Effects of Spontaneous Respiration on Canine Left Ventricular Function

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SUMMARY The purpose of this study was to determine the mechanism of the decrease in left ventricular stroke volume during spontaneous inspiration. We determined the transmural pressures of the left heart by measuring left atrial and diastolic left ventricular pressures relative to esophageal pressure. We estimated the directional changes in end-systolic and end-diastolic volumes of the left ventricle by determining the transit time of sound transmission between two ultrasonic crystals facing each other across the minor axis of the left ventricle. Left ventricular stroke volume decreased with spontaneous inspiratory effort as pleural pressure fell, regardless of whether lung volume increased or remained constant. The stroke volume was decreased during the fall in pleural pressure because of an increase in end-systolic volume with an essentially unchanged diastolic volume. Thus, the decrease in stroke volume was due to a decrease in ejection, rather than a decrease in filling of the left ventricle. We believe that left ventricular ejection was impeded by the fall in pressure around the heart relative to the pressure in the aorta. In spite of the essentially constant diastolic volume, diastolic left ventricular transmural pressure rose, suggesting that spontaneous inspiration decreases the diastolic compliance of the left ventricle. The change in diastolic compliance contributed to the decrease in stroke volume but was not the primary cause. Circ Res 45: 719–728, 1979

SPONTANEOUS inspiration is accompanied by an increase in right ventricular stroke volume (Shuler et al., 1942; Baxter and Pearce, 1951; Brecher and Hubay, 1955; Franklin et al., 1962; Hoffman et al., 1965; Morgan et al., 1966; Charlier, 1967; Guntheroth et al., 1967; Gabe et al., 1969). However, the effect of spontaneous inspiration on left ventricular stroke volume is often exactly opposite (Shuler et al., 1942; Shabetai et al., 1963; Hoffman et al., 1965; Morgan et al., 1966; Charlier, 1967; Guntheroth et al., 1967; Gabe et al., 1969; Ruskin et al., 1973; Schrijen et al., 1975; Robotham et al., 1978a). The mechanism of the increase in right ventricular stroke volume seems clear: the fall in pressure around the right heart relative to the pressure in the systemic veins increases the filling of the right ventricle (Brecher, 1956). The mechanism of the decrease in left ventricular stroke volume has been explained in a number of different ways, and there is no consensus in the current literature.

Some investigators have suggested that the apparent opposite effect on the left ventricle is due merely to a phase lag; i.e., the effect of spontaneous inspiration is an increase in right ventricular output, but there is a delay before this affects the left ventricular output (Franklin et al., 1962; Guntheroth et al., 1967; Morgan et al., 1966). Other investigators have suggested that spontaneous inspiration increases the capacitance of the pulmonary vessels and decreases the filling of the left ventricle (Charlier, 1967; Hoffman et al., 1965; Ruskin et al., 1973). Still others have considered that left ventricular filling is decreased because of compression of the left ventricle from the enlarging right ventricle (Santamore et al., 1976; Shabetai et al., 1965; Dornhorst et al., 1952). Implicit in all of these explanations is the assumption that the end-diastolic volume of the left ventricle is less during inspiration than expiration; i.e., the decrease in stroke volume is due to a decrease in filling.

The possibility that the effect of inspiration on left ventricular stroke volume is through a decrease in ejection, i.e., an increase in end-diastolic volume, rather than filling, has been given essentially no consideration. Whereas the fall in pressure around the right heart relative to the pressure in the extrathoracic veins increases the filling of the right ventricle, the fall in pressure around the left heart relative to the pressure in the extrathoracic arteries could impede the emptying of the left ventricle. Donders discussed the mechanical effect of lowering of pleural pressure on left ventricular ejection in 1853 (Donders, 1853). More recently, members of our group, on the basis of transmural pressures of the left heart in human subjects (Lichtenstein et al., 1975) and dogs (Robotham et al., 1978a; Schrijen et al., 1975), have suggested that the major effect of spontaneous inspiration on the left heart is an impediment to ejection rather than a decrease in filling.

In the present paper, we attempt to determine...
the mechanism of the decrease in left ventricular stroke volume during spontaneous inspiration through an analysis of the pressure-volume (PV) relations of the left ventricle at end-systole and end-diastole. On the basis of the transmural pressure-volume measurements of the left ventricle, we conclude that the cause of the decrease in left ventricular stroke volume in spontaneous inspiration is due principally to an impediment of ejection: the end-systolic volume was increased at constant end-diastolic volume. We believe that the increase in end-systolic volume is from the mechanical effect of the fall in pressure on the external surface of the left ventricle relative to the arterial pressure.

**Methods**

**Transmural Pressures**

Six mongrel dogs, weighing 18–32 kg, were anesthetized with intravenous pentobarbital, 30 mg/kg. Supplemental doses were given as needed. Spontaneous respiration was maintained throughout the study. A large metal cannula was inserted into the trachea through a tracheostomy. Airway pressure was measured through a port in the cannula near its outlet. Esophageal pressure was measured with a thin-walled 5-cm balloon, containing approximately 0.5 ml of air, attached to an air-filled polyvinyl catheter. The balloon was passed into the stomach under pressure monitoring and then withdrawn into the esophagus until maximum negative inspiratory pressure was observed. The aortic pressure was measured by a saline-filled polyvinyl catheter extended to the intrathoracic aorta from a femoral artery. Left atrial and left ventricular pressures were measured by a no. 7 cardiac catheter extended retrograde through the aortic valve from a carotid artery under fluoroscopic guidance. Only a single catheter was used, and thus left atrial and left ventricular pressure could not be measured simultaneously. Transmural pressures were obtained by electronic subtraction of esophageal pressure from the appropriate intravascular pressure. The dogs were maintained in a supine position. In this position, the weight of the heart on the esophagus would be expected to cause a systematic increase in esophageal pressure relative to pleural pressure, but the changes in pleural pressure with inspiration should not be affected significantly (McMahon et al., 1969).

**Left Ventricular Dimensions**

Two mongrel dogs were used for these studies. A thoracotomy was performed and ultrasonic crystals were implanted within the lumen of the left ventricle so that they faced each other across the internal minor axis. A polyvinyl catheter was placed within the left ventricular chamber and exteriorized. The thoracotomy was closed and a period of at least 4 weeks was allowed for recovery. The transit time of the ultrasonic impulses in an isolated left ventricular preparation showed an approximately linear relationship with directly measured left ventricular end-systolic and end-diastolic volume (Suga and Sagawa, 1975). The use of ultrasonic crystals to record the internal dimensions across the minor axis of the left ventricle in conscious dogs has been described previously by Horwitz et al (1968) and Bishop et al. (1969). For the respiratory studies, the dogs were anesthetized with pentobarbital, 30 mg/kg, intubated with a no. 32 endotracheal tube, and allowed to breathe spontaneously. Airway pressure was recorded through an adapter at the outlet of the endotracheal tube. Esophageal pressure was measured with a Millar catheter transducer in one dog and with a small water-filled balloon in the other.

**Mueller Manuever and Loaded Breaths**

In addition to obtaining measurements during spontaneous respiration, we also studied the effects of a Mueller maneuver by completely occluding the airway at end-expiration for one or several breaths. Under these conditions, inspiratory effort produced essentially equal falls in pleural and airway pressure with little change in lung volume. Increased inspiratory effort could be produced by partially occluding the airway for one or several breaths in sequence (loaded breaths).

**Results**

**Transmural Pressures**

The left atrial transmural pressure (LA\textsubscript{tm}) nearly always rose during spontaneous inspiration in all six dogs. Occasionally LA\textsubscript{tm} showed little change during inspiration, but it never fell. With the Mueller maneuver, LA\textsubscript{tm} increased to a greater extent than with the preceding or following spontaneous inspiration. The rise in LA\textsubscript{tm} seemed to follow the fall in pleural pressure. A typical example of the pressure responses is shown in Figure 1. During spontaneous inspiration (first and third breaths), the esophageal pressure fell while tracheal pressure remained constant. During Mueller maneuvers (second and fourth breaths), both pressures fell nearly equally.

During expiration, the esophageal pressure was slightly positive relative to atmospheric pressure and probably was somewhat higher than the actual pleural pressure. The overestimation of pleural pressure by an esophageal balloon is to be expected from esophageal elastance and the weight of the heart (McMahon et al., 1969). Nevertheless, the change in esophageal pressure probably reflected closely the change in pleural pressure, as evidenced by the comparable fall in tracheal and esophageal pressure during a Mueller maneuver. With the trachea occluded, the pleural pressure must fall by essentially the same amount as the tracheal pres-
FIGURE 1 The effect of spontaneous respiration and Mueller maneuvers on left atrial transmural pressure. Tracings from the top: aortic pressure relative to atmospheric pressure (P_a), electrocardiogram (ECG), esophageal pressure relative to atmospheric pressure (P_E), tracheal pressure relative to atmospheric pressure (P_T), left atrial pressure relative to esophageal pressure (P_LA - P_E), and left atrial pressure relative to atmospheric pressure (P_LA). P_LA - P_E is considered the transmural pressure of the left atrium (LA, in text).

sure because of the minimal change in lung volume. During a Mueller maneuver, the change in transmural pressure will be reflected accurately by subtracting airway pressure from vascular pressure (Fig. 2). A rise in left atrial pressure relative to airway pressure was found in all dogs during a Mueller maneuver; and this is strong evidence that the apparent increase in LA_un was not due to an inaccurate reflection of pleural pressure by the esophageal balloon.

Whereas LA_un was correlated closely with pleural pressure during inspiration, this was not so during early expiration. At the beginning of expiration, the pleural pressure rose rapidly, but LA_un fell slowly and still was elevated after the pleural pressure had returned to its preinspiratory level (Figs. 1 and 2).

We were not always certain of the exact location of the left ventricular end-diastolic pressure on the ventricular pressure record, so we used the lowest level of ventricular pressure in the cardiac cycle as a reflection of transmural diastolic ventricular pressure (LV_un). LV_un rose during inspiration, and the pattern was similar to that of LA_un (Fig. 3); during expiration, however, the pattern was very different. LV_un nearly always showed a significant decrease on the first beat of expiration and essentially was restored to the preinspiratory level, whereas LA_un only slowly returned.

The aortic pressure relative to atmospheric pressure was at its lowest level close to the lowest level of pleural pressure and at its highest level with the return of pleural pressure to the preinspiratory level early in expiration (Figs. 1-4). The aortic transmural pressure rose during inspiration and strikingly so during Mueller maneuvers (Fig. 4). Like LA_un, the aortic transmural pressure only slowly returned.

FIGURE 2 The effect of a Mueller maneuver on left atrial pressure relative to tracheal pressure (P_LA - P_T), which is equal to the change in left atrial transmural pressure.
FIGURE 3. The effect of spontaneous respiration and a Mueller maneuver on the minimal left ventricular pressures for each cardiac cycle \( (P_{LV}) \). Tracheal pressure is not shown, but the third breath was a Mueller maneuver.

to the preinspiratory level at the beginning of expiration.

**Left Ventricular Internal Diameter \( (\Psi) \)**

During steady state conditions, the length of the minor axis of the left ventricular chamber at end-systole \( (\Psi_S) \) always increased during spontaneous inspiration. The length of the minor axis at end-diastole \( (\Psi_D) \) showed much less change, so that the major cause of the decrease in the stroke dimension \( (\Psi_{DS}) \) was the increase in \( \Psi_S \) (Fig. 5). The increase in \( \Psi_S \) during inspiration and Mueller maneuvers was correlated closely with the fall in pleural pressure, and the highest level of \( \Psi_S \) occurred at the lowest level of pleural pressure. During expiration, \( \Psi_S \) fell more slowly than the pleural pressure and remained elevated after the pleural pressure had returned to the preinspiratory level. Although \( \Psi_D \) changed little during inspiration, there was a sharp rise in \( \Psi_D \) after inspiration ceased, and the peak \( \Psi_D \) occurred early in expiration.

The timing of the changes in \( \Psi_S \) and \( \Psi_D \) are shown in greater detail in Figure 6. Although the paper speed of the recorder was slow, the outlet of the endotracheal tube was constricted, and there followed a series of inspirations with increasing effort. The esophageal pressure was measured with a small water-filled balloon. Although the signal-to-noise ratio of the esophageal pressure was not great, the water-filled system ensured accuracy of timing. Between the fourth and fifth loaded inspirations, the speed of the recorder was increased so that one breath could be analyzed in detail. During this breath, there was a steady increase in \( \Psi_S \) as esoph-

**FIGURE 4.** The effect of spontaneous respiration and a Mueller maneuver on \( P_{ao} \) and relative to esophageal pressure \( (P_{ao} - P_{Es}) \). The arrow points to the breath during the Mueller maneuver.
The effect of spontaneous respiration on the dimensions of the internal minor axis of the left ventricle ($\phi$) along with left ventricular pressure, esophageal pressure, and tracheal pressure relative to atmospheric pressure. $\phi_D$ = maximum diameter during diastole; $\phi_S$ = minimum diameter during systole. The arrow points to a Mueller maneuver. The Mueller maneuver is characterized by a nearly equal fall in tracheal and esophageal pressure. The changes in tracheal pressure in the other breaths was due to resistance in the endotracheal tube.}

There was essentially no change in $\phi_D$ during inspiration. Nevertheless, in the four preceding loaded breaths, it can be seen that each decrease in esophageal pressure was followed by an increase in $\phi_D$ to a higher level.

In Figure 7 are shown two episodes of constriction of the outlet of the endotracheal tube. These episodes are indicated by the increased tracheal pressure swings. Each loaded breath was accompanied by an abnormally large increase in $\phi_S$, followed by a rise in $\phi_D$ with the peak of the latter not occurring until early expiration.

Independent of respiratory effects, $\phi_S$ showed the expected changes in relation to the two factors that are known to have an influence: (1) myocardial contractility and (2) the level of systemic blood pressure.

The iv administration of a negative inotropic agent, propranolol, 1.5 mg/kg, caused a progressive increase in $\phi_S$ (Fig. 8). It is of interest that the respiratory effect also was greater. This is clearly demonstrated by comparing the second inspiration, which occurred before the propranolol was given, with the 12th, 13th, and 15th inspirations from the
beginning of the record. These latter inspirations followed the administration of propranolol. They are selected for comparison with the second breath because the change in pleural pressure was nearly identical for all four breaths. Not only was there a greater $\Phi_s$ at end-expiration, but the change with inspiration was considerably greater.

Occasionally transient changes in left ventricular pressure were observed (Fig. 9). These changes often accompanied a shift in position or a movement of the head of the dog. We do not know the causes of these sudden changes. It is possible they were due to reflex changes in arteriolar resistance. Whatever the causes, we find the changes in $\Phi_s$ were correlated closely with changes in left ventricular systolic pressure. Note that following the second transient drop in ventricular pressure, the ventricular pressure did not return to the level before the transient change. It can be seen that $\Phi_s$ also remained at a lower level in the new steady state.

**Discussion**

**End-Systolic Respiratory Changes**

The data demonstrate that spontaneous inspiration was associated with an increase in the length of the minor axis of the left ventricle at end-systole. The increase in $\Phi_s$ was not dependent on the increase in lung volume, for it also occurred during Mueller maneuvers. If the increase in $\Phi_s$ reflects an increase in the volume of the left ventricle at end-systole, then the mechanism of the increase probably is the increase in the transmural pressure of the left ventricle at end-systole from the decrease in the pressure around the heart; i.e., the fall in pleural pressure. In early expiration, $\Phi_s$ remained elevated, even though the pleural pressure had returned to the preinspiratory level; but at this time the transmural pressure was elevated, as reflected by the increase in systemic blood pressure relative to atmospheric pressure.
The linear dimensions of the minor axis of the left ventricle correlated well with ventricular volume in an isolated heart preparation (Suga and Sagawa, 1975), but the possibility remains that the shape of the heart was changed by respiration. After completion of the studies, we placed the isolated heart in a box and demonstrated that lowering the pressure around the heart by only a few centimeters of H₂O, relative to aortic pressure, caused a significant increase in directly measured end-systolic volume. In the isolated heart preparation, the aortic valve was open at all times so that there was no isovolumic contraction or relaxation. From the simultaneous tracings of the length of the minor axis and ventricular pressure in Figure 6, it appears that there were small changes in the length of the minor axis during isovolumic contraction and relaxation. These changes did not prevent the directional change in 0s from following the expected change in volume when arterial blood pressure or contractility was changed (Figs. 8 and 9).

There is evidence that the volume of the right ventricle can influence the pressure-volume characteristics of the left ventricle during diastole, which we shall discuss later, but there is no evidence that a change in right ventricular volume has a significant influence on the pressure-volume characteristics of the left ventricle at end-systole. Indeed, Kelly et al. (1971) found considerable changes in diastolic pressure-volume relations of the left ventricle from right ventricular volume loading, but no changes at end-systole. In the isolated heart preparation, marked changes in right ventricular volume had essentially no influence on the end-systolic volume.

On the basis of the available evidence, we conclude that the change in 0s with inspiration was in the same direction as the change in ventricular volume.

The increase in end-systolic volume with spontaneous inspiration could result from a decrease in myocardial contractility or an impediment to ejection. The changes are too quick for any of the known reflex effects on contractility (Aviado and Schmidt, 1955). Although there is evidence that the increase in right ventricular volume occurring with inspiration can alter left ventricular contractility, as judged by the rate of shortening and pressure change (Elzinga et al., 1974; Kelly et al., 1971), the PV relations at end-systole are not affected (Kelly et al., 1971). There is, however, a sound theoretical basis for a mechanical impediment to emptying of the left ventricle resulting from a reduction in pleural pressure.

To gain insight into how the end-systolic volume would be expected to increase with a lowering of pleural pressure, consider the mechanical effects of a step decrease in pleural pressure at the beginning of diastole with constant lung volume. The immediate effect on all intrathoracic vascular pressures before there had been time for any volume shift would be a reduction in intravascular pressure by the same amount as the fall in pleural pressure (before a volume shift, the transmural pressure would remain constant). The fall in right atrial pressure would shift blood to it from the extrathoracic veins, and the transmural pressure of the right atrium and right ventricle would increase. At the same time, the decrease in pressure in the intrathoracic aorta would decrease the rate of flow from it toward the extrathoracic arteries. Thus, at the beginning of the next systole, the volume of blood in the intrathoracic aorta and its transmural pressure would be at a greater level than for the previous systole. The net effect on the next systole would be similar to that produced by constricting the descending aorta, which would also have produced an increase in the volume and transmural pressure of the aorta upstream from the constriction. At the beginning of the next systole, the volume of blood in the lungs and left heart would be the same as on the previous systole, for there would be no alteration in the distribution of volume between the closed pulmonary and aortic valves. Wilcken et al. (1964) showed that, for the first systole following inflation of a balloon in the descending aorta during diastole, the stroke volume was reduced from an increase in end-systolic volume. Of significance was that a similar increase in end-systolic volume occurred on the rapid infusion of blood into the aorta.

The similarity of a reduction of pleural pressure and a constriction in the aorta has led us to consider the reduction in pleural pressure as equivalent to an increase in left ventricular afterload (Lichtenstein et al., 1975; Robotham et al., 1978a; Schrijen et al., 1975). However, the use of the term afterload in relation to a reduction in pleural pressure, where the arterial blood pressure falls relative to atmospheric pressure, seems more often to confuse than clarify, so we have avoided its use in this paper.

The same mechanical forces responsible for the increased right ventricular output impede the output of the left ventricle. This was understood by Donders in 1853: "Forceful inspiration strongly sucks the venous blood into the thorax; forceful expiration, on the contrary, tends to expel venous blood from the thorax. Those factors which are promoting for venous circulation impede arterial circulation, and vice versa."

The effect of a reduction in pleural pressure on the venous circulation as a cause of the circulatory effects of respiration has never been in doubt since the work of von Haller in 1751 (Brecher, 1956). Yet, the impeding of left ventricular output through a reduction in pleural pressure hardly ever has been considered in the modern literature to account for the circulatory effects of respiration on the left heart. This lack of consideration must, in part, be due to conceptual problems: the mechanics of ejection are more complicated than the mechanics of filling, and there is considerable debate and confusion as to how the term afterload should be defined.
End-Diastolic Respiratory Changes

During inspiration, LA\textsubscript{im} and LV\textsubscript{im} increased out of proportion to the increase in \(\Phi_0\). The transmural pressure of the left ventricle nearly always increased, but there was essentially no change in \(\Phi_0\) until expiration began. These findings provide strong evidence that spontaneous inspiration is associated with a significant change in the elastic properties of the left ventricle during diastole. They are compatible with data for isolated hearts which have shown that an increase in right ventricular volume causes an increase in the transmural pressure of the left ventricle at the same volume (Santamore et al., 1976; Laks et al., 1967).

The changes in LA\textsubscript{im} are nearly identical to those obtained by Opdyke and Brecher (1950) for deep inspirations. Both our findings, and those of Opdyke and Brecher, were somewhat different from those of Coleridge and Linden (1959) for the beginning of inspiration. Coleridge and Linden reported that the left atrial transmural pressure remained steady or fell at the beginning of inspiration, but always increased as the inspiration was continued.

During early expiration, LA\textsubscript{im} remained elevated, but there was an immediate decrease in LV\textsubscript{im} toward its prespiratory level. These findings suggest that there was an essentially unchanged left ventricular volume during inspiration, and the rapid drop in transmural pressure in early expiration resulted from the decrease in right ventricular volume. The continued elevation of LA\textsubscript{im} during early expiration suggests that the left atrium was enlarged during inspiration. There are three additive factors that tend to enlarge the left atrium during inspiration: (1) the decrease in ejection from the left ventricle, (2) the "stiffening" of the left ventricle from the mechanical interdependence of the two ventricles, and (3) the increase in output from the right ventricle. Diastolic left ventricular volume, as reflected through \(\Phi_0\), increased considerably during early expiration because the increased volume of blood in the left atrium and pulmonary veins which had been held back during inspiration now could effectively fill the more compliant left ventricle. The significant increase in left ventricular filling during early expiration was probably responsible in part for the increased arterial blood pressure through a rapid increase in stroke volume.

Although the major cause of the decrease in stroke volume was the increase in end-systolic volume, the mechanical interdependence between the ventricles at end-diastole must have contributed significantly. If there were no decrease in end-diastolic compliance of the left ventricle, the increase in end-systolic volume would have led to a greater increase in end-diastolic volume and less reduction in stroke volume. The work of Shabetai et al. (1963, 1965) provides evidence that the increased pulsus paradoxus with cardiac tamponade is the result of the increased interdependence. They first showed that, during inspiration with tamponade, there was an even greater fall in left ventricular stroke volume than under normal conditions (Shabetai et al., 1963). In a subsequent study, they were able to...
abolish the exaggerated fall in blood pressure (and presumably stroke volume) with a right heart bypass which prevented the inspiratory increase in right ventricular volume (Shabetai et al., 1965). Even with the right heart bypassed, however, there was still a fall in blood pressure with inspiration under non-tamponade conditions. Thus, the exaggerated decrease in inspiratory stroke volume appears to be dependent on the right ventricle, but enlargement of the right ventricle is not the primary cause of the decrease. Recent studies in our laboratory (Robotham et al., 1978a) show that a Mueller maneuver is associated with a significant decrease in left ventricular stroke volume under conditions in which the right ventricle is empty and pulmonary arterial flow held constant. Interestingly, the decrease in stroke volume was not associated often with a significant rise in LA and LV. These findings suggest that the major cause of the rise in left heart transmural pressure during inspiration is not the decrease in ejection of blood from the left ventricle, but the mechanical interdependence of the ventricles and the increased output of the right ventricle, both of which were absent in these studies.

The Effect of an Elevation of Pleural Pressure on Left Ventricular Function

If a decrease in pleural pressure can impede left ventricular ejection, an increase should do the opposite, at least in terms of end-systolic volume. Perhaps the most dramatic demonstration of the effectiveness of an increase in pleural pressure on enhancing left ventricular output is under conditions of the nonbeating heart. Rudikoff et al. (1977) have shown that in cardiac resuscitation external thoracic compression ejects blood from the left ventricle through an increase in pleural pressure and only infrequently from the direct mechanical compression of the heart by the sternum. An even more striking demonstration is the ability of a patient with ventricular fibrillation to maintain consciousness with frequent and vigorous coughs (Cri- ley et al., 1976). In artificial ventilation, the pleural pressure rises with inspiration, and it would be expected on the basis of the present work that end-systolic volume would decrease. Charlier (1967) found that artificial ventilation caused an increase in left ventricular stroke volume. A similar increase occurred with a forced expiration when the pleural pressure was increased.

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Interaction between Cardiac Receptors and Sinoaortic Baroreceptors in the Control of Efferent Cardiac Sympathetic Nerve Activity during Myocardial Ischemia in Dogs

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SUMMARY The purpose of this study was to determine the relative influence of arterial baroreceptors and of cardiac receptors with vagal afferents on efferent cardiac sympathetic nerve activity during coronary artery occlusion. Changes in heart rate (beats/min), arterial pressure (mm Hg), and integrated cardiac sympathetic nerve activity (CSNA, percent change from control; recorded from the cut central end of the left ventral ansa subclavia) were determined during transient (90-second) circumflex (Cx) and anterior descending (LAD) coronary artery occlusions. In dogs with carotid and aortic baroreceptors intact, increases (mean ± SE) in CSNA during Cx (7 ± 2%) and LAD (9 ± 5%) occlusions were similar despite a significantly greater fall in arterial pressure during Cx (-14 ± 3 mm Hg) than during LAD (-5 ± 2 mm Hg) coronary artery occlusion. Heart rate did not change during these occlusions. In three dogs, hypotension induced by inferior vena cava occlusion resulted in greater increases in CSNA than did comparable decreases in arterial pressure resulting from occlusion of LAD or Cx. In dogs with sinoaortic denervation, Cx coronary occlusion resulted in decreases in CSNA (-14 ± 4%), arterial pressure (-38 ± 6 mm Hg), and heart rate (-13 ± 5 beats/min), whereas LAD occlusion resulted in a small decrease in arterial pressure (-12 ± 5 mm Hg) and no change in CSNA or heart rate. Vagotomy abolished the decreases in CSNA and heart rate and attenuated the arterial pressure responses to Cx occlusion. We conclude that cardiac receptors with vagal afferents exert an inhibitory influence on cardiac sympathetic nerve activity during myocardial ischemia, particularly during inferoposterior ischemia, and that this influence limits the arterial baroreceptor-mediated increases in CSNA resulting from ischemia-induced hypotension.

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AUTONOMIC reflex mechanisms activated during myocardial ischemia may significantly alter electrical and mechanical aspects of cardiac function. In humans and in unanesthetized nonhuman primates, bradycardia and hypotension occur frequently during inferior myocardial infarction, whereas tachycardia and increases in arterial pressure more commonly result from anterior myocardial infarction (Webb et al., 1972; Randall et al., 1978). Several factors may contribute to these distinctly different responses to ischemia of different regions of the left ventricle. First, the number of cardiac sensory receptors activated during coronary occlusion may depend on the specific region of the heart that is rendered ischemic. Thames et al. (1978b) have shown that inhibitory cardiac receptors with vagal afferents, which are activated during myocardial ischemia, are distributed preferentially to the inferoposterior left ventricle of the dog.

Second, the influence of three receptor groups
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