Preferential Distribution of Inhibitory Cardiac Receptors with Vagal Afferents to the Inferoposterior Wall of the Left Ventricle Activated during Coronary Occlusion in the Dog

MARC D. THAMES, HAROLD S. KLOPFENSTEIN, FRANCOIS M. ABBOUD, ALLYN L. MARK, AND JOHN L. WALKER

SUMMARY The purpose of this study was to determine the relative magnitudes of the reflex effects mediated by cardiac receptors during anterior as opposed to inferoposterior ischemia of the left ventricle of the dog. Cessation of perfusion (coronary "occlusion") of the circumflex coronary artery (Cx) in 29 chloralose-anesthetized dogs with common carotids ligated (group I) resulted in significant bradycardia and hypotension, but in no significant change in perfusion pressure in the gracilis muscle perfused at constant flow. Occlusion of the left anterior descending coronary artery (LAD) produced less hypotension, no change in heart rate, and vasoconstriction in the gracilis. After vagotomy and aortic nerve section, no significant change in heart rate or gracilis perfusion pressure was observed during LAD or Cx occlusion, and the blood pressure responses to LAD and Cx occlusion were not different. In nine dogs with sinoaortic denervation (group II), brief Cx occlusion resulted in bradycardia, hypotension, and vasodilation in the gracilis muscle. LAD occlusion in group II dogs caused less hypotension and no change in heart rate or gracilis perfusion pressure. After vagotomy, the bradycardia and vasodilation resulting from Cx occlusion were abolished and the blood pressure responses to LAD and Cx occlusion were not different. The weights of left ventricle perfused by each occluded vessel were not different. These data show that left ventricular receptors with vagal afferents which are activated during coronary occlusion and which mediate cardioinhibitory and vasodepressor responses are located mainly in the inferoposterior left ventricle of the dog heart.

PATIENTS with infarction of the inferior wall of the left ventricle have been noted to have more frequent bradycardia and hypotension than do those with anterior wall infarction. Similarly, injection of contrast medium into the coronary arteries supplying the inferior wall of the heart results in bradycardia and hypotension more frequently than does injection into the artery supplying the anterior wall. These effects seen during inferior infarct and coronary arteriography are thought to be reflex in origin and to result principally from activation of cardiac receptors. Further, these observations suggest that cardiac receptors mediating vasodepressor effects are preferentially distributed to the inferoposterior wall of the heart in man.

Myocardial necrosis in dogs and coronary occlusion in cats are known to activate cardiac receptors and to induce reflex responses of bradycardia, hypotension, and vasodilation or vasoconstriction. Vagotomy reduces or abolishes these reflex effects. Costantin has suggested that ventricular receptors are responsible for these effects and are activated by the bulging of the ischemic area; the recent study of Thoren supports this view.

Studies in our laboratories have recently shown that injection of veratridine into the circumflex branch of the left coronary artery in the dog results in significantly greater bradycardia, hypotension,
and vasodilation in the perfused gracilis than when the same dose is injected into the left anterior descending branch. Thus, in the dog, receptors which are chemically activated seem to be preferentially distributed to the inferoposterior wall of the left ventricle. Veratridine is a veratrum alkaloid, a class of chemical compounds which nonspecifically activates all cardiac sensory endings. It is not known whether occlusion of the circumflex coronary artery (Cx) and, thus, whether inferoposterior wall ischemia would give rise to greater reflex effects than is mediated by occlusion of left anterior descending coronary with resulting anterior wall ischemia. The purpose of the present study was to investigate the relative magnitudes of the reflex effects mediated by cardiac receptors during anterior as opposed to inferoposterior ischemia of the left ventricle in the dog. The data indicate that ventricular receptors with vagal afferents that are activated by myocardial ischemia and that mediate reflex cardioinhibitory and vasodepressor responses are preferentially distributed to the inferoposterior left ventricle.

**Methods**

Twenty-nine dogs weighing 18-32 kg were fasted for 24 hours and were anesthetized with α-chloralose (75 mg/kg, iv) and urethan (750 mg/kg, iv). An additional nine dogs weighing 16-30 kg were anesthetized with sodium thiopental (20 mg/kg, iv) and α-chloralose (80 mg/kg initially and 10 mg/kg hourly, iv). After intubation, the dogs were mechanically ventilated with air supplemented with oxygen (2-3 liters/min). Ventilatory rate ranged from 9-12 cycles/min and tidal volume was initially determined from a nomogram based on body weight. Blood Po2, PcO2, and pH were measured periodically. If necessary, sodium bicarbonate (2–5 ml of a 7% solution) was given to maintain pH between 7.3 and 7.4, and the ventilatory rate was adjusted to keep PcO2 between 30 and 40 mm Hg. Arterial Po2 was greater than 100 mm Hg in all studies.

During the surgical preparation of the dogs and during the protocol just before vagotomy, muscle movement was prevented with decamethonium bromide (0.3 mg/kg, iv). Body temperature was maintained between 37°C and 39°C by external warming.

**Preparation of the Animal**

**Group I (Carotids Ligated)**

In these 29 dogs a midline cervical incision was used to expose both common carotid arteries for ligation and both vagi for subsequent bilateral vagotomy. In 20 dogs the carotids were ligated in the course of completing the preparation. In 9 dogs the carotids were occluded just before coronary occlusion. The carotids were occluded to minimize reflex effects mediated by carotid baroreceptors which could mask differences in the responses to LAD as opposed to Cx occlusion.

Through a left thoracotomy, the pericardium was opened and short segments of the proximal anterior descending coronary artery (LAD) and the Cx were exposed for cannulation. Care was taken to avoid damaging the nerves that course near these arteries. Two loose ligatures were placed around each segment. After the remainder of the preparation was completed and after administration of sodium heparin (500 U/kg, iv), the two vessels were cannulated and perfused separately at constant flow via two roller pumps with blood obtained from the left femoral artery. The LAD and Cx were cannulated 5-7 mm and 10-15 mm, respectively, beyond the bifurcation of the left main coronary artery. Coronary flows were adjusted to maintain coronary perfusion pressure equal to control mean arterial pressure. Coronary perfusion was interrupted by turning off one of the pumps, which reduced coronary perfusion pressure to 20 mm Hg or less. In the interest of brevity this will, in most instances, be referred to in the text as coronary “occlusion.”

The isolated, innervated, constant flow-perfused gracilis muscle was used to assess changes in skeletal muscle vascular resistance which occurred in skeletal muscle during LAD or Cx occlusion. The gracilis muscle was surgically isolated, all vessels except the major artery and vein were ligated, the fascia was stripped from the muscle, and one tendon was cut. Care was taken to avoid damaging the gracilis nerve. The muscle was covered with mineral oil to prevent dehydration and was kept at 36-39°C by external warming. The artery to the muscle was cannulated, and the muscle was perfused with a small roller pump with heparinized blood obtained from the left femoral artery. Flow was adjusted to give perfusion pressures equal to or slightly in excess of mean arterial pressure. Adequacy of vascular isolation was established by a drop in perfusion pressure to less than 10 mm Hg when the pump was turned off.

**Group II (Sinoaortic Denervation)**

The preparation of the nine dogs included in this group was similar to that of dogs in group I with several important exceptions. First, in all nine dogs, the aortic nerve on each side was dissected free from the vagosympathetic trunk distal to the nodose ganglion and identified by recording of afferent traffic. The aortic nerve was traced to its junction with the vagus or sympathetic nerve and cut. This procedure has been shown to acutely abolish the baro- and chemoreflexes from the aortic arch and the baroreflex from the major intrathoracic arteries. The cervical sympathetic nerves were cut distal to the cranial cervical ganglion. Second, in four of these dogs, the carotid bifurcations were exposed so as to permit carotid denervation during the course of the protocol while, in the other five
dogs, the carotid sinuses and the immediately adjacent vessels were stripped of all visible innervation before the protocol was begun. The carotid regions were considered denervated when bilateral carotid occlusion after carotid denervation resulted in no change in either heart rate or blood pressure. Finally, in four dogs in this group, the LAD and Cx were not cannulated. Coronary occlusion was performed in these dogs, using snare placed around the proximal LAD and Cx.

Arterial pressure was measured via a cannula in the right femoral artery connected to a Statham P23Db transducer. Mean arterial pressure was obtained by electrical averaging. Heart rate was measured with a cardiotachometer preamplifier which was triggered by the arterial pressure wave. Gracilis perfusion pressure was measured via a cannula connected to the perfusion system just upstream from the gracilis artery with a Statham P23Db transducer. In those experiments in which the coronaries were perfused, the coronary perfusion pressure was measured in the same fashion as the gracilis perfusion pressure. In four experiments, mean left atrial pressure was recorded during LAD and Cx occlusion. All signals were amplified and displayed on a Beckmann Dynograph (model R411).

The experimental protocol was started 1 hour after completing the preparation.

Protocol

Group I

After the control steady state was established, perfusion of the LAD or Cx was interrupted for 120 seconds and the parameters indicated above were monitored. Following reperfusion of the vessel and the return of all measured variables to control, the perfusion of the other coronary artery was interrupted. The order in which the perfusion of the coronary arteries was interrupted was randomized. After each vessel was occluded, the vagi and aortic depressor nerves were sectioned, and after 30 minutes the protocol was repeated.

Group II

All nine dogs in this group were subjected to bilateral section of the aortic depressor nerves. Five of these nine dogs had carotid sinus denervation at the time the aortic nerves were sectioned and were studied by the same protocol used for group I dogs. In the remaining four experiments, responses to coronary occlusions were recorded first with carotid innervation intact and then with the carotid sinuses denervated. Duration of coronary occlusion in this group was limited to 60 seconds.

The amount of left ventricle perfused by each of the occluded vessels was estimated at the conclusion of each group II experiment. The heart was removed and each vessel was injected with a dye of different color (rose or Evans blue) at the site of occlusion. The myocardium stained by each of the two dyes could be distinguished easily. The segments of myocardium stained by each dye were removed and weighed separately.

Data Analysis

Peak heart rate, mean arterial pressure, and gracilis perfusion pressure responses to LAD and Cx occlusion were measured. The observations were summed to obtain the mean and standard error (SE) for each group. The statistical significance of the difference in means was in general evaluated by Student's t-test for paired observations. Comparisons between groups I and II were also made using Student's t-test for unpaired observations. The level of significance was taken as 0.05.

Results

Group I: Effects of Coronary Occlusion after Carotid Ligation

The data from 20 dogs whose common carotid arteries were ligated before starting the protocol and the data from nine dogs whose carotids were occluded just before coronary occlusion have been combined because neither the control values nor responses were different. As summarized in Figure 1, interruption of Cx perfusion resulted in significant decreases in mean arterial pressure (116 ± 6.6 to 89 ± 6.0 mm Hg) and heart rate (148 ± 4.8 to 138 ± 4.5 beats/min) and in an insignificant increase in gracilis perfusion pressure. LAD occlusion caused significant decreases in blood pressure (115 ± 6.3 to 97 ± 5.7 mm Hg) and increases in gracilis perfusion pressure (155 ± 7.9 to 170 ± 8.1 mm Hg), whereas heart rate did not change (147 ± 4.4 to 145 ± 4.3 beats/min). The fall in heart rate and in mean arterial pressure were significantly greater with Cx than with LAD occlusion.

After the vagi and aortic nerves were sectioned,
no significant change in heart rate or gracilis perfusion pressure was elicited by interruption of perfusion of either coronary (Fig. 1). Although occlusion of each coronary artery resulted in a significant decrease in mean arterial pressure after vagotomy, the decrease with circumflex occlusion was significantly less after vagotomy than that observed before vagotomy, in spite of the absence of the buffering influence of the aortic nerves which were sectioned with the vagi. In addition, the difference in blood pressure response during Cx as opposed to LAD occlusion was abolished by vagotomy. Control heart rate was significantly higher after vagotomy, but control mean arterial pressure and gracilis perfusion pressure 30 minutes after vagotomy and section of the aortic nerves were not different from prevagotomy controls.

Although not evident from Figure 1, a decrease in gracilis perfusion pressure was observed in eight of 29 dogs during Cx occlusion in spite of systemic hypotension and intact aortic baroreceptors. In contrast, a decrease in gracilis perfusion pressure was observed in only two of 29 dogs with LAD occlusion, even though the fall in arterial pressure was less than that with Cx occlusion. Figure 2 shows the original records from an experiment in which coronary occlusion caused vasodilation in the gracilis, particularly during Cx occlusion.

**Group II: Effects of Coronary Occlusion after Sinoaortic Denervation**

As summarized in Figure 3, cessation of perfusion or occlusion of the Cx coronary artery in dogs with sinoaortic denervation resulted in significant decreases in mean arterial pressure (109 ± 8.4 to 64.9 ± 5.6 mm Hg), in heart rate (143 ± 11.6 to 132 ± 12.2 beats/min), and in gracilis perfusion pressure (161 ± 8.4 to 137 ± 9.8 mm Hg). Cessation of perfusion or occlusion of the LAD in these dogs did not significantly change heart rate (147 ± 11.5 to 144 ± 10.0 beats/min) or gracilis perfusion pressure (162 ± 10.2 to 153 ± 11.8 mm Hg), although these tended to decrease. Even though LAD occlusion caused a significant fall in mean arterial pressure (104 ± 8.4 to 87.2 ± 10.4 mm Hg), this decrease was significantly less than was observed with Cx occlusion. Vagotomy abolished the bradycardia and the vasodilation of the gracilis muscle observed during
CX occlusion. Hypotension occurred with both LAD and CX occlusion after vagotomy, but the differential response noted above was abolished.

The principle difference between groups I and II relates to the changes in gracilis perfusion pressure. In this regard the major difference between the preparations was that, in group I, the carotids were ligated to minimize the influence of the carotid baroreceptors but the aortic depressor nerves were left undisturbed, whereas the group II dogs were sinoaortic denervated. To establish more clearly whether the presence of functioning baroreceptors accounted for the differences in gracilis responses between groups, four experiments were conducted in group II dogs in which the LAD and CX each were occluded after aortic nerve section, after sinoaortic denervation, and after sinoaortic denervation and vagotomy. In contrast to the other experiments in which the LAD and CX were pump-perfused and flow was interrupted by turning off the pump, in these experiments flow was interrupted with occluders placed around the proximal LAD and CX. The results are shown in Figure 4. After sectioning, only the aortic nerves CX occlusion caused vasoconstriction in three of four experiments and caused no change in the fourth; LAD occlusion caused vasoconstriction in two, vasodilation in one, and no change in the fourth experiment. After sinoaortic denervation, vasodilation occurred during CX occlusion in all four dogs and in three of four dogs during LAD occlusion. Finally, after vagotomy, the responses to LAD and CX occlusion were small and inconsistent.

In four dogs, the mean left atrial pressure was measured during LAD and CX occlusion. In each experiment the vasodepressor responses to CX occlusion exceeded those which resulted from LAD occlusion. In only one of four of these experiments did the rise in mean left atrial pressure during CX occlusion (2.5, 2.7, 1.7, and 2.5 mm Hg) exceed the increase associated with LAD occlusion (1.8, 3.7, 2, and 2.5 mm Hg, respectively).

Comparisons between Groups (Unpaired t-Test)

CX occlusion in group II dogs prior to vagotomy caused a greater fall in blood pressure than was seen in group I dogs. The gracilis perfusion pressure responses were significantly different for both LAD and CX occlusion. During LAD occlusion, a statistically significant vasoconstriction in group I dogs was observed, in contrast to the tendency toward vasodilation in group II dogs. During CX occlusion a tendency toward constriction in group I dogs was observed in contrast to a significant vasodilation in group II dogs. There was no difference between groups in heart rate responses prevagotomy and in all responses postvagotomy.

Quantitation of Ventricle Perfused by Each Occluded Vessel

In nine experiments, we estimated the weight of the myocardium perfused by each vessel. The CX perfused 58 ± 5.1 g of myocardium (left ventricle, right ventricle, and septum) and the LAD perfused 61.2 ± 5.9 g of myocardium. After the right ventricular myocardium was removed, these values were 51.0 ± 4.5 and 53.1 ± 5.6 g, respectively. Finally, the interventricular septum was removed so that the left ventricular free wall perfused by each occluded vessel was determined. The values for CX and LAD were 35.6 ± 3.1 and 28.8 ± 3.7 g, respectively. None of these differences was significant.

Discussion

The present experiments provide the first systematic evidence that cardiac receptors with vagal afferents which are activated during coronary occlusion...
clusion and which mediate reflex vasodepressor responses are preferentially distributed to the inferoposterior wall of the left ventricle in the dog. This is most clearly demonstrated in the dogs with sinoaortic denervation (Fig. 3). Ceasation of perfusion of the Cx coronary artery induced greater hypotension, bradycardia, and vasodilation in skeletal muscle than did LAD occlusion. Similar differences for heart rate and mean arterial pressure were noted in group I dogs (Fig. 1). Eight of 29 group I dogs had reflex vasodilation during Cx occlusion in spite of hypotension and the presence of functioning aortic baroreceptors. Only two of these dogs had vasodilation with LAD occlusion. The absence of vasoconstriction in these dogs during Cx occlusion (mean data) in spite of greater hypotension than was seen with LAD occlusion can be viewed as further evidence of the preferential distribution of inhibitory cardiac receptors to the inferoposterior wall of the heart.

It could be argued that the differences in the responses to LAD and Cx occlusion were due to the perfusion of more myocardium, particularly left ventricle, by the Cx than by the LAD, rather than to a preferential distribution of receptors in the distribution of the Cx. Two groups of data indicate that this is not the case. First, the weights of the myocardium perfused by the LAD and Cx were not different. This was true whether one compared only the left ventricular free wall perfused by each vessel or if the septum and/or right ventricle perfused by each vessel was included. Second, after vagotomy, the hypotension which resulted from LAD occlusion was not different from the hypotension resulting from Cx occlusion in either group. This observation could be viewed as the functional correlate of the weight measurements.

It is unlikely that differences in atrial receptor activation could account for the differential responses. The rise in mean left atrial pressure was similar during LAD and Cx occlusion. Thus, these data provide additional support for the view that ventricular receptors mediate these reflex effects in response to Cx occlusion.

We considered the possibility that the differences between the responses to LAD as opposed to Cx occlusion were due to the interruption of afferents which course near the LAD during cannulation of this vessel. Frink and James have mapped the intracardiac route of the Bezold-Jarisch reflex and concluded that the afferent nerves converge along the distribution of the left main coronary artery. Since the site of LAD cannulation was downstream from that region, it is unlikely that interruption of afferents accounted for the differences. Further, Walker and colleagues have carried out experiments which demonstrate that the technique used for cannulation of the LAD does not interrupt cardiac vagal afferents. They injected nicotine into the proximal LAD via a very small catheter which had been passed retrogradely from a small peripheral branch in six dogs. Then, the proximal LAD was exposed at the location of the catheter tip, two loose ties were placed around the vessel, and nicotine was injected again. The mean pre- and postdissection decreases in arterial pressure, heart rate, and perfusion pressure in the constant flow-perfused gracilis were not different. In two experiments in which the LAD was actually cannulated and perfused, the bradycardia and hypotension (gracilis perfusion pressure was not measured in these experiments) which resulted from intracoronary nicotine before and after cannulation were nearly identical. Thus the differences in the responses to LAD as opposed to Cx occlusion cannot be attributed to interruption of inhibitory cardiac vagal afferents which course near the LAD.

Another possible explanation for the differential responses we observed is that LAD occlusion might increase the discharge of inhibitory cardiac receptors but simultaneously augment the discharge of receptors mediating excitatory effects, thus reducing the cardioinhibitory and vasodepressor responses to LAD occlusion. We have considered this possibility because cardiac receptors with sympathetic afferents have been shown to mediate cardioaccelerator and vasopressor effects. However, our postvagotomy data show no evidence for such excitatory effects, i.e., no vasoconstriction in the gracilis or increase in arterial pressure. To our knowledge there are only two excitatory reflexes mediated through vagal afferents. The first is the modest cardioaccelerator response to volume expansion (Bainbridge reflex) which is not accompanied by significant vasopressor or vasoconstrictor responses. The second is the hypertensive, tachycardic response that results from injection of serotonin into the blood supply to the aortic bodies which, in some animals, originates from the left main coronary artery. It is unlikely that these receptors were activated in our studies, since the cannulas used to perfuse the LAD and Cx were at least 5 and 10 mm, respectively, beyond the bifurcation of the left main coronary artery. Finally, in the cat, activation of myelinated cardiac vagal afferents at high frequencies and in the absence of the arterial baroreceptors has been shown to have only weak cardioacceleratory and vasopressor effects, whereas activation of both myelinated and nonmyelinated cardiac vagal afferents resulted in profound bradycardia and hypotension in spite of functioning arterial baroreceptors. Although we have no way of completely excluding activation of excitatory receptors which might account for the observed differences, in view of our postvagotomy data and the other evidence cited above, it is unlikely that the differential responses can be explained on the basis of activation of excitatory receptors during LAD occlusion.

The absence of vasoconstriction after vagotomy and aortic nerve section in group I dogs (Fig. 1) deserves comment. First, these data indicate that
common carotid occlusion minimized the influence of the carotid baroreceptors in the present experiments. Second, since cardiac vagal afferents were shown in this study to induce reflex vasodepressor responses, the vasoconstriction in the gracilis during prevagotomy LAD occlusion was mediated by the aortic baroreceptors. The differences in responses to coronary occlusion between groups is attributable to the presence of functioning aortic baroreceptors only in group I.

Thörén elicited bradycardia, hypotension, and muscle vasodilation both with LAD and with right coronary occlusion in the cat with carotids clamped. Responses to occlusion of these vessels were similar in magnitude. The responses to Cx occlusion in the cat were not examined. The lack of consistent cardioinhibitory and vasodepressor responses in our experiments during LAD occlusion suggests that the distribution of inhibitory cardiac receptors in the dog is different from that in the cat. Our results are in agreement with those of Thorén in the cat which indicate that the buffering influences of the baroreceptors must be largely eliminated before a clear-cut reflex vasodilation in skeletal muscle is observed in response to coronary occlusion.13

The general characteristics and locations of left ventricular receptors with nonmyelinated vagal afferents have been studied in the dog. It has been suggested that these receptors mediate the vasodepressor response to coronary occlusion. Sleight and Widdicombe indicated that most of the left ventricular receptors they studied were in the anterolateral wall; the data of Muers and Sleight indicates no particular tendency toward anterolateral distribution. It is likely that the method of selecting receptors to be studied may have favored the detection of receptors in the anterolateral wall because of their accessibility. There has not been a systematic electrophysiological study of the distribution of left ventricular receptors which increases their discharge frequencies during coronary occlusion.

Our findings in group I dogs (Fig. 1) are basically in agreement with those of several investigators,1 9, 11, 12 including Costantin, who found that Cx occlusion caused vasodilation in the constant flow-perfused hindlimb in 13 of 23 dogs with sinoaortic baroreceptors undisturbed. Similarly, Toubes and Brody showed that hindlimb vascular resistance did not change significantly during sustained severe hypotension after coronary embolization in dogs with sinoaortic baroreceptors intact. Bilateral cervical vagotomy allowed normal reflex vasoconstriction to occur during hypotension. One important difference between our study and those of Costantin and of Toubes and Brody was our use of the isolated gracilis muscle rather than the hindlimb of the dog. Hanley et al did not observe a single episode of vasodilation in the gracilis muscle during circumflex occlusion in the dog, although hindlimb vasodilation was noted. The reason for the difference between their results and ours may relate in part to the fact that, in their dogs, the baroreceptors were not decentralized.

Although the durations of myocardial ischemia used in the present study admittedly were short, the responses appear to be similar to those observed following inferior myocardial infarction or during coronary arteriography in man. It also seems that, in both man and dog, anterior myocardial ischemia evokes these cardioinhibitory and vasodepressor responses less frequently.

Finally, even though vasodilation in skeletal muscle was not consistently observed during Cx occlusion in dogs with aortic baroreceptors operative (Fig. 1), the absence of frank vasoconstriction should be considered an inappropriate response to the accompanying hypotension, since a lesser decrease in arterial pressure during LAD occlusion induced significant baroreceptor-mediated vasoconstriction. Similarly, in man, increased input from cardiac receptors during inferior infarction may interfere with the normal role of the arterial baroreceptors in controlling arterial pressure. This mechanism may be even more important in man than in the dog, since evidence from studies on humans indicates that cardiopulmonary receptors preferentially influence skeletal muscle resistance with lesser effects on the kidney and on the splanchnic circulation, while the reverse is true for the dog. The latter observation may be the basis for the difficulty which is encountered in demonstrating frank vasodilator responses in skeletal muscle during coronary occlusion in the dog when arterial baroreceptors are operative. Furthermore, the inhibitory influence of cardiopulmonary receptors on other vascular beds (particularly renal and splanchnic) are known to be substantially greater (than their influence on skeletal muscle) in the dog so that evidence of sympathetic withdrawal during coronary occlusion may be more evident in other beds, even in the presence of functioning arterial baroreceptors. This is supported by previously observed vasodilation in the hindlimb during myocardial ischemia in the dog7, 8, 12 and by experiments recently completed in our laboratory.30

Acknowledgments

We wish to thank Ralph Richter for his skillful technical assistance and Karen M. Kinney for typing the manuscript.

References


RECEPTORS ACTIVATED DURING CORONARY OCCLUSION/Thames et al. 519

23. Coleridge HM, Coleridge JCG, Kidd C: Cardiac receptors in the dog, with particular reference to two types of afferent endings in the ventricular wall. J Physiol (Lond) 184: 323-339, 1964
Preferential distribution of inhibitory cardiac receptors with vagal afferents to the inferoposterior wall of the left ventricle activated during coronary occlusion in the dog.

M D Thames, H S Klopfenstein, F M Abboud, A L Mark and J L Walker

Circ Res. 1978;43:512-519
doi: 10.1161/01.RES.43.4.512

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/43/4/512

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

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

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