Site of Myocardial Infarction

A Determinant of the Cardiovascular Changes Induced in the Cat by Coronary Occlusion

PETER B. CORR, PH.D., DAVID L. PEARLE, M.D., JOHN R. HINTON, WILLIAM C. ROBERTS, M.D., AND RICHARD A. GILLIS, PH.D.

SUMMARY The influence of site of acute myocardial infarction on heart rate, blood pressure, cardiac output, total peripheral resistance (TPR), cardiac rhythm, and mortality was determined in 58 anesthetized cats by occlusion of either the left anterior descending (LAD), left circumflex or right coronary artery. LAD occlusion resulted in immediate decrease in cardiac output, heart rate, and blood pressure, an increase in TPR, and cardiac rhythm changes including premature ventricular beats, ventricular tachycardia, and occasionally ventricular fibrillation. The decrease in cardiac output and increase in TPR persisted in the cats surviving a ventricular arrhythmia. In contrast, right coronary occlusion resulted in a considerably smaller decrease in cardiac output. TPR did not increase, atrioventricular conduction disturbances were common, and sinus bradycardia and hypotension persisted in the cats recovering from an arrhythmia. Left circumflex ligation resulted in cardiovascular changes intermediate between those produced by occlusion of the LAD or the right coronary artery. Mortality was similar in each of the three groups. We studied the coronary artery anatomy in 12 cats. In 10, the blood supply to the sinus node was from the right coronary artery and in 2, from the left circumflex coronary artery. The atrioventricular node artery arose from the right in 9 cats, and from the left circumflex in 3. The right coronary artery was dominant in 9 cats and the left in 3. In conclusion, the site of experimental coronary occlusion in cats is a major determinant of the hemodynamic and cardiac rhythm changes occurring after acute myocardial infarction. The cardiovascular responses evoked by ligation are related in part to the anatomic distribution of the occluded artery.

CLINICAL DATA strongly suggest that cardiovascular changes occurring with human myocardial infarction vary importantly depending on the site of myocardial infarction. For example, second- and third-degree heart block occurs more frequently after posterior wall infarction (usually a consequence of right coronary artery compromise) than after anterior wall infarction (associated with left anterior descending lesions).1-3 Posterior wall infarcts often are associated with increased parasympathetic activity (sinus bradycardia, transient hypotension, or atrioventricular block), whereas anterior wall infarcts are associated with sympathetic overactivity (sinus tachycardia or transient hypertension).5 Heart rate and left ventricular filling pressure may be higher and stroke index and stroke work index lower in patients with anterior wall infarctions compared to those with posterior wall infarctions.6 Some studies suggest a higher mortality rate in patients with anterior wall infarcts than in those with posterior infarcts, although identical mortality rates have also been reported.10

These clinical data suggest that the cardiovascular consequences of human myocardial infarction vary dramatically according to the site of infarction. However, experimental evidence for this is almost totally lacking. We are aware of only two studies relevant to this question. In experiments performed in 1918, T wave changes and mortality incidence were compared when each major coronary vessel of the dog was occluded.11 More recently, the incidence of ventricular fibrillation and mortality were compared after occlusion of each major coronary artery of the dog.12 The purpose of our study was to compare systematically the cardiovascular events occurring with occlusion of the three major coronary vessels. Our studies were performed in the cat, since we were able to demonstrate that the coronary distribution in this animal is more nearly analogous to human anatomy than that of the dog.

Methods

Adult cats unselected as to sex and ranging in weight from 1.5 to 3.9 kg were anesthetized with intravenously administered α-chloralose (70–75 mg/kg). A tracheotomy was performed and mechanical ventilation was instituted with room air, at a tidal volume of 20–25 ml/kg and a rate of 22 breaths/min. Under these conditions, the pH of arterial blood ranged between 7.38 and 7.43. The cats were immobilized with decamethonium bromide (0.25 mg/kg) every 45–60 minutes. Catheters were inserted into the right femoral artery and vein of all cats to permit measurement of blood pressure and administration of drugs. Body temperature was maintained between 37.0°C and 38.0°C by an infrared lamp.

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The heart was exposed by excising the 2nd through 5th right ribs. The parietal pericardium was incised and sutured to the chest wall. Coronary occlusion was performed after exposing one of the three major coronary arteries and placing a ligature beneath its most proximal portion. Care was taken to separate the pericoronary nerves from the coronary arteries to prevent their inclusion in the ligature surrounding the vessel. The suture material (no. 4.0) was placed proximal to all branch points of each major vessel. This ligature was tied securely at the moment when occlusion was desired. Only one vessel was occluded in each cat, and the effects were followed visually for 2 hours. At the end of each experiment the ligature was checked to confirm that the vessel had been completely occluded. Systemic arterial pressure, heart rate, and rhythm [lead II of the electrocardiogram (ECG)] were continuously monitored on a Beckman multichannel recorder. "Severe ventricular arrhythmia" was considered to be sustained ventricular tachycardia or fibrillation resulting in a mean arterial pressure of less than 50 mm Hg for at least 5 seconds. Ascending aortic flow was measured with a Biotronex BL-610 pulsed-logic flowmeter utilizing a Biotronex electromagnetic flow transducer (BL-6060-E20). The flow probe was calibrated in vitro using saline. The diastolic level of aortic flow obtained from the pulsatile flow trace was used as the zero flow reference point. A Gould-Brush model 260 recorder was used to monitor aortic flow. Total peripheral resistance was calculated as peripheral resistance units (PRU):

\[
\text{PRU} = \frac{\text{mean arterial blood pressure (mm Hg)}}{\text{mean aortic flow (ml/min)}} \times 60 \text{ (sec/min)}
\]

Coronary blood supply to the sinoatrial and atrioventricular nodes was studied in an additional 12 cats. In 10, their major coronary arteries were cannulated and perfused with Vynlite solution using the method described by James. Once the cast had hardened, the two major coronary arteries arising from the aorta were freed from surrounding tissue under direct vision using a dissecting microscope. The sinus node and atrioventricular node arteries were identified.

Confirmation of the results provided by gross dissection of the coronary arteries was obtained by serially sectioning two additional whole hearts and histologically identifying both the sinoatrial and atrioventricular nodes along with their respective blood supplies. The hearts were excised and placed in buffered formalin. Following fixation, the hearts were processed in alcohol and xylene and embedded in paraffin, and a total of 5,520 sections (2,400 from heart 1 and 3,120 sections from heart 2) were cut at 6-μm intervals. Every fifth section was retained and stained by elastic—van Gieson or Movat methods. The nodal tissue was identified using the criteria described by Lev.

The following drugs were used: α-chloralose (Établissements Kuhlman, Paris), decamethonium bromide solution (Burroughs Wellcome), and sodium heparin injection (Organon). α-Chloralose was dissolved by heating in distilled water and the solution was cooled to body temperature before use. Doses of drugs were calculated and administered as the respective salt. The vinyl injection solution (blue and red) used to cast the coronary arteries was obtained from Carolina Biological Supply Co.

The data were analyzed by paired comparisons and grouped Student's t-test. Chi-square analysis for 2 × 2 contingency with the Yates corrections was applied to some of the data and this is indicated under Results. The criterion used for significance was P < 0.05.

**Results**

**LEFT ANTERIOR DESCENDING (LAD) OCCLUSION**

Data from 30 cats with LAD ligation, 20 of which have been reported previously, are summarized in Table 1. The ECG record of a representative cat is presented as part of Figure 1. With occlusion of the LAD, decreases in cardiac output (measured as aortic flow), heart rate, and blood pressure occurred within minutes (Table 1), and were followed by changes in cardiac rhythm. The decreases in each index were usually maximal within 2 minutes after occlusion and remained constant until the arrhythmia intervened. Therefore, the values in Table 1 were obtained immediately before the development of the arrhythmia. The arrhythmia consisted of unifocal or multifocal premature ventricular beats and occurred in all 30 cats after LAD occlusion. The time to onset of the arrhythmia in the LAD ligation group, as noted by the first ventricular premature beat, was 2.4 minutes. Eleven of the 20 cats developed severe ventricular arrhythmias and five of the 11 died (Table 2), all within 3.5 minutes after coronary occlusion. The other five cats spontaneously recovered and sinus rhythm eventually returned. The average duration of arrhythmia in the 24 surviving cats was 33.5 ± 1.5 minutes.

Total peripheral resistance (TPR) was calculated from the arterial pressure and aortic flow data of five cats (Table 1). Immediately after occlusion and before the arrhythmia, TPR increased significantly, and this increase still was present 1 hour later when the sinus rhythm was reestablished.

Data obtained from 24 surviving cats 1 hour after occlusion, at which time the arrhythmia no longer was present, also are summarized in Table 1. Both heart rate and blood pressure had returned to preocclusion levels. Some restoration of cardiac output had occurred but the difference between the values 1 hour after occlusion and the values before occlusion still were significant.

**RIGHT CORONARY ARTERY OCCLUSION**

These changes in 14 cats are summarized in Table 1. The ECG record of a representative cat appears as part of Figure 1. Cardiac output decreased only mildly and TPR did not increase significantly. The time to onset of the ventricular arrhythmia was significantly longer than after LAD occlusion. Thirteen of 14 cats developed ventricular arrhythmias, and in one cat this arrhythmia became severe (Table 2). The ventricular arrhythmia consisted of either unifocal or multifocal ventricular beats. The nature of the arrhythmias differed from LAD ligation. Four of the 14 cats developed second- or third-degree heart block, arrhythmias which never occurred after LAD ligation (Table...
Table 1 Influence of Site of Coronary Occlusion on Heart Rate (HR), Blood Pressure (BP), Aortic Flow, and Total Peripheral Resistance (PRU)

<table>
<thead>
<tr>
<th>Site of occlusion</th>
<th>Before occlusion (control)</th>
<th>Immediately after occlusion and prior to arrhythmia</th>
<th>1 hr after occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR (beats/min)</td>
<td>Mean BP (mm Hg)</td>
<td>Aortic flow (ml/min)</td>
</tr>
<tr>
<td>Left anterior descending coronary artery (30)</td>
<td>195.2 ± 5.5</td>
<td>110.5 ± 4.7</td>
<td>284.0 ± 9.8</td>
</tr>
<tr>
<td>Right coronary artery (14)</td>
<td>203.0 ± 4.8</td>
<td>120.5 ± 6.6</td>
<td>250.0 ± 21.0</td>
</tr>
<tr>
<td>Circumflex coronary artery (14)</td>
<td>200.5 ± 5.6</td>
<td>103.5 ± 5.4</td>
<td>254.2 ± 23.7</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± se. Numbers in parentheses indicate number of cats in each group.

* P < 0.05 with paired comparisons (comparison was made between data obtained during postocclusion period and data obtained during preocclusion period).
† P < 0.05 with group comparisons (comparison was made between data obtained with either circumflex coronary group or right coronary group vs. left anterior descending coronary group).
‡ One cat never developed an arrhythmia; two cats exhibited infrequent premature ventricular beats.

2). The heart block observed in these four cats occurred early after occlusion (1.2 ± 0.3 minutes) and was usually of short duration. Heart block reappeared in two of the four cats after an intervening period of sinus rhythm. In two of the four cats, heart block was followed by a ventricular arrhythmia.

One hour after right coronary artery occlusion there was no increase in TPR, the heart rate and blood pressure remained decreased (in contrast to the LAD results) (Table 1), and the duration of the ventricular arrhythmias was extremely variable, ranging from 6 to 48 minutes in nine of the cats that recovered. Data on the four cats that did not recover from the arrhythmia are not included in Table 1. In the group with LAD occlusion the range was 18–49 minutes and each cat that survived the bouts of ventricular fibrillation or premature ventricular beats recovered from the arrhythmia about 30 minutes after the artery was occluded.

Similarities between the responses that occurred with the two sites of occlusion are as follows: (1) equivalent maximal decreases in heart rate and blood pressure immediately after occlusion (Table 1); (2) equivalent incidence in the occurrence of severe ventricular arrhythmias (Table 2); and (3) significant decreases in cardiac output 1 hour after occlusion (Table 1).

**LEFT CIRCUMFLEX CORONARY ARTERY OCCLUSION**

This ligation in 14 cats resulted in cardiovascular changes intermediate between those after ligation of the LAD and right coronary arteries. Initially a significant decrease in aortic flow occurred (Table 1), but it was significantly less than that following LAD occlusion. No significant change occurred in TPR. For all three coronary arteries similar decreases in heart rate and blood pressure occurred immediately after occlusion (Table 1). Thirteen of the 14 cats developed an arrhythmia (Table 2), and the time to onset of the ventricular arrhythmia was significantly longer than that after LAD occlusion (Table 1). The ventricular arrhythmias were similar to those in the two other groups. Death due to ventricular fibrillation occurred in four of 14 cats (Table 2). The incidence of premature ventricular contractions was similar in all three groups (Table 2). As with right coronary artery occlusion, several cats developed heart block (Table 2). Again, this arrhythmia developed early (1.3 ± 0.3 minutes) and before the development of ventricular premature beats. As with LAD occlusion, all cats that did not develop a rhythm disturbance that progressed to fatal ventricular fibrillation eventually returned to sinus rhythm. The mean duration of the ventricular arrhythmia was 19.9 ± 3.2 minutes, and although shorter than the arrhythmia duration with LAD occlusion, the mean value was not significantly different. The ECG record of a representative cat is presented as part of Figure 1. The summarized data at 1 hour after ligation appears in Table 1. Heart rate returned to preocclusion levels as seen with LAD occlusion. However, as in the case of right coronary ligation, blood pressure remained depressed and TPR was unchanged. Unlike results from either of the other two groups, aortic flow was not significantly reduced at this time.

**ARTERIES TO THE SINUS AND ATRIOVENTRICULAR NODES**

Twelve hearts were studied. Both the sinus and atrioventricular nodes were supplied primarily from branches of the right coronary artery. In two of the 12 cats a branch of the left circumflex artery provided the blood supply to the sinus node. In three of the 12 cats a branch of the left circumflex artery provided the blood supply to the atrioventricular node. In the cats in which the nodes were supplied by branches of the right coronary artery, the branch to the sinus node appeared as the first major
branch off the right coronary artery (Fig. 2), while the branch to the atrioventricular node appeared as the last branch of the right coronary artery just before this major artery became the posterior descending coronary artery (Fig. 3). In the few cats in which the nodes were supplied by the circumflex artery, the artery to the sinus node appeared as the first branch of the left circumflex artery, while the vessel to the atrioventricular node appeared as a branch of the left circumflex artery just before this major artery branched into the posterior descending coronary artery. The origin of the blood supply to the posterior descending artery followed the same pattern as the origin of the atrioventricular node artery. The posterior descending artery was an extension of the right coronary artery in nine (Fig. 3) and an extension of the left circumflex artery in three of the 12 cats studied. (These results confirm earlier findings of Abramson and colleagues, who showed that the majority of cats are right coronary domi-
nant.) Hearts undergoing serial sectioning exhibited the following pattern: One had the sinus node supplied by a branch from the right coronary artery, the other had the sinus node supplied by the left circumflex artery; in both the atrioventricular node was supplied by a branch of the right coronary, and the posterior descending artery appeared as an extension of the right coronary artery (Fig. 4).

Discussion

Occlusion of the three major coronary arteries of the cat produced dissimilar cardiovascular effects. Moreover, the changes after ligation of the feline right coronary artery were similar in many respects to the effects of human posterior infarction, while LAD ligation caused changes analogous to human anterior infarction.

Four major differences in cardiovascular effects were observed and are as follows: (1) occurrence of heart block with right coronary or left circumflex ligation but not with LAD ligation; (2) persistence of sinus bradycardia with right coronary ligation but not with LAD or left circumflex ligation; (3) major decreases in cardiac output initially with LAD occlusion but only minimal decreases in cardiac...
output after right coronary ligation; and (4) increase in TPR with LAD ligation but not with right coronary or circumflex ligation.

The occurrence of heart block with right coronary and left circumflex but not with LAD occlusion may be related to the source of the blood supply to the atrioventricular node, to the anatomical location of cholinergic nerve fibers, or to both factors. The artery supplying the atrioventricular node of the cat was shown in the present study to originate from the right coronary artery in nine of 12 cats and from the circumflex in three of 12 cats. Presumably, lack of sufficient oxygen to the atrioventricular node leads to impaired conduction and heart block. Alternatively, numerous cholinergic nerve endings and ganglia have been described between the posterior margin of the atrioventricular node and the anterior wall of the coronary sinus. This area usually is perfused by arterial blood from the right coronary artery in the majority of cats. A study in the rat has shown that sinus rate slows when the sinus node artery is occluded. In addition, part of the slowing may be vagally mediated. Thoren has reported that ligation of the right coronary artery in cats increases the firing of afferent cardiac vagal fibers, and activation of these fibers by electrical stimulation has been shown to produce reflex bradycardia.

The greatest decrease in cardiac output occurred when the LAD was ligated. This was not unexpected since a greater portion of the left ventricular chamber is usually infarcted in occlusion of the LAD as compared to occlusion of the other major vessels. It has been shown that the ischemic region produced by LAD occlusion ceases to contract within 1 minute after occlusion, resulting in a rapid decompensation in ventricular function. Ventricular function then improves within 4–7 minutes by a compensatory increase in diastolic length and tension in the nonischemic portion of the myocardium. Compensation in ventricular function also may be aided by the sympathetic nervous system, since surgical removal of sympathetic ganglia of dogs before LAD occlusion has been shown to prevent the restoration of cardiac output that usually occurs hours after infarction. We found that cardiac output was returning toward preligation levels within 1 hour after infarction.

The degree of hypotension that occurred immediately after occlusion was the same regardless of which artery was occluded. The mechanism for the fall in pressure also seemed to be the same in each group, and presumably was due to the decrease in cardiac output because, in no case, was there a drop in TPR. If TPR had not risen with LAD occlusion, pressure presumably would have reached much lower levels than those seen with right coronary and left circumflex ligation.

The differences in TPR changes seen with occlusion of the various vessels might be explained by the size of the ischemic zone. In our study, we were interested only in the immediate hemodynamic effects of coronary occlusion. During this 1-hour time period, it is extremely difficult to assess quantity of ischemic myocardium, because available techniques do not define clearly the difference between ischemic and normal myocardium.

Alternatively, the variation in TPR response might be related to different reflex responses rather than to quantity of ischemic muscle. The increase in TPR after LAD occlusion is a predictable response. Hypotension immediately after occlusion reduces stretch on sensory elements in the carotid sinus and aortic arch, resulting in excessive discharge of sympathetic efferent nerves to the vasculature. Consequently, TPR should rise. A second factor which might increase TPR is catecholamine release from the
adrenal glands. LAD occlusion has been shown experimentally to increase plasma catecholamine levels via a vagal afferent reflex. Such an increase in adrenal medullary hormone could explain the elevated TPR in our cats 1 hour after LAD occlusion, a time when arterial pressure had returned to the preocclusion level.

The unexpected finding, therefore, is the absence of a rise in TPR with right coronary or left circumflex occlusion despite significant hypotension immediately and 1 hour after occlusion. This result suggests that interruption of blood flow in these arteries in some way inhibits the reflex sympathetic vasoconstrictor response that normally occurs when arterial pressure falls. Moreover, the previously mentioned reflex by which LAD occlusion releases catecholamines from the adrenal glands does not appear to be activated by right and left circumflex occlusions.

It is generally accepted that the high incidence of sudden death during acute coronary occlusion results from cardiac arrhythmias. Ventricular fibrillation was the terminal event in all cats dying in our study. Except for one cat in which fibrillation developed 33 minutes after occlusion, this arrhythmia occurred within 4 minutes after occlusion. The frequency of severe arrhythmia and death due to ventricular fibrillation was not significantly different for the three coronary ligations.

The cardiovascular data demonstrated in our study would not be relevant to human myocardial infarction unless the coronary artery distribution is similar in the two species. Our data suggest that the coronary artery anatomy of the cat is similar to that of the human: (1) both species have right coronary dominant systems; (2) in both species the atrioventricular node is perfused primarily by an artery originating from the right coronary artery; and (3) in both species the sinus node is perfused primarily by an artery originating from the most proximal portion of the right coronary artery.

In contrast, the dog (most commonly chosen for studies dealing with acute myocardial infarction) possesses a coronary artery distribution dissimilar to that of the human. In the dog, the atrioventricular node is supplied from a branch of the circumflex artery; the sinus node artery arises not from the proximal portion of the right coronary artery, but most commonly from the distal third of this artery as a terminal branch. Additionally, the crux of the heart usually is supplied from a posterior descending artery originating from the circumflex artery. Compared to the dog, the cat seems to be a better model for assessing the role of the occlusion site in human myocardial infarction.

Functionally, our data suggest that the cardiovascular changes that occur in the cat after experimentally induced coronary occlusion are analogous to those changes that occur in man. Sinus bradycardia and high degrees of atrioventricular block particularly were associated with posterior wall myocardial infarction in both cat and man. One apparent discrepancy between the cat and the human is the finding of Adgey and colleagues that bradycardia does not occur within the first 30 minutes after onset of symptoms of an anterior wall infarction. Bradycardia was a prominent event after LAD occlusion in our study, but was very brief (occurring only over the first 1-3 minutes after occlusion). This short-lived bradycardia may have been missed in even the earliest human studies. Webb and colleagues, observing patients within the 1st hour after symptoms of acute myocardial infarction, have reported that a large percentage with posterior wall infarcts show signs of parasympathetic overactivity, whereas a large percentage with anterior wall infarcts show signs of sympathetic overactivity. This agrees with our findings in cats. Cats with right coronary occlusion exhibited sinus bradycardia, heart block, and hypotension. Cats with LAD occlusion exhibited an increase in TPR and eventual recovery from their early hypotension. Furthermore, eight of the 24 cats with LAD occlusion that recovered exhibited sinus tachycardia at 1 hour after occlusion (+25.7 ± 4.8 beats/min). Finally, we have reported in an earlier study that the ventricular arrhythmia associated with LAD occlusion is maintained by the sympathetic nervous system.

Thus, the differences observed in human infarction sites are mirrored by the contrast between LAD and right coronary ligations in the cat. The different consequences of anterior and posterior infarction might imply different therapy according to infarction site. The cat appears to be a good model to investigate these differences.

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Intrarenal Site of Action of Calcium on Renin Secretion in Dogs

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THOMAS E. LOHMEIER, PH.D., AND RONALD H. FREEMAN, PH.D.

SUMMARY We studied the effects of intrarenal calcium infusion on renin secretion in sodium-depleted dogs in an attempt to elucidate the major site of calcium-induced inhibition of renin release. Both calcium chloride and calcium gluconate reduced renal blood flow and renin secretion while renal perfusion pressure was unchanged. These data indicate that calcium inhibition of renin secretion did not occur primarily at the renal vascular receptor; decreased renal blood flow is usually associated with increased renin secretion. Calcium chloride infusion increased urinary chloride excretion without affecting sodium excretion, and calcium gluconate failed to increase either sodium or chloride excretion. Also, the filtered loads of sodium and chloride were unchanged during the calcium infusions. These results give no indication that calcium inhibited renin secretion by increasing the sodium or chloride load at the macula densa. The effects of intrarenal calcium infusion on renin release were also assessed in dogs with a nonfiltering kidney in which renal tubular mechanisms could not influence renin secretion. The observation that calcium still suppressed renin release in these dogs provides additional evidence that the major effect of calcium involved nonnontubular mechanisms. Thus, it appears likely that calcium acted directly on the juxtaglomerular cells to inhibit renin secretion.

IT HAS BEEN demonstrated recently that intrarenal calcium administration inhibits renin secretion.1–5 Suppressed renin release was associated with a paniatriasis and these investigators suggested that calcium might act by increasing renal tubular sodium load at the macula densa. Also, it had been suggested earlier by Suki et al.4 that calcium might inhibit sodium transport by the thick ascending limb of the loop of Henle. However, recent evidence indicates that chloride, rather than sodium, is actively transported in the ascending limb of Henle's loop.5, 6 In view of this finding, the possibility has been considered7 that chloride might be sensed by the macula densa and influence renin secretion. Thus, calcium might alter renin secretion by influencing the renal tubular handling of chloride. It is also possible that calcium suppressed renin release by altering the renal vascular receptor mechanism, by influencing a hormone or intracellular mediator, or by a direct action on the juxtaglomerular cells.

The present study was conducted to determine the intrarenal site of action of calcium to suppress renin secretion. The effects of intrarenal calcium infusion on renal hemodynamics and electrolyte excretion were evaluated in an attempt to determine whether the primary renin inhibition occurred at a renal vascular or tubular site or in the juxtaglomerular cells. Also, calcium was infused intrarenally into dogs with a nonfiltering kidney in which tubular control of renin secretion had been eliminated.

Methods

Twenty-three experiments were conducted on 15 female hounds weighing 19.0 ± 1.5 (SEM) kg; range, 14–23 kg. To augment the rate of renin secretion, the dogs were sodium-depleted before the acute experiment. The dogs were fed a low sodium diet containing less than 5 mEq of sodium per day (Riviana Foods) and given intramuscular injections of meralluride (Mercuhydrin), 2 ml, on days 2 and 3 before the experiment. The average net loss of sodium was 138 ± 12 mEq as determined from the daily sodium balance measurements.

On the morning of the acute experiment each dog was
Site of myocardial infarction. A determinant of the cardiovascular changes induced in the cat by coronary occlusion.

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