The Cardiocardiac Sympathetic Reflex during Coronary Occlusion in Anesthetized Dogs

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SUMMARY  Cardiac sympathetic afferent fibers activated during coronary occlusion exert an excitatory influence on sympathetic discharge to the heart in cats after spinal cord section. The significance of this cardiocardiac sympathetic reflex response during myocardial ischemia in animals with an intact neuraxis is unknown. We studied the responses of efferent cardiac sympathetic nerve activity (CSNA), arterial pressure, and heart rate to coronary occlusion in two groups of dogs with cardiac sympathetic reflexes intact and with other reflex inputs affecting CSNA sectioned or controlled. In group I \( n = 10 \), the vagi were sectioned, the spinal cord remained intact, and the carotid sinuses were isolated and perfused to maintain baroreceptor input constant. Coronary occlusion was performed at moderate and low basal levels of CSNA by setting carotid sinus pressure at 125–150 and 200 mm Hg, respectively.

Under these conditions, CSNA was not altered by occlusion of either the anterior descending or the circumflex coronary artery. In group II \( n = 4 \), the vagi were sectioned and the spinal cord was interrupted. In these dogs, CSNA increased significantly \((61 \pm 19\%)\) during coronary occlusion. These results show that an excitatory cardiocardiac sympathetic reflex can be demonstrated in dogs with spinal cords sectioned but not with spinal cords intact. This finding is consistent with the view that inhibitory bulbospinal pathways minimize the influence of the spinal cardiocardiac sympathetic reflex during myocardial ischemia in anesthetized dogs. 

ACUTE myocardial ischemia may result in enhanced sympathetic discharge to the heart in man (Webb et al., 1972) and in experimental animals (Malliani and Lombardi, 1978; Felder and Thames, 1979). Although the factors contributing to this excitatory response remain controversial (Malliani, 1980; Felder and Thames, 1980), previous investigators (Malliani et al., 1969; Malliani and Lombardi, 1978; Brown and Malliani, 1971) have attributed it to activation during ischemia of a spinal cardiocardiac sympathetic reflex.

Much of the evidence cited to support a role for the cardiocardiac sympathetic reflex during ischemia has been obtained under non-ischemic conditions by employing electrical or chemical stimulation of cardiac sympathetic afferent fibers in experimental animals. Electrical stimulation of cardiac sympathetic afferent fibers has been shown to result in increases in heart rate (Malliani et al., 1973), in left ventricular contractility (Malliani et al., 1972), and in arterial blood pressure (Peterson and Brown, 1971). Bradykinin applied to the left ventricular epicardium activates cardiac receptors with sympathetic afferent fibers (Uchida and Murao, 1974a) and elicits an excitatory cardiocardiac reflex response (Staszewska-Barczak et al., 1976). Myocardial ischemia also has been shown to activate these cardiac receptors with sympathetic afferent fibers (Uchida and Murao, 1974b; Bosnjak et al., 1979). However, the occurrence of an excitatory cardiocardiac sympathetic reflex response to the stimulus of coronary occlusion has been demonstrated convincingly only after spinal cord section (Malliani et al., 1969; Brown and Malliani, 1971; Malliani and Lombardi, 1978). Since cord section interrupts important supraspinal inhibitory influences on preganglionic sympathetic discharge (Wurster, 1977; Dembowsky et al., 1979), the physiological significance of an excitatory reflex demonstrated under such conditions remains unclear.

In sinoaortic denervated vagotomized dogs with neuraxis intact and with sympathetic reflexes uninterrupted, coronary occlusion failed to elicit a cardiocardiac sympathetic reflex (Felder and Thames, 1979), even though a cardiac sympathetic reflex response to bradykinin or electrical stimulation was readily demonstrated (unpublished observations). However, the basal levels of sympathetic discharge may have been near maximal in those dogs, making it difficult to detect further small increases in sympathetic discharge to the heart during coronary occlusion. In the present experiments, the influence of the cardiac sympathetic afferent fibers on changes in sympathetic discharge to the heart during coronary occlusion was studied in dogs with intact or interrupted neuraxis. In experiments with the neuraxis intact, basal activity in...
the cardiac sympathetic nerves was maintained at moderate or low levels during coronary occlusion to increase the likelihood of observing an excitatory cardiovascular sympathetic reflex (Malliani and Lombardi, 1978).

**Methods**

Nineteen dogs weighing 20–33 kg were anesthetized with sodium thiopental (30 mg/kg, iv) and alpha chloralose (80 mg/kg, iv), and maintained with hourly doses of alpha chloralose (10 mg/kg). The animals were intubated and mechanically ventilated with room air supplemented with oxygen. Arterial blood gases were measured and PCO₂ and pH were corrected when necessary by adjustments of the tidal volume or administration of sodium bicarbonate, respectively. Body temperature was maintained by external warming. Estimated fluid losses resulting from surgery were replaced by 6% dextan in normal saline (5 ml/kg, iv) prior to initiating the protocols.

**Experimental Preparation**

A midline cervical incision was made to expose the vagi and carotid arteries bilaterally. The vago-sympathetic trunks, including the aortic nerves, were sectioned. All animals underwent a left thoracotomy with removal of the third and fourth ribs. The pericardium was opened, and the anterior descending and circumflex coronary arteries were exposed 1 to 2 cm from their origins. Care was taken to avoid damaging the nerves which course near these vessels. A snare was placed loosely around each vessel for subsequent coronary occlusion.

With the aid of a dissecting microscope, the ventrolateral cardiac nerve, which contains mainly efferent cardiac fibers (Armour and Randall, 1975), was identified as it coursed adjacent to the left thoracic vagus nerve and was dissected free from surrounding connective tissue. A long segment of the nerve was isolated to allow it to be repositioned in a stable location away from the heart and great vessels. The nerve was cut distally (before it branched into multiple small nerves supplying the heart) and the nerve sheath was removed. Nerve activity was recorded from the whole ventrolateral cardiac nerve or from fibers that were obtained from the nerve. Further surgical preparation depended on the experimental group to which the animal was assigned.

**Group 1 (Spinal Cord Intact)**

In 15 dogs, the spinal cord was left intact and the carotid sinuses were isolated from the circulation and perfused to permit control of basal sympathetic activity. The carotid sinuses were isolated by ligating the internal carotid, occipital, ascending pharyngeal, and small tributary vessels arising from the carotid sinus regions. Both carotid sinuses were perfused at constant flow using variable speed, peristaltic pumps (Harvard apparatus) with arterial blood obtained from the left femoral artery. Blood entered the isolated sinuses through the common carotid arteries and left the sinuses through the external carotid arteries to be returned to the animal through the external jugular veins. Starling resistors in the return lines provided a means of controlling pressure in the isolated sinuses. Sodium heparin (10,000 U, iv) was administered prior to perfusion of the carotid sinuses.

**Group 2 (Spinal Cord Interrupted)**

In four dogs, the spinal cord was destroyed by compression at the C1-C2 level in the following manner: the carotid and vertebral arteries were tied bilaterally in the neck to eliminate most of the circulation to the cervical spine and thus minimize bleeding during cord interruption. The spinal cord was approached through a dorsal incision over the C1-C2 interspace. With the head flexed, the connective tissue between the vertebral bodies was removed, leaving the dura intact. A suture was passed around the cord extradurally and tied tightly to compress the spinal cord. This suture then was lifted gently to visualize the spinal cord, and a second suture was placed around the cord and tied. Successful interruption of the spinal cord was accompanied by a characteristic rise in arterial pressure, followed by a fall in pressure to hypotensive levels. Arterial pressure was supported when necessary with norepinephrine (10 μg/ml at 0.1–1.0 ml/min) by constant infusion (Harvard pump).

**Nerve Recordings**

In each experiment the ventrolateral cardiac nerve was positioned on a dissecting stage and covered with a mineral oil pool to prevent dehydration. The stage was placed in a stable location away from vascular structures to eliminate movement artifact. Decamethonium bromide (0.3 mg/kg, iv) was administered to prevent muscle movement during the recording of nerve activity. Nerve traffic was recorded using silver/silver chloride or platinum-iridium electrodes connected to a Grass probe (HIP 511E) and amplified by a Grass (P 511) band-pass amplifier. The high frequency cutoff was set at 1000–3000 Hz and the low frequency cutoff at 30 Hz. The amplifier output was audible over a loudspeaker and visible on a Tektronics (D13) dual beam storage oscilloscope. The output was also led into a nerve traffic analyzer which counted spikes exceeding a selected voltage. Each action potential which exceeded the voltage setting of the window discriminator was rectified and integrated so that quantification of nerve traffic was independent of individual spike amplitude (Fig. 1, top). The counter was digital in design and the relationship between integrator output and spike frequency was linear up to a frequency of 10 kHz. The recorded spike activ-
Figure 1 Photographs of oscilloscope tracings illustrating the operation of the nerve traffic analyzer. Each record shows integrated nerve activity, sympathetic nerve spike potentials, and arterial pressure. Top was recorded at a rapid sweep speed to illustrate the manner in which the nerve traffic analyzer integrates action potentials exceeding the voltage setting of the cursor (indicated by the arrow). Bottom was recorded at slower speed to demonstrate the direct relationship between the number of action potentials and the integrator output as seen in Figure 2.

Three variables were recorded continuously before, during, and after the subsequent experimental interventions.

Hemodynamic Measurements

Arterial pressure was measured with a catheter in the right femoral artery connected to a Statham (P23dB) transducer. Mean arterial pressure was obtained by electrical averaging. Heart rate was monitored by a cardiotachometer triggered by the QRS complex of the electrocardiogram.

Protocols

After completion of the surgical preparation, adequate time was allowed for stabilization of heart rate, arterial pressure, and nerve activity. In the experiments on spinal sectioned animals, two to three hours elapsed between cord section and the initiation of coronary occlusion experiments. These three variables were recorded continuously before, during, and after the subsequent experimental interventions.

Group 1 experiments were designed to determine the responses to coronary occlusion performed at moderate and low levels of baseline efferent sympathetic activity with spinal cord intact. Figure 2 illustrates the manner in which these experiments were performed. Control measurements of arterial pressure, heart rate, and nerve activity were obtained for each experiment with carotid sinus pressure (CSP) at 50 mm Hg, a level at which carotid baroreceptors exert minimal inhibitory influence on the vasomotor centers. The pressure in both carotid sinuses was then elevated to 125-150 mm Hg, resulting in moderate reductions in arterial pressure and nerve activity. After these variables had stabilized for at least 30 seconds, transient (90-second) coronary occlusion was performed. Following release of the occlusion and an additional period of observation (at least 30 seconds), carotid sinus pressure was returned to 50 mm Hg. In a similar manner, a low baseline level of efferent cardiac sympathetic discharge was obtained by raising carotid sinus pressure to 200 mm Hg. Coronary occlusions thus were performed at both moderate (CSP = 125-150 mm Hg) and low (CSP = 200 mm Hg) basal levels of efferent cardiac sympathetic discharge. Sham experiments, which consisted of raising carotid sinus pressure for a comparable time interval without occluding a coronary artery, were performed prior to each coronary occlusion experiment.

Raising carotid sinus pressure to 200 mm Hg usually resulted in marked hypotension, so that the effects of reduced perfusion of the heart and of the central nervous system could have influenced the responses to coronary occlusion. To exclude that possibility, additional coronary occlusion experiments were performed in dogs with carotid sinus pressure set at 200 mm Hg but with arterial pressure maintained at or above control levels by a constant intravenous infusion of phenylephrine (100 μg/ml at 1-3 ml/min).

Group 2 experiments were performed to determine whether a cardiocardiac reflex was present in vagotomized dogs with the spinal cord interrupted. In these four dogs, efferent cardiac sympathetic activity was recorded during transient (1-2 minute) occlusions of the anterior descending (n = 3) or circumflex (n = 4) coronary arteries.

Data Analysis

Efferent cardiac sympathetic nerve activity, arterial pressure, and heart rate were measured continuously throughout each experiment. The intervals during which average responses for these values were determined for the group 1 experiments are shown in Figure 2. For each experiment in which carotid sinus pressure was raised and a coronary artery was occluded, a sham procedure was performed in which carotid sinus pressure was raised for a comparable period of time, but the
coronary artery was not occluded. Thus, a matched pair (experimental vs. sham) of observations was available for each occlusion in each dog. The following statistical comparisons were made (refer to Fig. 2): (1) within each set of observations (experimental or sham), the absolute values of nerve activity, arterial pressure, and heart rate were compared before and after carotid sinus pressure was raised, and before and after carotid sinus pressure was returned to control level; (2) within each set of observations (experimental or sham), the absolute values of these variables during coronary occlusion at elevated carotid sinus pressure were compared with the values at the same carotid sinus pressure immediately before and immediately after coronary occlusion. In the case of the sham procedures, measurements obtained at times comparable to those before, during, and after the coronary occlusion were compared; (3) the absolute values for each interval of the coronary occlusion experiments were compared with the absolute values for the corresponding intervals of the paired sham procedures (experimental vs. sham); (4) the responses to the coronary occlusion and sham experiments at CSP 125–150 mm Hg were compared with the responses to the coronary occlusion and sham experiments at CSP 200 mm Hg.

For Group 2, the data reported represent averaged values obtained over a 30-second interval prior to occlusion and over the 30-second interval of maximal response during coronary occlusion.

The values obtained from these experiments were subjected to statistical analysis of variance (Morrison, 1967), with differences considered significant at \( P < 0.05 \). Data are presented in the text and figures as the mean ± SE.

Results

Group 1

In 10 dogs with aortic and vagal nerves sectioned and carotid sinuses isolated and perfused, responses to anterior descending and circumflex coronary artery occlusion were obtained for eight experiments at CSP = 125–150 mm Hg, and six at CSP = 200 mm Hg (Figures 3 and 4). In these figures, the data for nerve activity are normalized and represented as a percent change from control. This method of presentation was used because the absolute nerve activity varied from one dog to another, depending on the number of active fibers on the recording electrodes. However, the absolute values reported in the text were used for all statistical comparisons. When carotid sinus pressure was raised from 50 to 125–150 mm Hg prior to anterior descending or circumflex coronary artery occlusion (Fig. 3, filled symbols), efferent cardiac sympathetic nerve activity decreased moderately but significantly to steady state levels (LAD experiments = 41.7 ± 22.0 to 28.0 ± 15.7 impulses/sec; Cx experiments = 32.8 ± 13.3 to 20.7 ± 8.8 impulses/sec). This was accompanied by significant decreases in arterial pressure (101 ± 8 to 60 ± 3 mm Hg and 100 ± 7 to 60 ± 3 mm Hg for LAD and Cx experiments, respectively) and in heart rate (LAD experiments = 185 ± 8 to 173 ± 9 beats/min; Cx experiments = 187 ± 4 to 175 ± 7 beats/min).
CARDIOCARDIAC SYMPATHETIC REFLEX/Felder and Thames

Increasing carotid sinus pressure from 50 to 200 mm Hg prior to anterior descending or circumflex coronary occlusion (Fig. 4, filled symbols) resulted in marked reductions in nerve activity (32.5 ± 13.3 to 8.0 ± 4.2 impulses/sec and 27.9 ± 9.7 to 3.2 ± 2.1 impulses/sec for LAD and Cx experiments, respectively), arterial pressure (LAD experiment = 87 ± 6 to 45 ± 2 mm Hg; Cx experiments = 87 ± 4 to 45 ± 3 mm Hg), and heart rate (LAD experiments = 182 ± 11 to 148 ± 16 beats/min; Cx experiments = 181 ± 9 to 149 ± 12 beats/min). These reductions were significantly greater than those observed when CSP was raised from 50 to 125-150 mm Hg. Even at this low baseline level of sympathetic activity, coronary occlusion did not further alter cardiac sympathetic discharge, heart rate, or blood pressure. The values for these variables during coronary occlusion were not different from the values before occlusion and after release of occlusion at CSP 200 mm Hg. There was no significant difference between the responses to increasing carotid sinus pressure alone (sham occlusion) and the responses to raising CSP to 200 mm Hg and occluding the circumflex or anterior descending coronary artery (experiment). When CSP was returned to 50 mm Hg, all three variables returned to values that were not different from those previously observed when CSP was 50 mm Hg.

In five additional dogs, carotid sinus pressure was raised from 50 to 200 mm Hg but hypotension was prevented by infusing phenylephrine intravenously. In these animals, the mean arterial pressure and heart rate immediately before coronary occlusion with carotid sinus pressure set at 200 mm Hg (144 ± 15 mm Hg; 160 ± 7 beats/min) were similar to those observed in the control period with carotid sinus pressure set at 50 mm Hg (122 ± 13 mm Hg; 152 ± 9 beats/min). Cardiac sympathetic nerve activity was greatly reduced from 9.4 ± 1.6 impulses/sec with carotid sinus pressure at 50 mm Hg to 0.6 ± 0.2 impulses/sec with carotid sinus pressure at 200 mm Hg. There was no further change in cardiac sympathetic nerve activity during circumflex (n = 5) or anterior descending (n = 3) coronary occlusion.

Group 2

Responses to circumflex occlusion and to anterior descending occlusion were obtained in four dogs with the vagal and aortic nerves sectioned and spinal cord interrupted. Interruption of the cervical cord eliminated the influence of the carotid sinus baroreceptors as well as all other descending pathways. A representative record from a coronary occlusion experiment in a vagotomized spinal dog is shown in Figure 5. During circumflex occlusion (n = 4), arterial pressure decreased (87 ± 7 to 69 ± 7 mm Hg) and nerve activity increased (8.3 ± 1.3 to 13.1 ± 2.8 impulses/sec) significantly. Heart rate increased during circumflex occlusion in one animal (132 to 162 beats/min) but did not change in the
FIGURE 4 Changes (top to bottom) in efferent cardiac sympathetic nerve activity, mean arterial pressure, and heart rate during anterior descending (filled circles) and circumflex (filled triangles) coronary occlusion at low levels of basal cardiac sympathetic discharge (CSP 200 mm Hg). The arrows indicate the responses during the period in which each coronary artery was occluded. The unfilled symbols show the responses to raising CSP alone (sham occlusion). The vagal and aortic nerves were sectioned prior to these experiments. Data shown as mean ± SE. The absolute values for each variable at the control carotid sinus pressures of 50 and 200 mm Hg are given in the text (Results section).

other three (180 ± 30 to 184 ± 34 beats/min). Anterior descending occlusion (n = 3) also resulted in significant decreases in arterial pressure (78 ± 6 to 65 ± 7 mm Hg) and increases in sympathetic nerve activity (8.1 ± 0.6 to 13.1 ± 3.4 impulses/sec) but heart rate did not change (186 ± 39 to 186 ± 39 beats/min). The increases in nerve activity during coronary occlusion in these dogs stand in striking contrast to the responses of group I dogs. The onset and duration of these excitatory sympathetic responses to coronary occlusion were variable. In some cases, efferent cardiac sympathetic activity increased within a few seconds of occlusion and returned to control levels soon after release. In others, the nerve traffic increased after the vessel had been occluded for as long as 45 seconds and was sustained for as long as 3 minutes after release.

In two spinal animals, repeated occlusion of the same vessel resulted in similar changes in all measured variables.

Discussion

Excitatory responses to myocardial ischemia have been previously observed in humans (Webb et al., 1972) and in conscious animals (Peterson and Bishop, 1973; Randall et al., 1978), particularly during anterior ischemia. There is substantial evidence that myocardial ischemia activates cardiac receptors with sympathetic afferent fibers (Brown, 1967; Uchida and Murao, 1974b; Bosnjak et al., 1979).

Although cardiocardiac sympathetic reflex responses to chemical and electrical stimulation of sympathetic afferent fibers have been observed in animals with neuraxis intact (Peterson and Brown, 1971; Malliani et al., 1972, 1973; Staszewska-Barczak et al., 1976) the studies that document excitatory reflex responses to myocardial ischemia mediated by a spinal cardiocardiac sympathetic pathway have been performed exclusively in cord-sectioned animals (Malliani et al., 1969; Malliani and Lombardi, 1978). Since spinal cord section interrupts important descending inhibitory pathways.
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patients with angina caused by coronary artery elevation with no change in heart rate and a de-

and blocked by crushing the stellate ganglia and stimulating the cardiac sympathetic afferent fibers
do affective reaction, which can be reproduced by 

the primary function of these receptors may be to 
ing ischemia? Previous studies have suggested that 
receptors influence the cardiovascular system dur-

thealyzed cats, coronary occlusion results in a pseu-
do ischemia to higher cerebral centers. In lightly anes-
thetic discharge to the heart is altered when the 
carotid baroreceptor fibers are acti-
vated by myocardial ischemia. These findings stand
in striking contrast to the marked increases in af-
f erent cardiac sympathetic nerve activity during 
coronary occlusion which have been reported by 
others (Brown, 1967; Uchida and Murao, 1974; Bo-
snjak et al., 1979) and observed in our laboratory (unpublished observations). The studies in animals 
following spinal cord interruption show that a car-
diacoardiac sympathetic response to coronary occlusion, similar to that described in the spinal cat, 
also occurs in the dog when supraspinal influences are eliminated. These findings suggest that the in-
fluence of the cardiocardiac sympathetic reflex, like 
that of other spinal reflexes (Barman and Wurster, 1978; Dembowsky et al., 1979), is modulated by 
descending inhibitory spinal pathways of barore-
ceptor or brainstem origin when the spinal cord is 
intact.

If activation of cardiac sympathetic afferent fi-
bers does not initiate a spinal cardiocardiac reflex when bulbospinal tracts are intact, how do these 
receptors influence the cardiovascular system dur-
ing ischemia? Previous studies have suggested that the 
primary function of these receptors may be to 
transmit pain sensation to higher centers, which 
may result in centrally mediated enhancement of sympathetic discharge to the heart and other vascu-
lar beds.

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CALCICUM IONS (Ca\(^{2+}\)) play a central role in the control of smooth muscle contractility. Various stimuli initiate contraction of vascular smooth muscle by increasing the concentration of free Ca\(^{2+}\) in the cytoplasm, thereby activating contractile proteins (Somlyo and Somlyo, 1970; Johansson, 1978; Winquist and Bevan, 1980). There are two main sources of activator Ca\(^{2+}\), namely, the pool of extracellular Ca\(^{2+}\) including ions loosely bound to the external muscle membrane and the tightly bound Ca\(^{2+}\) which is sequestered inside the muscle fiber, especially within the cell membrane, sarcoplasmic reticulum, and mitochondria (Hurwitz and Suria, 1980). There are two main sources of activator Ca\(^{2+}\), namely, the pool of extracellular Ca\(^{2+}\) including ions loosely bound to the external muscle membrane and the tightly bound Ca\(^{2+}\) which is sequestered inside the muscle fiber, especially within the cell membrane, sarcoplasmic reticulum, and mitochondria (Hurwitz and Suria, 1980). The investigations were supported in part by U.S. Public Health Service Grant AM00940.

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