Acute Intestinal Infarction or Obstruction: Search for Better Laboratory Tests in an Animal Model

Steven C. Kazmierczak,1 John A. Lott,1 and James H. Caldwell2

Mesenteric vascular occlusion and intestinal obstruction are difficult-to-diagnose medical emergencies. We evaluated a large panel of biochemical markers as diagnostic and prognostic indicators in a rat model of intestinal infarction and partial and complete, and strangulated intestinal obstruction. After intestinal infarction and obstruction, laboratory data are distinctly abnormal. Serum urea nitrogen dramatically increased in all groups, but most rapidly in the groups with infarction or strangulated obstruction. Inorganic phosphorus proved to be a sensitive indicator of infarction, but less so for any form of obstruction. While all members in the infarct group demonstrated significant increases in the aminotransferases, creatine kinase, and alkaline phosphatase, such increases in the groups with obstruction were less pronounced. Serum maltase assays revealed decreasing activities in all members of the groups with complete and strangulated obstruction, but in only 17% of the rats with partial obstruction. Serum maltase activity increased from abnormally low values after surgery to abnormally high values in the six animals that recovered from partial intestinal obstruction. The proportion of hexosaminidase A (of total beta-N-acetylhexosaminidase, EC 3.2.1.30) was generally abnormal in rats with complete and strangulated obstruction.

Additional Keyphrases: diagnostic and prognostic markers; creatine kinase; maltase; alkaline phosphatase; urea N-phosphorus; calcium; bicarbonate; chloride; cholesterol; aminotransferases; veterinary chemistry; creatinine; emergency procedures; rats; reference intervals

Acute mesenteric vascular occlusion and obstruction of the bowel, with or without strangulation, are important medical emergencies. Surgical intervention within the first 24 h after onset is essential; further delay results in a sharp increase in mortality (1). As yet, the laboratory has not played an important role in the diagnosis of such patients. Sarr et al. (2), in a study of patients with intestinal strangulated obstruction, found that acidosis and leukocytosis, two common findings, lacked diagnostic efficiency. Other assays such as those for ALP, creatine kinase, phosphorus, and urea nitrogen have been used with limited success.8 ALP, although present in large amounts in the proximal small bowel, is an insensitive test. Animal studies revealed an increased ALP in experimental intestinal ischemia; however in humans, ALP is of limited value, possibly owing to its short biological half-life in blood. Serum urea nitrogen and phosphorus are significantly increased in patients with mesenteric vascular occlusion (3–6) and there appears to be a direct relationship between the serum phosphorus and the amount of bowel that is infarcted or strangulated (3). Unfortunately, tests for both urea nitrogen and phosphorus are nonspecific, values increasing in renal failure and in tissue breakdown of any cause.

Serum CK increases in mesenteric artery infarction (7–11); its isoenzyme CK-BB reportedly is significantly increased (12) or unchanged (9, 13). Animal studies revealed larger increases in serum CK in mesenteric artery infarction than in obstruction or strangulation of the small bowel (8, 10).

Hexosaminidase (Hex), its isoenzymes Hex A and Hex B, and maltase (EC 3.2.1.20; alpha-glucosidase; alpha-D-glucoside glucohydrolase) are present in substantial activities in the intestinal mucosa. Poison et al. (14) found an increase in the proportion of Hex B in serum from patients with mesenteric artery infarction; Lobe et al. (15) found similar changes in infants with necrotizing enterocolitis. In animals, there was a significant increase in Hex in serum within 2 h of experimentally produced ischemia (14). Maltase activities are low in amniotic fluid in fetuses with cystic fibrosis or those with imperforate anus (16).

Our goals were to evaluate the changes in a large group of serum analytes in a rat model of surgically created infarction of the superior mesenteric artery and in three types of intestinal obstruction. Which tests show the greatest changes from normal, and what are the kinetics of these changes? Because these conditions in humans are life threatening, we wanted to identify those laboratory procedures that show the greatest promise for use as diagnostic tests.

Materials and Methods

Animals

Male Wistar rats weighing from 300 to 325 g were obtained from Harlan, Inc., Indianapolis, IN 46229; the animals were acclimated to their surroundings for one week before use, and received water and Purina rat chow ad libitum. We assigned the rats to one of six groups as shown in Table 1, and collected arterial blood from each animal at the beginning of the study to establish reference intervals for the 26 tests that were performed.

Surgery

All animals. Anesthesia was induced in 10 min by placing the rat into an anesthesia jar containing cotton gauze soaked with diethyl ether; an anesthesia mask was placed over the head to maintain anesthesia during surgery. A 2 to 4 cm top-to-bottom, midabdominal incision was made 1 cm below the base of the xypoid process, exposing the small intestine. Following surgery, 10 000 units of penicillin G was instilled through the incision into the abdominal cavity, and a through-and-through continuous stitching with no. 00 silk suture was used to close the wound. The animals awoke fully from anesthesia 10 to 15 min after surgery.

Vascular occlusion. Acute mesenteric vascular occlusion was produced by doubly ligating the superior mesenteric
Table 1. Design of Intestinal Infarction and Obstruction Studies

<table>
<thead>
<tr>
<th>Procedure</th>
<th>No. animals</th>
<th>Blood-collection schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarction study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-operated controls</td>
<td>12</td>
<td>Two animals in each subgroup; blood drawn from a subgroup after surgery and at times shown in Figure 1a and b. Blood drawn as above.</td>
</tr>
<tr>
<td>Mesenteric vascular occlusion</td>
<td>45</td>
<td>Four to 10 animals in each subgroup. Blood drawn as above.</td>
</tr>
<tr>
<td>Obstruction study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-operated controls</td>
<td>15</td>
<td>After surgery and at times shown in Figure 2a and b.</td>
</tr>
<tr>
<td>Partial intestinal obstruction</td>
<td>12</td>
<td>After surgery and at times shown in Figure 2a.</td>
</tr>
<tr>
<td>Simple complete obstruction</td>
<td>12</td>
<td>After surgery and at times shown in Figure 3a.</td>
</tr>
<tr>
<td>Complete strangulated obstruction</td>
<td>6</td>
<td>After surgery and at times shown in Figure 3a.</td>
</tr>
</tbody>
</table>

artery at its origin from the abdominal aorta with no. 00 silk suture (17). The controls were treated the same way; however, the suture was placed around the artery and then removed without occluding the artery.

Partial obstruction. Partial intestinal obstruction was created at a position 4 cm proximal to the ileocecal valve by placing a 4 x 0.2 cm diameter stainless-steel rod alongside the intestine and then firmly tying no. 00 silk suture around the intestine and metal rod. The rod was then removed and the surgery was concluded.

Complete obstruction and obstruction with strangulation. Complete intestinal obstruction was created in the small intestine 4 cm proximal to the ileocecal valve with a double ligation with no. 00 silk suture. In other rats, we created the strangulated obstructed bowel model, using the same section of small intestine. However, a 5-cm loop of intestine was formed, and the suture was tied around the base of the loop and its mesentery. The control rats were operated upon in the same way, except that the intestine was not ligated. One group of rats served as controls for the three types of experimentally induced obstruction.

Blood Collection

Vascular-occlusion group. Blood was collected only once from a given rat by inducing ether anesthesia and catheterizing the abdominal aorta with a 21-gauge needle. Time zero was defined as the time the mesenteric artery was ligated. The presence or absence of intestinal infarction was noted during blood collection. With infarction, the intestine took on a dark red, dusky appearance. The rats were killed by exsanguination. About 8 mL of blood was collected, allowed to clot, centrifuged at 600 x g for 10 min, and stored at -70 °C if the assays were not carried out the same day.

Obstruction groups. The animals were anesthetized with ether as described above, and a heparinized microhematocrit capillary tube (no. B4417-2; American Scientific Products, McGaw Park, IL 60085) was gently inserted about 5 mm with a continuous twisting and turning motion into the border of the eye next to the lacrimal duct and then into the surpraoorbitaf artery. One to 1.5 mL of blood was drawn and processed as described above. With this procedure, multiple blood specimens could be drawn from a single animal.

Tests

Hex and Hex isoenzymes. Serum Hex, Hex A, and Hex B were determined with the method of O’Brien et al. (18) without modifications. As substrate, we used 4-methyl-umbelliferyl-N-acetyl-beta-D-glucosamine (no. M-2133; Sigma Chemical Co., St. Louis, MO 63178). Fresh reagent was prepared on the day it would be used. We used an Aminco-Bowman spectrophotofluorometer (American Instruments Co., Silver Springs, MD 20907) with an excitation wavelength of 365 nm and an emission wavelength of 450 nm.

Maltase. Serum maltase was assayed by the procedures of Dahlqvist (19) and Hansen and Schreyer (20), with minor modifications. Maltase cleaves maltose to glucose, and the hexokinase reaction (21) was used to measure the three sources of glucose: that formed by the action of maltase on maltose, the endogenous serum source, and any glucose in the maltose, i.e., the reagent blank.

Hansen and Schreyer (20) used a 116 mmol/L maleate buffer, pH 6.5, containing 150 mmol of NaCl per liter. We did the same in optimizing the concentration of maltose in the substrate. Pooled rat serum was tested with this buffer; maltose was present at 5 to 240 mmol/L. The reaction rate increased rapidly with increasing concentrations of maltose and became constant at a maltose concentration of about 100 mmol/L.

With a reagent containing, per liter, 180 mmol of maltose, 150 mmol of NaCl, and 100 mmol of maleic acid, we measured maltase activities in rat serum at pHs ranging from 3.5 to 8.5. Maltase activity was greatest at pH 6.5. The rate decreased by about 10% for each 0.5 pH unit away from 6.5. The final reaction conditions for all maltase assays were at the above reagent concentrations and pH 6.5. We found that rat serum lacks the other disaccharidases, sucrase and lactase. The substitution of sucrose or lactose for maltose in the reagent eliminated the reaction with serum. Maltase in a pooled specimen of rat serum was stable for at least eight weeks at -20 or -70 °C; we observed no trends in the activity with time, and eight measurements of the maltase activity at one-week intervals gave a mean (±SD) of 1249 (84) for specimens stored at -20 °C and 1261 (56) U/L for others stored at -70 °C.

CK isoenzymes. Serum CK isoenzymes were determined by electrophoresis on agarose as described elsewhere (22).

Other tests. For all other tests described here we used a SMA analyzer (Technicon Instruments, Tarrytown, NY 10591) with the contemporary and unmodified methods as available from Technicon.

Results and Discussion

Reference Ranges

Table 2 gives the central 95th percentile ranges of the 26 tests we performed on normal rats before any experimentation. Also shown here are the quality-control data collected during the course of the entire study. From the total and analytical variability, the between-individual biological variability of the different tests can be estimated.

Mesenteric Vascular Occlusion

The slopes of the concentrations (or activities) of the analytes vs time were determined for the test group and compared with that of the sham operated controls. Some of these data are shown in Figure 1a to d; there is a significant difference (P < 0.001) in the slopes for the test and control animals for the four analytes shown. For ALP, ALT, AST, CK-MM, CK-BB, LD, sodium, and uric acid, the data were much like those for CK. The remaining 13 tests showed trivial or no differences between the slopes for the test and control groups. For CK-MB and CK-BB, the values were...
significantly different 2 h after surgery. The P values for the
slope comparisons of the test and control animals are given
in Table 3; each slope for the test animals was compared
with a slope of zero and with that of the controls.

In the test rats, the serum urea nitrogen, uric acid,
creatinine, and inorganic phosphorus increased steadily
with time, probably reflecting necrosis of the small bowel
and release of these metabolites into the peripheral blood.
The findings also suggested renal dysfunction owing to a
decreased blood volume and reduced cardiac output. It is not
possible to say whether tissue necrosis or renal dysfunction
is the more important factor in increasing the concentra-
tions.

The data for CK, Figure 1c, are typical for the enzyme
changes we observed. Serum ALP, AST, ALT, and LD
increased steadily after surgery. Their increases with time
strongly suggest tissue destruction.

The serum bicarbonate decreased in both the control and
test animals, but more rapidly in the latter. None of the
other tests shown in Table 2 exhibited this pattern. Those
tests listed in Table 2 but not in Table 3 showed no
significant differences between the test and control rats. All
test results required several hours to become abnormal. In
the test rats, the "early warning" analytes—those for which
the results were significantly changed within 4 h after
surgery—were ALT, AST, and bicarbonate. Those analytes
for which results were consistently abnormal after 6 h in
the test rats were inorganic phosphorus, urea nitrogen, CK,
ALP, AST, ALT, LD, bicarbonate, uric acid, and calcium.

We conclude that ligation of the superior mesenteric
artery results in necrosis of the small bowel distal to the
artery and in release of metabolites, enzymes, and proteins.
Concomitant renal dysfunction probably occurred. No rats
survived for more than 10 h after the onset of vascular
occlusion. Necropsy revealed small-bowel necrosis in all the
test animals; the small bowel was distended, dusky in
appearance, and differed grossly from normal small intes-
tine. All the test animals experienced significant biochemical
disturbances owing to tissue hypoxia and necrosis.
Acidosis was probably ascribable to tissue hypoxia
and accumulation of lactic acid. The tests with greatest diagno-
tic efficiency for this purpose appear to be bicarbonate, urea
nitrogen, inorganic phosphorus, CK, and the aminotransfer-
ases. Our findings for Hex, Hex A, Hex B, and maltase were

<table>
<thead>
<tr>
<th>Test</th>
<th>Central 95% range,</th>
<th>(no. of</th>
<th>Mean control value</th>
<th>CV, %</th>
<th>(n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin</td>
<td>28–38 g/L (95)</td>
<td>40</td>
<td>1.2</td>
<td>(38)</td>
<td></td>
</tr>
<tr>
<td>ALP</td>
<td>106–234 U/L (95)</td>
<td>29</td>
<td>1.5</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>ALT</td>
<td>6–72 U/L (95)</td>
<td>26</td>
<td>8.8</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>AST</td>
<td>57–186 U/L (95)</td>
<td>24</td>
<td>5.1</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>18–27 mmol/L (95)</td>
<td>8.8</td>
<td>4.9</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>2.0–2.6 mmol/L (95)</td>
<td>28.8</td>
<td>2.3</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>Chloride</td>
<td>105–117 mmol/L (95)</td>
<td>2.0</td>
<td>1.8</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>510–910 mg/L (95)</td>
<td>92</td>
<td>1.4</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td>192–1884 U/L (91)</td>
<td>508</td>
<td>3.6</td>
<td>(36)</td>
<td></td>
</tr>
<tr>
<td>CK-BB</td>
<td>45.5–73.1% of total (10)</td>
<td>4.0</td>
<td>10.6</td>
<td>(64)</td>
<td></td>
</tr>
<tr>
<td>CK-MB</td>
<td>13.6–31.2% of total (10)</td>
<td>31.3</td>
<td>6.4</td>
<td>(64)</td>
<td></td>
</tr>
<tr>
<td>CK-MM</td>
<td>5.4–33.7% of total (10)</td>
<td>104.7</td>
<td>3.5</td>
<td>(64)</td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td>3–11 mg/L (95)</td>
<td>7</td>
<td>8.1</td>
<td>(37)</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>870–2560 mg/L (95)</td>
<td>37</td>
<td>2.3</td>
<td>(37)</td>
<td></td>
</tr>
<tr>
<td>Hex (total)</td>
<td>7.0–13.4 U/L (15)</td>
<td>11.7</td>
<td>5.1</td>
<td>(7)</td>
<td></td>
</tr>
<tr>
<td>Hex B</td>
<td>3.9–8.3 U/L (15)</td>
<td>4.4</td>
<td>6.3</td>
<td>(7)</td>
<td></td>
</tr>
<tr>
<td>Hex A</td>
<td>16–54% of total (15)</td>
<td>7</td>
<td>5.9</td>
<td>(38)</td>
<td></td>
</tr>
<tr>
<td>Inorganic P</td>
<td>54–765 mg/L (95)</td>
<td>364</td>
<td>3.1</td>
<td>(53)</td>
<td></td>
</tr>
<tr>
<td>LD</td>
<td>202–1701 U/L (91)</td>
<td>145</td>
<td>2.1</td>
<td>(38)</td>
<td></td>
</tr>
<tr>
<td>Malatase</td>
<td>731–1427 U/L (74)</td>
<td>1249</td>
<td>5.1</td>
<td>(53)</td>
<td></td>
</tr>
<tr>
<td>Potassium</td>
<td>3.6–6.0 mmol/L (95)</td>
<td>129</td>
<td>6.7</td>
<td>(18)</td>
<td></td>
</tr>
<tr>
<td>Sodium</td>
<td>136–152 mmol/L (95)</td>
<td>158</td>
<td>1.3</td>
<td>(37)</td>
<td></td>
</tr>
<tr>
<td>Total protein</td>
<td>50–64 g/L (95)</td>
<td>63</td>
<td>1.3</td>
<td>(42)</td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1200–1360 mg/L (93)</td>
<td>46</td>
<td>0.8</td>
<td>(42)</td>
<td></td>
</tr>
<tr>
<td>Urea nitrogen</td>
<td>80–260 mg/L (95)</td>
<td>469</td>
<td>4.1</td>
<td>(37)</td>
<td></td>
</tr>
<tr>
<td>Uric acid</td>
<td>8–27 mg/L (94)</td>
<td>47</td>
<td>1.9</td>
<td>(42)</td>
<td></td>
</tr>
</tbody>
</table>

*CLINICAL CHEMISTRY, Vol. 34, No. 2, 1988 283*
Table 3. *P* Values for the Infarction and Obstruction Groups*<sup>a</sup>

<table>
<thead>
<tr>
<th>Test</th>
<th>Infarction</th>
<th>Partial obstruction</th>
<th>Complete obstruction</th>
<th>Strangulated obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Vs 0</td>
<td>Vs cont.</td>
<td>Vs 0</td>
<td>Vs cont.</td>
</tr>
<tr>
<td>ALP</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>ALT</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>AST</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>CK</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>% Hex A</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Phos.</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Malatase</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Urea N</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Uric acid</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

<sup>a</sup>Slopes of analyte concentration or activity (*y*) vs time (*x*) compared with a slope of zero or with the slopes of the controls (cont.) NS, not significantly different; Phos., inorganic phosphorus. All tests on the control rats were NS as compared with a slope of zero.

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Disappointing. Results for these analytes did not show significant differences between the test and control groups at any time. They may be better diagnostic tools for chronic rather than acute intestinal injury.

**Partial Intestinal Obstruction**

Some of the data for the partial intestinal obstruction group are shown in Figures 2a to d. All show significant differences between the 12 test and five sham-operated control animals. Table 3 summarizes the statistical data. Three rats died after 168 h, three more after 288 h, and six survived for 720 h—i.e., until the end of the study. Our results for the various analytes can be grouped into three categories: significant changes occurring within the first three to seven days, changes present only after seven days, and no significant changes developing as compared with the controls.

Urea nitrogen, uric acid, CK, and AST were generally
Serum urea nitrogen and uric acid probably reflect early and acute tissue breakdown and (or) renal dysfunction, probably as a result of decreased plasma volume and decreased cardiac output in the nonsurviving rats. Why values for ALP, ALT, and inorganic phosphorus peaked at seven to 10 days is unclear. However, these changes probably reflect release of metabolites from the distended bowel. The biochemical sequelae, as observed in the peripheral blood, probably are different for necrosis of the small bowel as compared with chronic distention without necrosis.

The increase in serum maltase in the surviving test group animals may represent recovery of injured bowel rather than changes caused by necrosis or distention. Apparently the normal small bowel continually releases maltase into the peripheral blood, where it is cleared. Thus, in contrast to ALT, increases in serum maltase may reflect proliferation of normal small-bowel mucosa rather than destruction. Another possibility is increased release of maltase from the tissues because of food stagnation and bacterial overgrowth. The exact mechanism causing an increase in serum maltase is unknown. Declining serum maltase activities portended a poor prognosis.

Complete and Strangulated Obstruction

Results for serum urea nitrogen, inorganic phosphorus, ALT, and maltase are shown in Figure 3, a to d, respective-
ly. The statistical data are summarized in Table 3. Urea nitrogen and uric acid increased with time in the complete and strangulated-obstruction groups; the rate of increase for both assays was greater in the latter. The changes in ALT, ALP, and AST were very similar. The values increased with time in both groups, but faster and to higher activities in the strangulated-obstruction group.

Serum maltase showed general decreases in both test groups. This may reflect loss of functional bowel, with less maltase than normal entering the peripheral blood.

Body weight in the complete-obstruction group decreased on average by 27% of the starting body weight in 720 h, or about 1% per day. Those with strangulated obstruction lost about 8% of their starting body weight in 65 h, or 3% per day. Complete obstruction and the early phase of partial obstruction mimicked starvation. A slow decline in the serum cholesterol and glucose and an increase in triglycerides in these groups also suggested starvation. Complete and strangulated obstruction were more lethal than partial obstruction.

After surgery to create complete obstruction, two animals died after 200 h, three more after 250 h, an additional three after 300 h, and all were dead by 350 h post-obstruction. Strangulated obstruction was the most lethal form. For the six test rats in this group, two were dead after 40 h, one more after 60 h, and all by 70 h after surgery.

At necropsy, the small intestine was greatly distended and filled with fluid in the test rats with complete obstruction. The small intestine distal to the obstruction was empty and flaccid. The strangulated segment of bowel in the test animals with strangulated obstruction was similar in appearance to the small intestine in rats with mesenteric infarction, being dark and dusky in appearance.

Whether our findings in rats are applicable to the same condition in humans remains to be established, and the data may also be inapplicable to other mammalian species. Boyd (23) showed large between-species differences for the commonly performed serum-enzyme assays. We found that normal human serum contains little or no maltase activity, but we have found increased maltase activities in the serum of dogs with bacterial overgrowth of the small intestine and in horses with colic or torsion of the colon. The serum maltase test may have its greatest utility in the diagnosis of intestinal injury in animals.

Conclusions

Ligation of the intestinal blood supply significantly affected survival time in rats with mesenteric artery occlusion and strangulated obstruction. Mesenteric vascular occlusion eliminated the blood supply to the entire small intestine,
while strangulated obstruction eliminated the blood supply to only approximately 5 cm of small intestine. Partial and complete intestinal obstruction did not block the vascular supply to intestinal tissue. A maximum survival time of 9 to 10 h in rats with mesenteric-artery occlusion and 72 h in rats with strangulated obstruction reflected the amount of small intestine that was infarcted.

Results of biochemical analyses in rats with mesenteric artery occlusion more resembled the findings in animals with strangulated obstruction than findings in rats with partial and complete intestinal obstruction. Serum inorganic phosphorus increased in animals with mesenteric vascular occlusion and all three types of intestinal obstruction. Concentrations of inorganic phosphorus were greatest in rats with mesenteric vascular occlusion, but two animals with complete intestinal obstruction also showed very high values for phosphorus. Urea nitrogen concentrations increased most rapidly in the mesenteric artery occlusion group, followed, in order, by the groups with strangulated, complete, and partial intestinal obstruction. The highest urea nitrogen concentrations were recorded in rats with complete intestinal obstruction.

Uric acid concentrations were most consistently increased in the complete-obstruction group. The mesenteric artery occlusion and strangulated-obstruction groups showed some increase in uric acid, but not as much as in animals with complete intestinal obstruction. Apparently, significant increases in serum uric acid take several days to develop.

The largest increases in serum enzymes, particularly ALP, occurred in those rats with mesenteric artery occlusion. Animals with strangulated obstruction showed unchanged or low ALP activities in serum. Some of the rats with partial and complete intestinal obstruction demonstrated increased serum ALP activity after seven or more days of obstruction.

AST and ALT increased most in those animals with interruption of the intestinal vascular supply. Animals with partial and complete intestinal obstruction had much smaller increases in serum aminotransferase activities. In the partial and complete intestinal obstruction groups, serum ALT revealed a more nearly linear increase in activity over time than did AST. It appears that tissue necrosis must occur before the aminotransferases increase.

The largest increases in CK activity in serum were observed in rats with either mesenteric artery occlusion or strangulated obstruction. Blockage of intestinal blood flow, with concomitant tissue necrosis, caused the greatest increases in serum CK activities. Changes in CK were less dramatic and more erratic in those animals with partial and complete intestinal obstruction. Abnormal total Hex activities were not observed in any groups. The percent Hex A activity demonstrated decreasing values in all groups except the mesenteric artery occlusion group, where no change was seen. A seven- to 10-day period was required for the proportion of Hex A activity to become abnormally decreased in the partial- and complete-obstruction groups.

Maltase activity in serum changed significantly in all groups except the mesenteric artery occlusion group. Animals with partial, complete, and strangulated obstruction demonstrated decreasing maltase activity in serum before death. However, rats surviving the partial obstruction showed increasing serum maltase activity after an initial decline. Animals with partial intestinal obstruction required seven to 10 days, those with complete obstruction, 72 to 120 h, and animals with strangulated obstruction required 16 to 24 h before an abnormally low serum maltase was observed. The most lethal forms of intestinal obstruction were associated with the greatest and most rapid decline in serum maltase activities.

Mesenteric artery infarction and strangulated obstruction were the most lethal, complete obstruction was intermediate, and partial obstruction was the most benign. In rats, and probably in dogs and horses, the laboratory can play a role in the diagnosis of these conditions; however, multiple tests such as ALP ALT, AST, bicarbonate, inorganic phosphorus, maltase, and urea nitrogen must be performed over time. The trends and magnitudes of abnormalities of the laboratory values are the most useful in making a diagnosis.

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