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**Effects on Myocardial Contractility of Blood-Borne Material Released from the Feline Small Intestine in Simulated Shock**

**Ove Lundgren, M.D., Ph.D., Ulf Haglund, M.D., Ph.D., Olle Isaksson, M.D., Ph.D., and Tetsuo Abe, M.D.**

**SUMMARY** There is a pronounced derangement in cardiovascular function in the cat after a 2- or 3-hour period during which shock is simulated in the small intestine by regional hypotension (BP = 30–35 mm Hg) during activation of vasoconstrictor nerve fibers. It has been proposed that these effects are caused by blood-borne cardiodepressant substance(s) released from the "shocked" small intestine. To obtain further evidence for this hypothesis we performed a study on two heart preparations in vitro. Rabbit papillary muscles or isolated beating rat hearts were exposed to intestinal venous plasma obtained from control cats and from cats subjected to simulated intestinal shock for 2 or 3 hours. While control plasma induced only a slight depression of myocardial contractility, plasma from "shocked" intestine caused a significant decrease in peak isometric tension of the papillary muscles or a fall in systolic pressure of the rat hearts. Since the experiments on papillary muscle indicated that time to peak tension was largely unaffected by the plasma samples, we conclude that the feline intestine in shock releases material into blood that exerts a negative inotropic effect on the myocardium. From the Departments of Physiology and Surgery II, University of Göteborg, Göteborg, Sweden. Supported by grants from the Swedish Medical Research Council (14X-2835 and 875-4802), from the Clara and Lily Dabiström Fund, from the Harald and Greta Jeansson Fund, and from the Faculty of Medicine, University of Göteborg. Dr. Abe was on leave of absence from the Department of Surgery, Yokohama City University, Yokohama, Japan. A brief report of parts of this study has been published as an abstract in Acta Physiol Scand. 396 (suppl 118), 1973. Received June 3, 1975; accepted for publication December 16, 1975.
EXPERIMENTS ON CATS

The experimental procedures for the experiments on cats have been described in detail elsewhere and only a brief summary is given here. The cats were anesthetized with chloralose (50 mg/kg, iv). They had been deprived of food for 12 hours and had no obvious signs of intestinal diseases. An intestinal segment weighing 50–70 g was isolated and the remainder of the intestine, the spleen, the greater omentum, and the major part of the pancreas were extirpated. The superior mesenteric artery was the only artery supplying the isolated intestinal segment. It was dissected free close to the aorta and surrounded by an adjustable clamp so that the regional arterial inflow pressure could be lowered to any desired level. The left adrenal gland was denervated and the vessels supplying the right adrenal were ligated. The nerves surrounding the superior mesenteric artery were cut and their distal ends were placed on a ring electrode for electrical stimulation. Atropine (1 mg/kg, iv) was given. After heparinization the left femoral artery and a minor branch of the superior mesenteric artery distal to the adjustable clamp were cannulated to record blood pressure by a pressure transducer (Statham P23). The venous outflow from the intestinal segment and its lymph nodes was recorded continuously by an optical drop recorder and an ordinate writer. Venous outflow pressure was set at 10 mm Hg. The blood was returned to the animal through a catheter in a jugular vein. Recordings were made on a Grass polygraph.

A slow intravenous infusion of a glucose solution containing bicarbonate (10 mmol of NaHCO$_3$ per 100 ml of 10% glucose solution, given at a rate of 0.1–0.2 ml/min) was started on induction of anesthesia and continued throughout the experiment. This infusion has been shown to maintain arterial pH at a normal level, despite the operative trauma and intestinal hypotension.

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Methods
SOLUTIONS

In the initial experiments on isolated rabbit papillary muscles the bathing solution had the following composition (mmol/liter): NaCl, 122; KCl, 4.7; NaHCO₃, 15.5; KH₂PO₄, 1.2; MgCl₂, 1.2; CaCl₂, 2.5; and glucose, 11.5. This solution was modified during the course of experiments in which we tested the effect of the various ions on the papillary muscle contractility. In Table 1 are listed the compositions of the different solutions used. The solution that we used as a control when testing plasma samples was identical to that of the Na⁺ series but did not contain mannitol (Table 1). The plasma samples were heated to 37°C before addition to the organ bath. To avoid foaming when gassing the samples, polysorbate 20 (Tween 20), 1-5 µl, was added to the plasma samples in the organ bath. The pH of all plasma samples was corrected to 7.35-7.40 when gassed with 4% CO₂ in O₂.

Krebs-Henseleit bicarbonate buffer* containing the disodium salt of ethylenediaminetetraacetic acid (EDTA), 0.5 mM, was used as the perfusion medium in the experiments on isolated hearts. The disodium salt of EDTA was included in the Na+ series but did not contain mannitol (Table 1). The solution that we used as a control when testing plasma samples was identical to that of the Na⁺ series but did not contain mannitol (Table 1). The pH of all plasma samples was corrected to 7.35-7.40 when gassed with 4% CO₂ in O₂.

Biochemical Analyses

Plasma concentrations of Na⁺, K⁺, and Ca²⁺ were determined by an Eppendorf flame photometer.

Statistics

The quantitative effect of any given test solution on the papillary muscle was defined as the ratio (R) of peak isometric tension developed in the test solution to that developed in the control solution, i.e.,

\[ R = \frac{\text{peak isometric tension, test solution}}{\text{peak isometric tension, control solution}} \]  

(1)

More than one muscle was used to test each plasma sample, and to calculate the average value we used the geometric rather than arithmetic mean. Thus, if the isometric tension of two muscles doubled and that of two muscles was halved, the geometric mean was 1.00 in contrast to the arithmetic mean of 1.25. The geometric mean isometric tension ratio (\( \bar{R} \)) of \( n \) muscles was calculated conventionally as

\[ \bar{R} = \text{antilog} \left( \frac{1}{n} \sum_{i=1}^{n} \log R_i \right) \]  

(2)

For statistical purposes log \( \bar{R} \) was used. Statistical significance was calculated by Wilcoxon's test for two samples or by Wilcoxon's matched pair signed ranks test. A \( P \) value of 0.05 or less was judged as significant.

Results

Experiments on Cats

The results of these experiments are summarized in Figure 1 (control experiments and hypotension for 2 hours) and in Figure 2 (hypotension for 3 hours). It can be seen that lowering arterial inflow pressure to about 30 mm Hg during a continuous stimulation of the regional sympathetic vasoconstrictor fibers induced an initial pronounced vasoconstriction; intestinal blood flow was reduced to 10-20% of control. Within 20-40 minutes blood flow had increased to a steady state level of 25-35% of control; this was evidently due to a decrease of regional flow resistance. In the control experiments a continuous, slow decline of blood pressure and intestinal blood flow was recorded.

Isolated Rabbit Papillary Muscles

The Influence of Various Ions on Peak Isometric Tension. We studied the effect of varying the concentration of sodium [Na⁺] in the perfusion medium on peak isometric tension in six muscles by changing the concentration of NaCl of the solution. [Na⁺] was varied between 120 and 170 mmol/liter.

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**Table 1: Composition of Control Solutions Used When Testing the Effect of Various Ions on Papillary Contractility**

<table>
<thead>
<tr>
<th>Control solutions (mmol/liter)</th>
<th>K⁺ series</th>
<th>Ca²⁺ and HCO₃⁻ series</th>
<th>Na⁺ series</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl</td>
<td>122.0</td>
<td>122.0</td>
<td>120.0</td>
</tr>
<tr>
<td>KCl</td>
<td>4.7</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>NaHCO₃</td>
<td>15.5</td>
<td>15.5</td>
<td>25.0</td>
</tr>
<tr>
<td>KH₂PO₄</td>
<td>1.2</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>MgCl₂·6H₂O</td>
<td>1.2</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>CaCl₂</td>
<td>2.5</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>Glucose</td>
<td>11.5</td>
<td>11.5</td>
<td>11.5</td>
</tr>
<tr>
<td>Mannitol</td>
<td></td>
<td></td>
<td>47.0</td>
</tr>
</tbody>
</table>

---

*Figure 1: Left panel: Blood pressure, intestinal blood flow, and intestinal PRU₁₀₀ in the control animals of the present study (\( n = 10 \)). Right panel: Changes in intestinal blood flow and regional flow resistance induced by simulated shock in the small intestine for 2 hours (\( n = 9 \)). During the period of reduced perfusion pressure the regional vasoconstrictor fibers were stimulated electrically at 6 Hz. Bars denote SE.*
mmol/liter, the range usually encountered in plasma of cats used in this study. Since such large variations in [NaCl] would cause corresponding changes in osmolality, this was kept constant at 348 mOsmol/kg by adding appropriate amounts of mannitol. The results are presented in the upper panel of Figure 3, where peak isometric tension is expressed as a fraction of that observed at an [Na+] of 145 mmol/liter. It is evident that as [Na+] increases, peak isometric tension decreases.

In another series of experiments (seven muscles), [K+] was varied between 2 and 5 mmol/liter by changing the concentration of KCl. The results are presented in the lower panel of Figure 3, peak isometric tension being expressed as a fraction of that in control solution containing 5.9 mmol of K+ per liter. There were no large changes in contractile force although a slight increase was noted at the lowest values of [K+]. Increasing [K+] to 8 times control in the perfusion medium totally abolished contraction of the papillary muscles.

The effect of changing [Ca2+] in the organ bath was tested by varying [CaCl2] in the solution. The results are illustrated in the upper panel of Figure 4. The control solution contained 2.5 mmol of Ca2+ /liter. An almost linear relationship existed between the calcium concentration and the peak isometric tension developed by the papillary muscle.

By changing [HCO3-], pH of the incubation medium was varied and the results obtained for six muscles are shown in the lower panel of Figure 4. [HCO3-] of the control solution was 15 mmol/liter. With increasing [HCO3-] and, hence, increased pH peak, contractile tension increased. The pH of the solution containing 25 mmol of HCO3- per liter was 7.42 when gassed with 4% CO2 in O2.

Influence of Control and Shock Plasma on Peak Isometric Tension. All intestinal venous plasma samples were tested on one of the bioassay systems used. With few exceptions the samples were also analyzed chemically for concentrations of Na+, K+, and Ca2+ (Tables 2 and 3). Before the plasma samples were tested on the papillary muscles, pH was adjusted to 7.35-7.40 when the solution was gassed with 4% CO2 in O2.

The effect of the plasma samples on peak isometric tension was observed for 15-20 minutes (Figure 5). Ten different control plasma samples were tested on a total of 39 muscles (Table 2). The cumulative data obtained for control plasma are shown in Table 2 and in Figure 5, which illustrates how peak isometric tension changed with time.
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FIGURE 4 The relative changes in peak isometric tension of rabbit papillary muscles induced by altering [Ca\(^{2+}\)] and [HCO\(_3\)\(^-\)] in the incubation solution. R represents the ratio of peak isometric tension in test solution to that developed in control solution.

during incubation in plasma and after rinsing. It is evident from the results obtained that control plasma caused a slight reduction of the peak tension developed by the papillary muscle.

Experiments corresponding to those described above for control plasma also were performed with intestinal venous plasma from "shocked" animals. Plasma from nine cats in which simulated intestinal shock was maintained for 2 hours was assayed on a total of 38 papillary muscles. In six cats the shock period was prolonged to 3 hours and the plasma samples from these experiments were tested on 28 muscles. Shock plasma markedly reduced peak isometric tension, as shown in Figure 5 and Table 3, and this reduction was statistically different from that of control plasma. This significant difference persisted after rinsing (Fig. 5).

Figure 5 and Table 3 suggest that 2-hour shock plasma caused more marked reduction of peak isometric tension than 3-hour shock plasma, although the difference did not reach statistical significance according to the Wilcoxon two-sample test. It is, however, clear from Table 3 that [Ca\(^{2+}\)] of two of the 2-hour plasma samples (samples 7 and 9) was decidedly lower than in all other samples of shock plasma. Since an adequate [Ca\(^{2+}\)] in the incubation medium is of great importance for contraction, the low [Ca\(^{2+}\)] of plasma samples 7 and 9 in all probability enhanced their ability to lower peak isometric tension. If these samples are excluded from the material, one arrives at a log R value of 

$$-0.317 \pm 0.044 \text{ (mean} \pm \text{SE); n} = 6$$

corresponding to an R value of 0.48.

In an attempt to determine whether the cross-sectional area of the papillary muscles was of significance in relation to responses to control or shock plasma, we compared the effects of plasma on "thin" muscles (cross-sectional area <0.6 mm\(^2\)) and on "thick" muscles (cross-sectional area >0.9 mm\(^2\)). No statistically significant difference could be demonstrated.

Since a small volume of Tween 20 was added to all plasma samples to avoid foaming, we tested the effect of this agent on peak isometric tension on eight muscles. About 5 µL of

TABLE 2 Experimental Results and Electrolyte Composition of Control Plasma Assayed on the Papillary Muscles

<table>
<thead>
<tr>
<th>Plasma sample no.</th>
<th>No. of muscles</th>
<th>Peak isometric tension</th>
<th>Time to peak tension (msec)</th>
<th>Plasma concentration (mmol/liter)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>R</td>
<td>log R</td>
<td>Control</td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>1.35</td>
<td>0.1312</td>
<td>104</td>
</tr>
<tr>
<td>2</td>
<td>1.06</td>
<td>0.0275</td>
<td>100</td>
<td>120</td>
</tr>
<tr>
<td>3</td>
<td>1.04</td>
<td>0.0185</td>
<td>85</td>
<td>90</td>
</tr>
<tr>
<td>4</td>
<td>0.88</td>
<td>-0.0578</td>
<td>115</td>
<td>108</td>
</tr>
<tr>
<td>5</td>
<td>0.78</td>
<td>-0.1102</td>
<td>116</td>
<td>111</td>
</tr>
<tr>
<td>6</td>
<td>0.77</td>
<td>-0.1135</td>
<td>119</td>
<td>118</td>
</tr>
<tr>
<td>7</td>
<td>0.75</td>
<td>-0.1251</td>
<td>110</td>
<td>102</td>
</tr>
<tr>
<td>8</td>
<td>0.72</td>
<td>-0.1413</td>
<td>110</td>
<td>102</td>
</tr>
<tr>
<td>9</td>
<td>0.71</td>
<td>-0.1478</td>
<td>110</td>
<td>102</td>
</tr>
<tr>
<td>10</td>
<td>0.70</td>
<td>-0.1556</td>
<td>151</td>
<td>153</td>
</tr>
<tr>
<td>(\bar{X})</td>
<td>-0.0674</td>
<td>111.1</td>
<td>112.0</td>
<td>142.9</td>
</tr>
<tr>
<td>SD</td>
<td>0.0960</td>
<td>18.3</td>
<td>18.0</td>
<td>15.6</td>
</tr>
<tr>
<td>SE</td>
<td>0.0304</td>
<td>6.1</td>
<td>6.0</td>
<td>5.2</td>
</tr>
</tbody>
</table>

Peak isometric tension is expressed in terms of a ratio, R, depicting the peak isometric tension developed by the papillary muscle after 18 minutes of incubation in the plasma divided by the peak isometric tension in the control solution.
Tween 20 were added to the control incubation medium; this reduced isometric tension slightly. At 18 minutes after addition of Tween 20 log $R$ was $-0.0873 \pm 0.0285$, corresponding to an $R$-value of 0.82. This effect was not statistically different from the effect of control plasma on peak tension.

The Influence of Control and Shock Plasma Samples on Time to Peak Tension. Time to peak tension was determined for all muscles during the control period and after incubation for 15–20 minutes in plasma; the results are given in Tables 2 and 3. Control plasma and 2-hour shock plasma induced no statistically significant change in the time to peak tension. Incubation with 3-hour shock plasma, on the other hand, caused a slight but statistically significant decrease.

ISOLATED RAT HEARTS

Control and shock plasma samples were also tested on isolated working hearts of rats. The effects of cat plasma on the myocardium were judged from the change in systolic pressure recorded in the aorta 8–10 minutes after the administration of the samples and expressed as percent of the pressure recorded under the control situation. Two samples of shock plasma, tested on nine hearts, reduced peak systolic pressure to $38 \pm 5\%$ (range, 10–74\%) of control, while three control plasma samples tested on 14 hearts induced no significant change in peak systolic pressure (Fig. 6).

The ionic composition of the plasma samples was of no critical importance for the bioassay system in these experiments, because the plasma samples were diluted 10 times by the recirculating fluid.

**Discussion**

The present study was undertaken to demonstrate the possible release of cardioactive material into the intestinal venous blood of cats after a period of simulated intestinal
from the cat small intestine in shock is not known. In order to exclude the most obvious candidates, concentrations of certain key electrolytes were determined for almost all plasma samples used in this study and pH of each plasma sample was adjusted to 7.35–7.40 before it was tested. There was no significant difference in sodium and calcium concentrations between control and shock plasma, although a very low [Ca++] may have contributed to the negative inotropic effects of certain samples of shock plasma (see Results). A significantly higher [K+] was found in shock plasma, probably because of the release of intracellular potassium from the damaged parts of the intestinal mucosa. However, control experiments (Fig. 3) showed that the observed difference in plasma [K+] could not explain the negative inotropic effect of the shock plasma, since the increase of plasma [K+] was not of sufficient magnitude to depress myocardial contractility.

The concept that humoral substances are released from splanchnic organs in shock is not new and several factors have been proposed as causes of depressed cardiac function. That investigated most thoroughly is one described by Lefer and named "myocardial depressant factor" (MDF). According to Lefer, MDF, a polypeptide with a molecular weight of 800–1000, is released into the lymph mainly from the ischemic pancreas. There are at least two observations reported in our present study and in earlier studies that seem to suggest that MDF is not responsible for the negative inotropic effects reported in this study.

First, a large portion of the pancreas was extirpated in our model of shock and the remaining part was perfused at normal arterial blood pressure. Second, our observed myocardial depressant effect was caused by material in the intestinal venous blood, whereas MDF is transported from the pancreas via the lymphatics. As a consequence of this the myocardial depression reported by Lefer was seen in vivo 60 minutes after induction of shock, while the negative inotropic effect, induced by the present shock model, was apparent within minutes, at least to judge from recordings of arterial blood pressure and cardiac output. Furthermore, Glenn and Lefer were not able to demonstrate any production of MDF by the duodenum under in vitro conditions, although MDF was produced by incubation with pancreatic tissue. These observations argue against the view that MDF is involved in the intestinal shock model, but they do not completely rule out such participation since the chemical
nature of the cardiodepressant material in this study is unknown.

It was proposed by Glenn and Lefer\(^1\) and Glenn et al.\(^2\) that lysosomal enzymes released into blood during shock from, e.g., the pancreas, is of great importance in the pathogenesis of circulatory shock. Such a chain of events could not be demonstrated in this shock model by Haglund et al.,\(^3\) who were unable to demonstrate any increase of lysosomal enzymes in intestinal venous blood after a period of simulated intestinal shock.

Lillehei\(^4\) provided experimental support for the presence of an "intestinal factor" in irreversible shock and the results of our study are in line with this general concept. However, Lillehei suggested that the deterioration of cardiovascular homeostasis was due to peripheral vascular failure secondary to a "stagnant anoxia" in the small intestine which included pooling of blood in the splanchnic region and large transcapillary fluid losses from the intestinal vascular bed. As pointed out above, none of these events seems to occur during simulated intestinal shock in the cat, and the present study strongly supports the idea that the "intestinal factor" induces heart failure.

The presence of toxic factor(s) in portal blood in connection with shock produced by occluding the superior mesenteric artery has been demonstrated experimentally by Janoff et al.\(^5\) and by Williams and co-workers.\(^6\)\(^-\)\(^11\) The latter collected blood plasma from the canine portal vein before, during, and after a 2-hour period of superior mesenteric artery occlusion. These plasma samples were assayed on isolated rat trabeculae or on cat papillary muscles in a manner similar to ours. They found that peak isometric tension decreased when the myocardial tissue was incubated with, e.g., the pancreas, is of great importance in the pathogenesis of circulatory shock. Such a chain of events could not be demonstrated in this shock model by Haglund et al.,\(^3\) who were unable to demonstrate any increase of lysosomal enzymes in intestinal venous blood after a period of simulated intestinal shock.

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In their experiments no fall in blood pressure was apparent until release of the occlusion of the superior mesenteric artery.

Fine and co-workers have championed the idea that bacterial endotoxins are the crucial substances in various types of shock. According to Fine,\(^12\) endotoxins always are absorbed from the intestinal tract but during normal conditions the reticuloendothelial system, particularly in the liver, efficiently inactivates the toxins. Fine believes that in shock, however, the liver fails in this respect because of a marked reduction in liver blood flow which is induced primarily neurogenically.

We performed no determination of endotoxins in our study, but for the following reasons it seems improbable that endotoxin was involved. First, the cardiovascular deterioration induced by endotoxin is believed to be induced mainly by vascular mechanisms, possibly via an intense pulmonary vasoconstriction.\(^12\)\(^-\)\(^13\) Moreover, endotoxins have not been shown to have any acute effects on myocardial contractility.\(^14\) Second, perfusion of the feline gut lumen with a large volume of oxygenated saline during the hypotensive period prevents the development of the mucosal lesions seen during hypotension, and then there is no fall in blood pressure after hypotension (Haglund et al., in preparation). Perfusion with nitrogenated saline, on the other hand, prevented neither the mucosal lesions nor the fall in blood pressure. Hence, there seems to be a causal relationship between the occurrence of mucosal lesions and the cardiovascular deterioration in the present shock model. Third, in our experiments the liver probably is supplied by the same volume flow of blood as in control experiments, since arterial perfusion pressure to the liver was not changed by partially occluding the superior mesenteric artery. Furthermore, the intestinal venous outflow bypassed the liver in both control and shock experiments. Hence, there probably was no difference in the detoxicating capacity of the liver between control and shocked animals.

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REFERENCES

Blood Pressure and Plasma Angiotensin II Concentration after Renal Artery Constriction and Angiotensin Infusion in the Dog

[5-Isoleucine]angiotensin II and Its Breakdown Fragments in Dog Blood

A.F. Lever, M.B., J.J. Morton, Ph.D., J.D. Powell-Jackson, M.D., J.I.S. Robertson, M.B.,
and P.F. Semple, M.B.

SUMMARY We measured arterial plasma angiotensin II concentration, renal blood flow, and arterial blood pressure in six conscious dogs during intravenous infusion of angiotensin II (5, 10, and 20 ng/kg per min). The same measurements were made on a different occasion in the same six animals, while they were conscious, before and during constriction of a main renal artery. Arterial blood pressure and plasma angiotensin II rose and renal blood flow decreased in both experiments. The similarity of regressions for plasma angiotensin II concentration and arterial blood pressure in the two experiments strongly suggests that the rise of circulating angiotensin II after renal artery constriction is sufficient to account for the hypertension by its direct pressor action. As discussed, a different mechanism seems likely to be involved in the later stages of renal hypertension. Angiotensin II is more likely to be in the 5-isoleucine form than in the 5-valine form in the dog. In contrast to the rat, plasma concentrations of the heptapeptide (angiotensin III), hexapeptide, and pentapeptide fragments of angiotensin II are low in the dog.

THE ROLE of renin in the pathogenesis of hypertension remains uncertain: after renal artery constriction in animals arterial pressure and plasma levels of renin and renin activity increase but thereafter hypertension persists while the renin level falls. Recent experiments with inhibitors of the renin-angiotensin system suggest its involvement in the early stages of renal hypertension. The purpose of the experiments described here was to determine whether the plasma concentrations of angiotensin II (the vasoactive component of the renin-angiotensin system) also rises in these circumstances and whether the levels reached are sufficient to account for the increase in arterial pressure in the early stages of renal hypertension. Scornik and Paladini used bioassay to show angiotensin blood levels to be high in early stages of renal hypertension, but normal in the later stages.

We measured arterial pressure and plasma concentrations of renin and angiotensin II in conscious dogs before and shortly after constriction of the main artery to one kidney; the results were compared with similar measurements in the same animals before and during pressor infusions of angiotensin II. The experiment was then repeated after removal of the contralateral kidney because of evidence (see Discussion) that hypertension of this type is less dependent on renin and angiotensin. To validate the angiotensin assay, we studied the immunoreactive breakdown fragments of angiotensin II in dog plasma and the reactivity of canine angiotensin II with the antisera used.

Methods

TERMINOLOGY

Dogs with one renal artery constricted and the opposite kidney removed are described as one-kidney hypertensive dogs; dogs with unilateral renal artery constriction but with the opposite or "untouched" kidney in situ as two-kidney hypertensive dogs.

TECHNIQUES FOR EXPERIMENTS ON DOGS

Six male mongrel dogs were housed prior to and during the experiment in an air-conditioned room. After a period of training, they were anesthetized with pentobarbitone (pento-
Effects on myocardial contractility of blood-borne material released from the feline small intestine in simulated shock.
O Lundgren, U Haglund, O Isaksson and T Abe

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