Different Reflex Responses in Cardiac and Renal Sympathetic Nerve Activities during Coronary Occlusion in the Dog

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Abstract This study was undertaken to scrutinize the difference in the response of cardiac sympathetic nerve activity (CSNA) and renal nerve activity (RNA) to coronary artery occlusion. The magnitudes and time courses of per cent changes in CSNA and RNA, recorded simultaneously in 22 dogs, were compared with each other during a 1 min occlusion of the left circumflex coronary artery under four different conditions. With afferent nerve intact, changes in CSNA and RNA showed similar biphasic patterns comprising initial increases (CSNA, 9±3.1% (S.E.); RNA, 16±6.4%, at 20 sec of occlusion) and subsequent decreases (CSNA, -3±4.3%; RNA, -11±6.5%, at 60 sec), despite the progressive fall in arterial pressure (from 109±4 to 89±4 mmHg). After carotid sinus denervation, the initial increases in both nerve activities were reduced and the subsequent decreases became more evident. The decreases in RNA (-47±8.0%) were significantly greater than those in CSNA (-23±5.6%). After bilateral cervical vagotomy, changes in both CSNA and RNA showed in contrast similar monophasic increasing patterns (CSNA, 18±4.0%; RNA, 25±5.6%, at 60 sec), where RNA increased more than did CSNA. After carotid sinus and vagoaortic denervation, CSNA and RNA increased only slightly throughout the occlusion. These results conclusively indicate that the reflex responses in CSNA and RNA during coronary occlusion are quantitatively different, though qualitatively similar, and that RNA is inhibited significantly more than CSNA by the reflex mediated through the afferent vagal nerves.

Key Words: coronary occlusion, cardiac sympathetic nerve activity, renal nerve activity, afferent vagal nerves, carotid sinus nerves.

For a better understanding of the neural regulation of the cardiovascular

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control operating during myocardial ischemia, it is extremely important to know quantitatively the magnitudes and time courses of the different sympathetic reflex responses influencing both contractility and afterload of the ischemic heart. Previous studies have shown that the activities of efferent sympathetic nerves to the heart (Costantin, 1963; Felder and Thames, 1979) and the kidney (Kezdi et al., 1974; Uchida and Sakamoto, 1974; Thames and Abboud, 1979), recorded only separately, were reflexly inhibited during experimental myocardial ischemia. In these studies, the major interest seems to be in afferent inputs or pathways producing this inhibition, and to date, no detailed analysis of time courses of sympathetic reflex responses occurring during a brief coronary occlusion has been made.

We would assume that the reflex inhibition of cardiac sympathetic nerve activity contributes to the prevention of the ischemic myocardium from progressively deteriorating, and that the reflex inhibition of the activities of sympathetic nerves to the vascular system partly contributes to the protection of the heart by decreasing cardiac afterload through vasodilatation. Our present questions are which of these inhibitory reflex responses is more dominant, and whether or not the time courses of these sympathetic reflex responses are different. A conclusive answer can be obtained only if the activities of sympathetic nerves to the heart and the major resistance vessels are recorded in the same animal.

To this end, we simultaneously measured cardiac and renal sympathetic nerve activities during a short occlusion of the left circumflex coronary artery. Renal nerve activity was measured as representative of the activities of sympathetic nerve in the resistance of the vascular system, because renal blood flow occupies as high as 20% of the resting cardiac output, and hence, small changes in renal vascular resistance can result in major alterations in the distribution of cardiac output. The magnitudes and time courses of the changes in these sympathetic nerve activities were compared with each other during coronary artery occlusion.

METHODS

Experimental preparations. Twenty-two mongrel dogs weighing 8–12 kg were initially anesthetized with sodium pentobarbital (30 mg/kg, i.v.), and additional doses (5 mg/kg) were injected intravenously at intervals as required (every 3 hr on average). The dogs were intubated and artificially ventilated with room air mixed with oxygen at a tidal volume of 15 ml/kg. During the experiments arterial blood was occasionally sampled for measurements of pH and blood gases, and pH, \( P_{O_2} \), and \( P_{CO_2} \) were kept within normal ranges by changing respiration rate or intravenous administration of sodium bicarbonate. Muscle movements were prevented with pancuronium bromide (4 mg, i.v.) throughout the experiment.
The neck was opened by midline incision. In a first group of 10 dogs, bilateral vagosympathetic trunks were dissected and an umbilical tape was placed around each trunk for later sectioning of the trunks. In a second group of 7 dogs, bilateral carotid bifurcations were exposed and the carotid sinuses were denervated by sectioning all visible attaching nerves and vessels except for the internal and external carotid arteries under a dissecting microscope (Olympus OME). In a third group of 5 dogs, both carotid sinuses were denervated in the same way and then both vagosympathetic trunks, which include the aortic depressor nerves, were sectioned immediately after the isolation of the trunks at the middle of the neck.

In the right lateral position, left thoracotomy was performed in the fifth intercostal space in the three groups of dogs. The pericardium was incised parallel to the left phrenic nerve, and the edges of the pericardium were suspended to the chest wall to make a pericardial cradle. With the aid of the dissecting microscope, a longitudinal incision was made in the epicardial tissue overlying the proximal left circumflex coronary artery (LCX), which was dissected free 1–2 cm from its origin taking special care not to damage the pericoronary nerve. A thread was passed around the LCX to be used later as an occlusive snare.

The parietal pleura was incised near the left middle cervical ganglion, and the ventrolateral cardiac nerve was isolated by freeing it from the surrounding connective tissue under the dissecting microscope. Postganglionic cardiac sympathetic nerve activity (CSNA) (RANDALL and ARMOUR, 1977) was recorded from this cardiac nerve. Through the extraperitoneal approach the left renal nerve along the left renal vessels was identified and isolated for recording renal nerve activity (RNA). The above nerves were covered with liquid paraffin immediately after preparation.

**Experimental measurements.** Efferent sympathetic nerve activities of the ventrolateral cardiac nerve and the left renal nerve were recorded from their cut central ends, which were placed on bipolar Ag-AgCl electrodes. The method of analyzing the electroneurograms used in this study was simple integration. A full description and evaluation of this method have been reported previously (NINOMIYA and IRISAWA, 1967). Briefly, the signals were amplified by a biophysical preamplifier (Nihon Kohden, AVB-8, input impedance; 1 MΩ) with a high cutoff frequency of 3,000 Hz and a low cutoff frequency of 15 Hz. The amplified signals were monitored with an oscilloscope (Hitachi, V152B). Each of these signals was then rectified with a full-wave rectifier circuit and then integrated continuously with an RC integrator having a time constant of 1 sec to obtain mean sympathetic nerve activities (mean CSNA and mean RNA). Changes in CSNA and RNA were expressed as per cent changes from the pre-occlusion control values to normalize the marked differences in absolute magnitudes of nerve activities (in μV) among different preparations.

Arterial blood pressure (AP) was measured in the left subclavian artery using
a polyethylene tube inserted through the left mammalian artery. The distal end of the tube was connected to a Statham P23 ID transducer.

**Experimental protocol and data analysis.** Before any intervention was performed, the preparation was allowed to stabilize for about 30 min, as assured by relatively constant mean levels of CSNA, RNA, and AP. Control recordings were obtained for 60 sec and the LCX was then occluded for 60 sec by tightening the previously placed snare. We preferred LCX occlusion to anterior descending coronary arterial (LAD) occlusion, because inhibitory effects on RNA (Thames and Abboud, 1979) and CSNA (Felder and Thames, 1979) were greater during LCX occlusion than during LAD occlusion. Occlusion of the left main coronary artery was not chosen, because of the high incidence of ventricular fibrillation during occlusion (Allen and Laadt, 1950). In the first group of 10 dogs, occlusion of the LCX was repeated approximately 30 min after sectioning of the bilateral vagosympathetic trunks. In the second group of 7 dogs, LCX occlusion was performed only after carotid sinus denervation. In the third group of 5 dogs, the LCX was occluded only after carotid sinus denervation and sectioning of the bilateral vagosympathetic trunks. Thus, the LCX was occluded under four different conditions, i.e., with intact afferent nerves, after carotid sinus denervation, after cervical vagotomy, and after carotid sinus and vagoaortic denervation.

Simultaneous measurements of CSNA, RNA, and AP were made before, during and after each LCX occlusion. As reported previously (Ninomiya et al., 1971), CSNA and RNA changed periodically with cardiac and respiratory cycles in this study also. Therefore, control values of mean CSNA and mean RNA were determined by planimetry of their tracings over 60 sec immediately preceding LCX occlusion. In the same way, the time-averaged values for mean CSNA and mean RNA during and after occlusion were determined for each consecutive 10-sec interval beginning immediately with the on and off of the occlusion. For example, a CSNA value at 10 sec of occlusion indicate the mean CSNA level between 0 and 10 sec of occlusion. Changes in CSNA and RNA were evaluated as per cent changes of the 10 sec averaged values of both mean nerve activities from the 60 sec averaged control level. Changes in mean AP (MAP) from the control value that was obtained during the 60-sec interval preceding occlusion, were also determined for each consecutive 10-sec period and were evaluated in each of 22 dogs.

The data of CSNA and RNA obtained simultaneously in 22 dogs were pooled and subjected to the following analyses. The responses of CSNA and RNA as well as MAP during the LCX occlusion were evaluated by the analysis of variance (ANOVA) for randomized block design, and the time trend was tested statistically (Snedecor and Cochran, 1971; Wallenstein et al., 1980). The difference between responses in CSNA and RNA at identical times was evaluated by the Student's t-test for paired observation. The level of significance was taken as 0.05 in all the statistics. All data presented in this paper indicate mean ± S.E.
RESULTS

With intact afferent nerves

Figure 1 shows a representative example of responses of CSNA, RNA, and AP to LCX occlusion in a dog with intact afferent nerves. CSNA increased slightly from the onset of occlusion up to 40 sec and thereafter decreased to the control level, while RNA apparently increased from the onset of occlusion up to 20 sec and thereafter decreased below the control level during and after the occlusion, despite the progressive fall in AP. The magnitudes of the changes in RNA were clearly greater than those in CSNA.

Figure 2 shows the responses of CSNA, RNA, and MAP resulting from the 1-min LCX occlusion in the first group of 10 dogs with intact afferent nerves. The control value of MAP was 109 ± 4 mmHg. From 0 to 20 sec of occlusion, MAP fell slightly while CSNA and RNA increased significantly. Thereafter, both CSNA and RNA decreased to the control level during the next 10-sec period of occlusion, i.e., between 20 to 30 sec after occlusion. During the latter half of occlusion, RNA and MAP did decrease below the control level and CSNA tended to decrease below the control level. At the end of the 1-min period of occlusion, CSNA, RNA, and MAP decreased by 3 ± 4.3%, 11 ± 6.5%, and 20 ± 3.9 mmHg, respectively. After the release of occlusion, CSNA, RNA, and MAP remained below the control level for about 1 min, and all these variables gradually recovered to the control level by about 2 min after the release. These responses of CSNA, RNA, and MAP with time during LCX occlusion were all statistically significant by the ANOVA (null hypothesis: no response from the control level). Tests for time trend revealed that the initial increases in CSNA and RNA and the subsequent decreases in CSNA and RNA from 20 to 60 sec of occlusion were all statistically significant.

As for the difference of the CSNA and RNA responses, RNA increased
significantly more than CSNA at 10 sec of occlusion and decreased more significantly at 60 sec of occlusion. The difference between the two RNA values (−27 ±5.2%) at 20 and 60 sec of occlusion was significantly greater than that between the two CSNA values (−12 ±2.8%).

These results indicated that 1) a characteristic time trend of the changes in either CSNA or RNA had a biphasic pattern, i.e., initial increases and subsequent decreases, despite AP falling progressively, and 2) the magnitudes of the biphasic changes in RNA were greater than those in CSNA.

**After carotid sinus denervation**

Figure 3 shows the typical responses of CSNA, RNA, and AP resulting from LCX occlusion in a dog after carotid sinus denervation. As shown in Fig. 3, from the onset of occlusion to 30 sec, CSNA and RNA increased slightly or remained near the control level. Thereafter, both nerve activities abruptly and profoundly decreased below the control level. The decreases continued during the latter half of occlusion and after the release of occlusion, where the decreases in RNA were clearly larger than those in CSNA.

A similar analysis to that performed in Fig. 2 was made in the second group of 7 dogs and the results are summarized in Fig. 4. The control value of MAP
Fig. 3. Typical responses in mean CSNA, mean RNA, and AP resulting from LCX occlusion in a dog after carotid sinus denervation.

Fig. 4. Time courses of ASNA to the heart (open circles) and to the kidney (solid circles) and ΔMAP (open triangles) during and after LCX occlusion in 7 dogs after carotid sinus denervation.

was 132±5 mmHg. From 0 to 20 sec of occlusion, CSNA and RNA increased above the control level, but the increases were small as compared to those in the dog with intact afferent nerves and were statistically insignificant. From 20 to 60 sec of occlusion, all CSNA, RNA, and MAP progressively decreased below the control levels and decreased by 23±5.6%, 47±8.0%, and 33±4.4 mmHg.
respectively, at the end of occlusion. The responses of CSNA, RNA, and MAP with time during LCX occlusion were again statistically significant by the ANOVA. The decreases in CSNA and RNA from 20 to 60 sec of occlusion were statistically significant, RNA decreasing significantly more than CSNA.

These results indicated that 1) both CSNA and RNA showed a significant decrease below the control level and 2) the decreases in RNA were greater than those in CSNA during LCX occlusion after carotid sinus denervation also.

After cervical vagotomy

Figure 5 shows the typical responses of CSNA, RNA, and AP resulting from

![Graph of typical responses of mean CSNA, mean RNA, and AP to LCX occlusion in a dog after cervical vagotomy. The aortic depressor nerves were sectioned with the vagi.]

Fig. 5. Typical responses of mean CSNA, mean RNA, and AP to LCX occlusion in a dog after cervical vagotomy. The aortic depressor nerves were sectioned with the vagi.

![Graph of time courses of ΔSNA to the heart (open circles) and to the kidney (solid circles) and ΔMAP (open triangles) during and after LCX occlusion, obtained in 10 dogs after cervical vagotomy. The aortic depressor nerves were sectioned with the vagi.]

Fig. 6. Time courses of ΔSNA to the heart (open circles) and to the kidney (solid circles) and ΔMAP (open triangles) during and after LCX occlusion, obtained in 10 dogs after cervical vagotomy. The aortic depressor nerves were sectioned with the vagi.
LCX occlusion in a dog after cervical vagotomy. CSNA and RNA clearly increased throughout the occlusion, where the increase in RNA was greater than that in CSNA, the reduction of AP being small.

Figure 6 shows the time courses of CSNA, RNA, and MAP resulting from LCX occlusion in the first group of 10 dogs after cervical vagotomy. As shown in Fig. 6, a small but statistically significant fall in MAP was observed during the entire course of occlusion. The maximum fall in MAP during the occlusion was 6 ± 1.3 mmHg. On the other hand, CSNA and RNA increased throughout the occlusion, the increases in nerve activities occurring being statistically significant. After the release of occlusion, all these variables regained the control level within 1 min. RNA increased more than CSNA during the occlusion, and the difference was statistically significant from 20 to 40 sec of occlusion.

These results indicated that 1) both CSNA and RNA changed in a monophasic increasing pattern, i.e., continuous increases throughout the occlusion, and 2) RNA increased significantly more than CSNA during LCX occlusion after cervical vagotomy.

After carotid sinus and vagoaortic denervation

Figure 7 shows the typical responses of CSNA, RNA, and AP to LCX occlusion in a dog after carotid sinus and vagoaortic denervation. As shown in Fig. 7, CSNA and RNA remained near the control levels throughout the occlusion and after the release of occlusion.

Figure 8 shows the time courses of CSNA, RNA, and MAP resulting from LCX occlusion in the third group of 5 dogs after carotid sinus and vagoaortic denervation. The control value of MAP was 131 ± 10 mmHg. Both CSNA and RNA increased only slightly throughout the occlusion, although the increases shown were not statistically significant. On the other hand, the fall in MAP was

Fig. 7. Typical responses of mean CSNA, mean RNA, and AP to LCX occlusion in a dog after carotid sinus and vagoaortic denervation.

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significant and MAP decreased by $14 \pm 2.8$ mmHg at the end of occlusion. This decrease in MAP was significantly smaller than that observed in the dog after carotid sinus denervation.

These results indicated that 1) the significant decreases in CSNA and RNA observed in carotid sinus denervated dogs were completely abolished, and 2) CSNA and RNA increased only slightly, the increases shown not being different, after carotid sinus and vagoaortic denervation.

DISCUSSION

The new findings in this study are 1) that the responses of the simultaneously recorded cardiac and renal sympathetic nerve activities showed similar time trends and 2) that these responses were markedly different in a quantitative manner during a 1-min occlusion of the circumflex coronary artery in the dog under three of the four different tested conditions, i.e., with intact afferent nerves, after carotid sinus denervation and after cervical vagotomy.

The similar time courses of responses in CSNA and RNA can be interpreted as follows. The well-known normal responses to hypotension are reflex increases in CSNA and RNA mediated by arterial baroreceptors (Kezdi and Geller, 1968; Ninomiya et al., 1971), thereby counteracting hypotension. However, the biphasic pattern that we observed in the dog with intact afferent nerves consists of the initial increases followed by the significant decreases in CSNA and RNA in spite of the progressive fall in arterial pressure. These decreases in CSNA and RNA subsequent to the initial increases cannot be accounted for by the baroreflex, but instead can be regarded as the result of a reflex inhibition by certain
other mechanisms. The change of the CSNA and RNA response patterns by carotid sinus denervation seems to have been caused by the elimination of reflex responses mediated through the carotid sinus nerves. The almost negligible excitatory responses of CSNA and RNA observed after carotid sinus denervation suggest that the excitatory influences other than the carotid sinus reflex were very small. The point where the once-increased CSNA and RNA returned to the control levels in the dogs with intact afferent nerves (Fig. 2) can therefore be regarded as a time of balance at which the excitatory response primarily mediated by the carotid sinus nerves and some inhibitory responses were equal in degree. The almost-equal durations, from the onset of occlusion to the time of balance, of CSNA and RNA seem to indicate that the inhibitory responses started at about the same time (20–30 sec of occlusion). These results and interpretations indicate that the time courses of the inhibitory influence on CSNA and RNA were about the same during coronary occlusion.

The complete disappearance of the inhibitory responses of CSNA and RNA after the cervical vagotomy with (Fig. 6) or without the carotid sinus baroreflex (Fig. 8) indicates that the reflex inhibition of CSNA and RNA was abolished by the vagotomy per se. In the present study the bilateral aortic nerves were also cut along with the vagi on cervical vagotomy. Thus the changes in CSNA and RNA after cervical vagotomy should be regarded as the responses observed with afferent input from neither vagi nor aortic nerves. Because the reflex response, mediated by the aortic nerves alone, to the fall in arterial pressure under coronary occlusion is known to increase both CSNA and RNA, the observed reflex inhibition of CSNA and RNA can be concluded as being mediated through the afferent vagal nerves. This conclusion is consistent with the previous contentions (COSTANTIN, 1963; THOREN, 1973, 1976; THAMES and ABOUD, 1979) that the vagal afferents originating in the heart play a dominant role in the occurrence of inhibitory sympathetic reflex responses during coronary occlusion.

From the three different patterns of the sympathetic responses of either CSNA or RNA (Figs. 2, 4, 6), we can analyze the time course of interaction of the vagally mediated inhibitory response and the arterial baroreceptor-mediated excitatory response. Summation of the reflex responses in CSNA and RNA after carotid sinus denervation (Fig. 4) and the responses after cervical vagotomy (Fig. 6) reasonably simulates the biphasic pattern (Fig. 2) of CSNA and RNA in the dog with intact afferent nerves. This demonstrates the possibility that the initial baroreceptor-mediated increases in CSNA and RNA are subsequently inhibited by vagal afferents from cardiac receptors, and that the relative interaction of the responses induced by these afferents can result in the characteristic biphasic pattern of CSNA and RNA. The above interpretation is possible because we analyzed the magnitudes and the time courses of the CSNA and the RNA responses measured simultaneously. Previous investigations have only showed the relative responses of the two above afferent nerves on either the 90-sec average (as opposed

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to our consecutive 10-sec average) change of CSNA or the maximum response of RNA during a brief coronary occlusion (Felder and Thames, 1979; Thames and Abbooud, 1979), and therefore the results thereof cannot offer such an interpretation as mentioned above.

We demonstrated for the first time the quantitative differences in the magnitudes of the response between CSNA and RNA throughout the coronary occlusion, in the present study. The differences are such that not only the early excitatory responses but also the subsequent decreases were significantly greater in RNA than in CSNA. In spite of the presence of the greater excitatory influence on RNA, RNA decreased significantly more than CSNA during the last 30 sec of occlusion in the dog with intact afferent nerves. The greater inhibitory response of RNA observed in the carotid sinus-denervated dog was completely abolished by additional vagotomy. These results indicate the existence of the greater inhibitory influence on RNA than on CSNA of the vagal afferents from cardiac receptors. After the time of balance of the excitatory and inhibitory responses mentioned above, RNA decreased below the control level because the inhibitory effects exceeded the excitatory ones, while CSNA remained near the control level due to the equal magnitude of the inhibitory and excitatory effects in the dog with intact afferent nerves.

The excitatory responses of CSNA and RNA observed in the four tested conditions are considered to be induced by arterial baroreceptors and probably by sympathetic afferents from cardiac receptors (Malliani et al., 1969; Weaver et al., 1981). The excitatory responses of CSNA and RNA to the circumflex coronary occlusion in a dog after sinoaortic deafferentation can be regarded as those mediated by activation of sympathetic afferents. The almost negligible increases in CSNA and RNA that we observed in the dog after carotid sinus and vagoaortic denervation (Fig. 8) suggest that the relative contribution of the sympathetic afferents to the excitatory responses is small.

The significant attenuation of the depressor response by vagotomy that we observed indicates that the fall in arterial pressure during the circumflex coronary occlusion is partly induced by the reflex mediated by the vagal afferents. Thus, the hypotension during the coronary occlusion has two potential causes: 1) a fall in cardiac output as a result of cardiac function impaired by myocardial ischemia (cardiogenic) and 2) a reflex mediated by afferent vagal nerves (reflexogenic). The fall in arterial pressure in the dog after carotid sinus and vagoaortic denervation (Fig. 8) should be regarded as ischemia-induced cardiogenic hypotension. Therefore, the profound fall in arterial pressure observed in the dog after carotid sinus denervation (Fig. 4) indicates that the significant part of the reduction of arterial pressure is induced by the vagally mediated reflex. This profound fall in arterial pressure in the dog after carotid sinus denervation can result from the possible renal vasodilatation and the decreased myocardial contractility due to the remarkable decrease in CSNA and RNA mediated by the afferent vagal nerves.

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The progressive and profound fall in arterial pressure observed in the dog with intact afferent nerves can be partly accounted for by renal vasodilatation from decreases in RNA after the time of balance during the circumflex occlusion. Failure of renal vasoconstriction (Maximov and Brody, 1976) and rather, renal vasodilatation (Hanley et al., 1972; Falicov et al., 1975) have been observed in previous studies. The RNA decreased below the control level in the dog with intact afferent nerves as seen in the present study is compatible with the failure of renal vasoconstriction or the renal vasodilatation. The failures of vasoconstriction under myocardial ischemia have been shown also in the carotid and the mesenteric vascular beds (Maximov and Brody, 1976) and the vascular beds in the hind limb (Toubes and Brody, 1970). The renal vasodilatation due to the decreased level of RNA, and failures of vasoconstriction in the other vascular beds may have resulted in the progressive fall in arterial pressure observed in the dog with intact afferent nerves. Such a progressive reduction in arterial pressure during coronary artery occlusion can decrease the afterload to the ischemic heart.

On the other hand, augmentation of contractility of the ischemic heart may not have occurred in the dog with intact afferent nerves, because of the almost unchanged cardiac sympathetic nerve activity after the time of balance during coronary occlusion. Although both the unchanged contractility and the decreased afterload are induced by the different sympathetic reflex responses, the latter may have resulted in a reduced level of myocardial oxygen consumption, thereby preventing the progressive deterioration of the ischemic heart. Furthermore, the decrease in pump performance resulting from myocardial ischemia may have been compensated for by increased ventricular emptying due to the decreased cardiac afterload.

To summarize, we conclude that the reflex responses in cardiac and renal sympathetic nerve activities during the coronary occlusion are quantitatively different, and that the renal nerve activity is inhibited significantly more than the cardiac sympathetic nerve activity by the reflex mediated through the afferent vagal nerves.

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