely mechanism for this apparent negative stiffness (Kennish et al., 1975). It was felt that the negative stiffness was a manifestation of incomplete relaxation (Kennish et al., 1975). The observations were thought to be explainable on the basis of a model containing an element whose deformation is rate dependent, that is, a parallel viscous element.

A continuing active decay of stress within the left ventricle may be an important factor underlying the continuing decline of pressure within the left ventricular wall during rapid filling (Kennish et al., 1975; Weiss et al., 1976). The likelihood of this is supported by the observation that pressure within the wall of the left ventricle continues to fall well after the onset of diastole (Stein et al., 1980). To explain the increasing left ventricular volume that accompanies the diminishing left ventricular pressure, one must postulate the existence of a time-varying reduction of stiffness that is sufficient to allow distention of the left ventricle by a progressively diminishing pressure.

The observed variation of the difference between left atrial and left ventricular pressure during rapid filling also is incompatible with the concept of passive filling during this period. If passive filling were present, pressure would not diminish in the left ventricle at a greater rate than in the left atrium.

The issue of whether the ventricle is filled passively or whether it draws blood from the atrium has rested primarily on whether ventricular diastolic suction can be demonstrated under normal physiological conditions. In 1956, Brecher recorded negative diastolic pressures, −1.3 to −10.8 cm H₂O, in the left ventricle of dogs when the mitral orifice was temporarily occluded. Others subsequently showed similar results either following obstruction of mitral inflow (Bloom and Ferris, 1956a; Fowler et al., 1958; Tyberg et al., 1970), or following rapid bleeding (Fowler et al., 1958). An autopsy report of a man who died after a gunshot wound of the right ventricle seems to reflect this mechanism (Roberts et al., 1979). The left ventricle following hemorrhage appeared to create a sucking action sufficiently strong to cause the left atrial appendage to invaginate through the mitral orifice into the left ventricle (Roberts et al., 1979).

Observations of the capability of the mammalian left ventricle to create a negative diastolic pressure usually have been limited to nonphysiological conditions, such as inflow occlusion, severe hemorrhage, or stimulation by isoproterenol (Brecher, 1956; Bloom and Ferris, 1956a; Fowler et al., 1958). Whether diastolic suction can occur under physiological conditions has been unsettled. These observations, however, did not reflect the effects of negative intrathoracic pressure, because the chest was open in the dogs in these studies. Several investigators have suggested that a negative diastolic pressure occurs in the left ventricle only when the end-systolic volume is small (Bloom, 1956; Fowler et al., 1958; Brecher and Kissen, 1967; Roberts et al., 1979). We have demonstrated, however, that negative left ventricular pressure can occur in patients with mitral stenosis in the presence of a normal end-systolic volume (Sabbah et al., 1980). The negative diastolic pressure in these patients appears to be related to the maintenance of a vigorous contraction in the presence of mitral stenosis. The ventricle appears to have exerted a sucking action to draw blood through the reduced mitral orifice.

Any force that lowers pressure in a region toward which flow occurs can be termed suction, whether or not the pressure developed in that region drops below atmospheric zero (Brecher and Galletti, 1966). The term “suction” has been defined as a reduction of pressure at some point in a system by the application of a force which results from an energy conversion process, such as muscular contraction or elastic recoil (Brecher and Galletti, 1966). The definition of suction in these terms was reputed by Rushmer et al. (1953), who stated that the term must be used only when a negative pressure is present. Our observations of pressure-diameter relations during rapid filling suggest that the ventricle draws blood from the atrium. This occurred in the absence of a negative left ventricular pressure. The observation is consistent with suction, as defined by Brecher and Galletti (1966).

Tyberg et al. (1970) suggested that the compliance of the atrium allows large changes of volume without changes of pressure. Consequently, one would be less likely to observe negative ventricular diastolic pressure in the normal situation than in an inflow limiting disease (Tyberg et al., 1970). They added that the absence of negative diastolic pressure does not necessarily imply that the ventricle is not contributing to the filling process, especially if the function of the inflow channel is normal. They also suggested that, in the presence of normal ventricular volume and afterload (aortic pressure), restoring forces in diastole are probably very small, but may still be evident.

One postulated form of active process by which the ventricle may draw blood from the atrium is the contraction of muscle fibers (Gausp, 1954). It was suggested that, owing to their anatomic arrangement, such contraction of the muscle fibers could enlarge the ventricular cavity during diastole (Gausp, 1954). Another suggested active process is the development of a force during diastole that acts to lengthen the muscle fibers upon completion of their contraction (Brecher and Galletti, 1966). Another process that may permit the ventricle to draw blood from the atrium is elastic recoil (Bloom and Ferris, 1956b; Brecher and Kissen, 1967). Such an elastic recoil would tend to restore ventricular diastolic dimensions. Perhaps myocardial fibers of dif-
fenter layers pull against each other and apply stretch to the connections between them (Rushmer et al., 1953). This action may represent potential energy which is wasted as far as systolic ejection is concerned. However, as the ventricles begin to relax, 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 distention. Therefore, in early diastole, rapid filling would be facilitated (Rushmer et al., 1953). The restoring force also may result from distortion of the intramural structure of the wall and in the heart may derive in part from compression of the ultrastructural unit of contraction, the sarcomere (Tyberg et al., 1970). If individual muscle fibers are made to contract, their sarcomeres will shorten and with relaxation will elongate themselves (Parsons and Porter, 1966). With relaxation, sarcomeres will spring back to the longer length, demonstrating a restoring force. In this sense, the shortening sarcomere overcomes a resistance of unknown magnitude and retains this energy as elastic recoil on relaxation (Tyberg et al., 1970).

A change of diameter of the left ventricle occurred in some of the dogs before the crossover of left atrial and left ventricular pressure (Fig. 1). This may have related to the role of the papillary muscle in opening the mitral valve (Marzilli et al., 1980). Opening of the mitral valve has been observed before the crossover of left atrial and left ventricular pressure (Tsakaris et al., 1978). Action of the papillary muscle may explain why opening of the mitral valve is present at that time (Marzilli et al., 1980). On the other hand, it is also possible that changes of shape of the left ventricle may occur during isovolumic relaxation. It has been shown, nevertheless, that the minor axis of the left ventricle relates directly to left ventricular volume (Rankin et al., 1980).

In conclusion, left ventricular filling during early diastole is accompanied by pressure-diameter changes that are incompatible with passive filling of the ventricle. The decreasing left ventricular pressure during rapid ventricular filling appears to reflect an active restoration of left ventricular dimensions. The nature of such restorative forces is undetermined.

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Coarctation of the Aorta and Baroreceptor Resetting

A Study of Carotid Baroreceptor Stimulus-Response Characteristics before and after Surgical Repair in the Dog

FRANZ O. IGLER, LAWRENCE E. BOERBOOM, PAUL H. WERNER, JUDITH H. DONEGAN, E.J. ZUPERKU, LAWRENCE I. BONCHEK, AND J.P. KAMPINE

SUMMARY We studied baroreceptor function in dogs before and after surgical repair of coarctation of the aorta by direct recording of multifiber carotid sinus (CS) nerve activity (NA) during alteration of pulsatile arterial pressure with systemic phenylephrine and nitroprusside, and during static pressure changes using a CS pouch preparation. Coarctation was induced by banding the proximal thoracic aorta in ten 3- to 5-day old puppies. One and one-half years later, five of these coarctated animals were studied before, and five were studied 3-7 months after, surgical repair. Five adult animals also were studied 4-6 months after the proximal thoracic aorta had been banded. Controls were eight normal adult dogs. Threshold pressure at which NA began, saturation pressure at which NA reached a maximum, and slope (% Max NA/mm Hg) of the linear portion of the stimulus-response curve were determined. Pulsatile manipulations of pressure elicited normal sensitivity (slope) in dogs with coarctation but static nonpulsatile pressure changes showed depressed sensitivity compared to controls. After surgical repair, threshold and saturation returned toward normal; sensitivity determined with static pressure manipulations returned to control value. Coarctation reset CS baroreceptors to operate at higher pressures in both puppies and adult dogs and repair of coarctation returned function toward normal. We conclude that resolution of hypertension after repair of coarctation may depend upon baroreceptor readaptation.

SEALY et al. (1957) suggested that the initial rise in systolic pressure following repair of aortic coarctation in children (paradoxical hypertension) may be due to resetting of systemic arterial baroreceptors to the chronically elevated pressure. Acutely unloading baroreceptor afferents by surgical repair would lead to an acute increase in sympathetic outflow and thus an increase in systemic arterial pressure. Rocchini et al. (1976) found that paradoxical hypertension after surgical repair of coarctation in children was biphasic in nature. An initial rise in systolic pressure within the first 24 hours after repair was related to increased sympathetic activity. The subsequent gradual rise in diastolic pressure was related to increased plasma renin activity. Upward resetting of the baroreflex heart rate response in the awake, adult canine with aortic coarctation from birth was demonstrated recently by Bonchek et al. (1976). The stimulus-response curve relating systemic arterial pressure to heart rate was shifted to the right with no detectable change in sensitivity (slope). Resetting of carotid and aortic baroreceptors to chronically elevated pressure in renal hypertension (Aars, 1976; McCubbin, 1958; Salgado and Krieger, 1973), and in the spontaneously hypertensive rat (Nosaku and Wang, 1972; Sapru and Wang, 1976) has been demonstrated.
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