Hemorrhagic Necrosis of the Gastrointestinal Tract and Its Relation to Cardiovascular Status

By SI-CHUN MING, M.D.

WILSON and Qualheim in 1954\(^1\) and Ming and Levitan in 1960\(^2\) described a hemorrhagic process diffusely involving the gastrointestinal mucosa. In 1959 Katz\(^3\) reported a similar condition in the duodenum. These authors stressed the prevalence of cardiac disease in their patients, and recently special emphasis has been placed on various aspects of the cardiac status.\(^4-6\)

Since a previous report from this hospital,\(^2\) 64 additional cases have been autopsied, bringing to 75 the total at the Beth Israel Hospital, Boston, over a period of 7 years. Although the etiology remains obscure, the condition is relatively frequent and the clinical and pathologic manifestations are reasonably distinctive. In spite of this, the condition remains relatively unknown and a correct diagnosis is rarely made, even after surgical exploration. In this report, therefore, an attempt will be made to review the clinical and pathologic manifestations based on an analysis of these cases and to discuss possible etiologic factors, particularly in relation to cardiovascular status.

Material and Method

The 75 cases that form the basis of this report were collected from 1,535 consecutive adult autopsies performed during the past 7 years. The pertinent clinical and pathologic data were reviewed with particular attention to conditions such as heart disease, infection, and shock, which might be responsible for the gastrointestinal disease.

Although most patients died with a variety of diseases, only those in whom the entity was fatal or closely related to the death of the patient were selected for analysis. For instance, hypertensive heart disease alone was considered significant in only 60 cases even though myocardial hypertrophy due to hypertension was present in 457 cases. Acute myocardial infarction, on the other hand, was considered fatal regardless of coexisting disease, and all such cases have been treated as one group.

The pathologic changes in the gastrointestinal tract were evaluated with regard to degree of inflammatory response, presence and extent of intravascular thrombosis, evidence of epithelial regeneration, and presence of necrosis in the muscular coat. The probable duration of the pathologic alterations was estimated and compared with the clinical history.

Results

Clinical Aspects

The incidence of this disease among the adult patients examined post mortem was 4.8 per cent. The ages ranged from 38 to 93 years. Only one case (1 per cent) occurred under the age of 40, 13 cases (17 per cent) were between 40 and 59, 12 cases (16 per cent) were between 60 and 69, and 49 cases (66 per cent) were aged 70 and older. The corresponding age incidence in the whole autopsy series was 4, 21, 27, and 48 per cent, respectively. There was no sex preference.

The clinical features are summarized in table 1. Shock was the most frequent symptom, occurring in 60 cases. It lasted for 1 day or less in 46 cases; the longest duration was 11 days. Vasopressor agents, usually metaraminol, were used to combat the shock in 33 patients but usually had only temporary or no beneficial effect. The shock eventually became irreversible in all 60 cases.

Thirty-eight patients had abdominal symptoms. Twenty-five had clinical evidence of

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Supported by Public Health Service Program Project Grant HE 06316, U. S. Public Health Service.
gastrointestinal bleeding, massive in 12. The most common symptom was abdominal pain, usually epigastric, and severe in seven cases. In two instances severe pain in the right upper quadrant led to a diagnosis of acute cholecystitis. Mild tenderness and spasm of the abdominal wall were frequently noted, but board-like rigidity was not observed. In 13 cases the bowel sounds were decreased, becoming absent terminally in eight. In only one instance were they increased. The duration of symptoms ranged from a few hours to 5 weeks; in half the cases they lasted 2 days or longer. Thirty of these 38 patients had shock, concomitant with the abdominal symptoms in 17.

Many patients had fever and leukocytosis, probably secondary to other underlying diseases rather than as a manifestation of the gastrointestinal condition.

Nine patients underwent surgical exploration of the abdomen. Three of these were found to have thrombosis of mesenteric arteries with infarction of a segment of the small bowel, which was resected. The remaining portions of the digestive tract in these three cases appeared normal at operation but showed hemorrhagic necrosis at autopsy. In six other patients no occlusion was found in the mesenteric vessels at surgery. Two of these were explored for massive rectal bleeding; in one, pinpoint hemorrhages were noted in the stomach and jejunum, and, in the other, blood found in the colon was thought to be originating in sigmoid diverticula. After resection of these areas only small areas of mucosal hemorrhage were found. In the third case a dark red segment of ileum was thought to be infarcted. The main superior mesenteric artery pulsed normally but the ileal branch did not; this branch was opened but was not occluded. The resected ileum showed typical hemorrhagic necrosis and the remaining digestive tract was involved similarly at autopsy. The fourth case had extensive hemorrhage and necrosis extending from upper jejunum to sigmoid colon and there was no arterial pulsation. At autopsy the superior mesenteric artery was diffusely calcified but widely patent. There was a thrombus in the inferior mesenteric artery, apparently the result of an abdominal perineal resection for a rectal carcinoma, performed 1 month earlier. In the two remaining cases segments of marked cyanosis alternated with pink areas throughout the intestine. Although

Table 1

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Cases</th>
<th>Per cent of all cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock</td>
<td>60</td>
<td>80</td>
</tr>
<tr>
<td>Abdominal symptoms</td>
<td>38</td>
<td>51</td>
</tr>
<tr>
<td>Gastrointestinal hemorrhage</td>
<td>25</td>
<td>33</td>
</tr>
<tr>
<td>Abdominal complaints</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>23</td>
<td>31</td>
</tr>
<tr>
<td>Distention</td>
<td>17</td>
<td>23</td>
</tr>
<tr>
<td>Vomiting</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>Decreased bowel sounds</td>
<td>13</td>
<td>17</td>
</tr>
<tr>
<td>Duration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Day or less</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>Longer than 1 day</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>Clinical diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cause of bleeding unknown</td>
<td>12</td>
<td>16</td>
</tr>
<tr>
<td>Mesenteric thromboembolism</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Intraabdominal infection</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Enteritis</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Ileus</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>None</td>
<td>49</td>
<td>66</td>
</tr>
</tbody>
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Table 2

Distribution of Disease in 75 Cases of Hemorrhagic Necrosis

<table>
<thead>
<tr>
<th></th>
<th>All cases</th>
<th></th>
<th>Severe cases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Per cent</td>
<td>Number</td>
<td>Per cent</td>
</tr>
<tr>
<td>1. Involvement of individual organs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stomach</td>
<td>45</td>
<td>60</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>Duodenum</td>
<td>34</td>
<td>45</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>Jejunum</td>
<td>51</td>
<td>68</td>
<td>25</td>
<td>33</td>
</tr>
<tr>
<td>Ileum</td>
<td>60</td>
<td>80</td>
<td>29</td>
<td>39</td>
</tr>
<tr>
<td>Colon</td>
<td>50</td>
<td>67</td>
<td>18</td>
<td>24</td>
</tr>
<tr>
<td>Rectum</td>
<td>23</td>
<td>31</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>2. Extent of disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stomach and small and large intestines</td>
<td>31</td>
<td>41</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>Stomach and small intestine</td>
<td>14</td>
<td>19</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Stomach and large intestine</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Small and large intestines</td>
<td>16</td>
<td>21</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Small intestine alone</td>
<td>9</td>
<td>12</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Large intestine alone</td>
<td>3</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3. Blood in the lumen</td>
<td>52</td>
<td>69</td>
<td>31</td>
<td>41</td>
</tr>
</tbody>
</table>

Figure 1

Gross appearance of hemorrhagic necrosis. Top: diffuse hemorrhage of jejunal mucosa and normal mesentery. Bottom: extensive but patchy involvement of colonic mucosa from another case.

The arteries pulsed normally, the changes were interpreted as infarction. At autopsy, the mesenteric arteries were widely patent, and extensive hemorrhagic necrosis was present.

Pathologic Aspects

Despite the varied nature of the associated disorders, the pathologic changes in the gastrointestinal tract were uniform, varying only in the degree of severity and extent of involvement. The distribution of the disease is listed in table 2.

Grossly (Fig. 1), the mucosa in the involved areas was dark red and there were patchy or confluent areas of hemorrhage, most prominent along the plicae circulares. In 18 cases there were shallow ulcerations with necrotic bases. The serosal aspect appeared purple or dark red partly due to the large amount of blood present within the lumen in 52 of the cases. This discoloration was sometimes very similar to that seen in infarction and the appearance could be differentiated externally only by the smoothness of the serosal surface and the normal-appearing mesenteric fat seen in hemorrhagic necrosis. Thrombosis of the major mesenteric vessels leading to the diseased segment was noted in one case only; in this instance a nonocclusive recent thrombus was present at the orifice of the superior mesenteric artery.

Microscopically, the changes were characteristic. There was diffuse hemorrhage in the mucosa with necrosis beginning at the surface and extending downward to involve the whole mucosa and occasionally the muscularis mucosa as well (Figs. 2 and 3). The blood vessels, especially the veins in the submucosa, were markedly dilated and there
were associated varying degrees of edema and later young collagenous tissue. A mild to moderate neutrophilic reaction was noted in only 30 cases, and usually accompanied superficial ulceration. Although necrotic cell debris mixed with extravasated blood and fibrin was commonly seen on the mucosal surface microscopically, gross membrane formation was rare. The muscularis propria was normal in 67 cases, focally necrotic in seven, and hemorrhagic in one. Uniform and extensive necrosis as seen in infarction due to mesenteric thrombosis was not observed. Thrombosis, usually in the dilated capillaries at the base of mucosa (fig. 4), was observed in 18, or 24 per cent of cases, usually accompanying the inflammation or ulceration. In four of these, thrombi were also found in the dilated submucosal veins (fig. 4). The thrombi were few and scattered in most of these cases, and in all cases the hemorrhage far exceeded the extent of thrombosis. Evidence of mucosal regeneration (fig. 5) was noted in 14, or 19 per cent of cases, and presented as pseudostratified glands with

**Figure 2**
Whole thickness of jejunum showing diffuse hemorrhage in the mucosa, venous dilatation in the submucosa, an intact muscularis propria and a normal serosa.

**Figure 3**
The mucosa of the same specimen shown in figure 2. There are diffuse hemorrhage and necrosis. Inflammation and thrombosis are absent.

**Figure 4**
Portion of ileum adjacent to an ulcerated area showing thrombosis in the mucosal capillaries and in a dilated submucosal vein. The arteries appear normal.

**Figure 5**
Regenerating mucosa of colon.
mitoses in the bases of the crypts of Lieberkühn.

On the basis of the microscopic appearance of the lesion, the duration of the pathologic alteration was rather short; in only 16 cases could the lesion have been present longer than 1 day. The estimated duration by microscopic appearance corresponded with the clinical duration in 21 of the 38 patients with abdominal symptoms, and with that of shock in 38 of the 60 cases with shock.

The peritoneal cavity contained no free fluid in 42 cases, clear fluid in 15, bloody fluid in 16, and purulent exudate in two.

**Associated Conditions**

Most patients died with a multiplicity of diseases; the most significant or fatal conditions are listed in table 3.

**Cardiovascular Status**

Although heart disease was considered to be the cause of death in only 56 per cent of the cases with hemorrhagic necrosis, significant cardiac pathology with failure was present in an additional 23 per cent. These incidences were much higher than those of the entire autopsy population, being 46 and 15 per cent, respectively. In 17 cases of the study group, 10 of which died of fatal thromboembolism and seven of severe infection, cardiac decompensation probably made a significant contribution to the development of the gastrointestinal lesion, even though the cardiac condition did not appear to be the immediate cause of death. Two patients received only nitroglycerin for severe angina, whereas all the remaining 57 cases with heart disease had been digitalized. In 20 patients there was cardiac arrhythmia.

Shock was present twice as commonly in the cases of the study group as in the total autopsy population. Similarly, hemorrhagic necrosis of the gastrointestinal tract occurred twice as commonly in patients with shock as in those without it. All but one of the cases in the study group had evidence of heart disease, shock, or both.

**Infection**

Significant infection was evident in 607 autopsies during this period. It was considered a cause of death in 25 per cent of the entire autopsy series and in 20 per cent of the study group. Associated shock was present in 12, or 80 per cent, of the study cases with fatal infection. Cultures of the infected organs

### Table 3

<table>
<thead>
<tr>
<th>Associated Conditions</th>
<th>Study group</th>
<th>All autopsies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Frequency(%)</td>
</tr>
<tr>
<td>Heart disease</td>
<td>59</td>
<td>79</td>
</tr>
<tr>
<td>Shock</td>
<td>60</td>
<td>80</td>
</tr>
<tr>
<td>Heart disease</td>
<td>74</td>
<td>99</td>
</tr>
<tr>
<td>Fatal diseases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart disease</td>
<td>42</td>
<td>56</td>
</tr>
<tr>
<td>Acute infarction</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>Infections</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Peritonitis</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Endocarditis</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Thromboembolism</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>Peripheral</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Mesenteric</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Other diseases</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100</td>
</tr>
</tbody>
</table>
in nine of these 15 cases revealed *Escherichia coli* in four, coagulase-positive staphylococci in two, mixed gram-negative bacilli and staphylococci in two, and enterococci in one. Cultures of the postmortem blood in 14 cases revealed no growth in four, gram-negative bacilli in six, staphylococci in three, and enterococci in one. Blood cultures were also performed in 48 additional cases; there was no growth in 18, gram-negative bacilli grew in 24, staphylococci in two, and mixed gram-negative and positive organisms in four, including clostridia in two. Cultures of the contents of bowel or peritoneal cavity were made in 16 cases; gram-negative bacilli were present in all, mixed with staphylococci in five, clostridia in one, and enterococci in one.

Other Conditions

Eleven of the study cases had fatal thromboembolism. Cardiac failure was present in 10 and shock in nine of these 11 cases.

Blood urea nitrogen was determined in 35 cases; it was elevated in 27 and over 100 mg. per cent in five. In only one case did uremia appear to be the only significant condition.

There was a coagulation defect of the blood in 11 patients, associated with hepatic dysfunction in two, and with afibrinogenemia following cardiac valvulotomy in another. The defects in the remainder were induced by either heparin or coumarin. None of the patients manifested abnormal bleeding tendency in other organs.

Discussion

As previously noted,2 the basic pathologic change in hemorrhagic necrosis consists of extensive hemorrhage in the mucosa together with noninflammatory necrosis of the superficial layer and marked vasodilatation, most prominent in the submucosa. Several additional observations brought to light in this study are worthy of mention, however. Patchy ischemic necrosis was also noted in the muscularis propria in the severe cases. Fibrin thrombi were sometimes found within the capillaries in the necrotic mucosa and, less commonly, in the dilated veins of the viable submucosa. Regenerative processes in the mucosa indicated that the condition was not always terminal. Finally, bloody peritoneal fluid was occasionally present in spite of the absence of serositis. These additional changes coincide with clinical features such as prolonged duration, absence of bowel sounds, and evidence of peritoneal irritation in some cases.

Although a pathologic distinction between this condition and bowel infarction due to vascular thrombosis is possible, cases similar to those reported here have been interpreted as infarction due to vasospasm.7,8 Six of the present cases had exploratory laparotomy. At operation, two cases revealed hemorrhage in the digestive tract, and the discoloration of the intestines in the remaining four cases was thought to be the result of infarction. In none of these cases was there evidence of a significant functional obliteration of the vessels at the time of surgery, and postmortem examination demonstrated no organic occlusion of the vessels leading to the diseased bowel.

All these observations clearly indicate that the pathogenesis of this disease lies in a disturbance of circulation in the small intramural vessels of the digestive tract. The only conceivable organic basis for such a disturbance is the presence of small fibrin thrombi, but these thrombi were found only in a minority of cases. Even when present, they were too limited in their distribution to be incriminated as a cause for the widespread hemorrhagic necrosis.

Three conditions dominate the clinical picture: cardiac dysfunction, severe infection, and shock.

Cardiac Status

Earlier reports1–7 have emphasized the predominance of cardiac disease in patients suffering from hemorrhagic necrosis of the gastrointestinal tract. The data in this report show an increased prevalence of heart disease in these patients as compared with that of the entire autopsy population of the hospital. In addition to the 56 per cent of patients who died of heart disease, cardiac decompensation was present in an additional 23 per cent who
died of such complications as massive thromboembolism or infection. Even though such terminal events seemed directly related to the development of the pathology in the digestive tract, the pre-existing circulatory malfunction probably played an equally important role.

Various aspects of heart disease have thus far been incriminated, including acute infarction, digitalization, aortic stenosis, congestive failure, arrhythmia, and cardiac surgery. All these conditions are capable of causing an increased vascular resistance with subsequent decrease of blood flow in the splanchic bed. One or a combination of these conditions was present in 79 per cent of the study cases and probably contributed significantly to the development of hemorrhagic necrosis of the bowel.

Infection

Twenty per cent of the patients with hemorrhagic necrosis had fatal infection, and 35 of the 65 patients on whom postmortem cultures were obtained had either gram-negative bacilli or coagulase-positive staphylococci in the blood. The toxins of these organisms have been shown to have a vasoconstricting effect on the mesenteric vessels and are capable of producing hemorrhage and necrosis of the intestine.

There have been many reports on so-called staphyloccocal enterocolitis in man, although the actual role of the organism has been in doubt. This form of pseudomembranous enterocolitis was brought to general attention in 1939 by Penner and Bernheim. It appears that membrane formation in such enterocolitis may have been over-emphasized, however, and the pathologic descriptions of some of these cases are similar to those of the cases reported here. In this regard Penner and Bernheim suggested the causative role of shock and Kay noted the frequent association of this condition with heart disease.

Intestinal lesions due to endotoxin, though commonly observed in animals have rarely been recorded in man. The cases reported by McKay and Wahle have been explained on the basis of a localized Shwartzman phenomenon with abundant fibrin thrombi in the small vessels, but in our cases, the extent of such thrombosis seems inadequate to produce the extensive hemorrhagic necrosis. Furthermore, while anticoagulants may prevent thrombosis due to endotoxin as well as the Shwartzman phenomenon, the coagulative defects described in some of the patients in this report failed to prevent the development of the intestinal lesions. The significance of a vasomotor factor, on the other hand, is suggested by our studies on the mechanism of the Shwartzman reaction and from this point of view the lesions of hemorrhagic necrosis may be akin to, although not necessarily identical with, the Shwartzman phenomenon. The absence of a positive blood culture in these patients in no way eliminates the possibility of an endotoxic etiology, since endotoxin produced by organisms among the regular inhabitants of the bowel can be absorbed into the circulation, and its damaging action is highly intensified during shock.

Shock

Shock was present in 80 per cent of the patients, irrespective of their underlying diseases, and hemorrhagic necrosis occurred twice as frequently in those with overt shock. Although shock had been thought to be responsible for enteric pathology as early as 1939, the intestinal manifestations have generally been considered mild. More recent experiments on shock produced by a variety of means, however, have shown its detrimental effect on the intestinal circulation to be manifested by a decrease in the hepatic and mesenteric blood flow accompanied by constriction of arteries, suppressed vasomotion of arterioles, sluggish capillary circulation, and stasis and dilatation of the venous channels. A sphincter mechanism in the hepatic venous circulation has been postulated to explain the diminished flow in the portal system in shock. Direct measurement of capillary blood flow in the ileal loops of the dog shows that the mucosal capillaries are most seriously affected, with a reduction to about one fourth of the normal flow. These observations fit well with the pathology of hemor-
hemorrhagic necrosis which is most severe in the mucosa and is accompanied by marked venous dilatation.

It has also been shown that blood sludging alone is sufficient to cause tissue necrosis.\(^{52}\) Such a mechanism may be responsible for intestinal necrosis in the presence of an excessively sluggish blood flow without postulating a direct action of endotoxin, thrombosis, or a spastic occlusion of the arterial tree.

Whatever the precipitating cause, hemorrhagic necrosis of the gastrointestinal tract may be considered to represent a conglomeration of microinfarcts secondary to local derangement of the microcirculation within the tissue. Such derangement in the splanchnic system appears to be the basic physiologic response of the body to the three factors mentioned, and may be viewed as an integral part, not necessarily the result, of the complex phenomenon known as shock. It is possible that it may also occur in seriously ill patients even in the absence of overt clinical shock.

Since vasospasm in the splanchnic bed plays a major role in this defensive response of the body and is at least partly responsible for the gastrointestinal complication, it is not surprising that vasopressor agents fail to alleviate either the shock or the intestinal condition. By further increasing the vascular resistance, they may worsen the circulatory status and thereby aggravate the intestinal pathology. The support for such a possibility may be found in the experimental production of intestinal necrosis by the intraperitoneal injection of epinephrine\(^ {29}\) and in the reports of bowel infarction in patients with pheochromocytoma.\(^ {33,\,34}\) Furthermore, the role of the autonomic and central nervous systems in shock and endotoxemia has also been amply shown by experiments in which both death and hemorrhagic lesions are reduced when the influence of the nervous system has been eliminated either surgically or by anticholinergic agents prior to the induction of shock.\(^ {35-37}\) It is also relevant to note that circulating norepinephrine may by itself induce myocardial damage and arrhythmia,\(^ {29}\) which will further increase the vascular resistance.\(^ {9,\,14}\) Whether vasodilators will have a beneficial effect on hemorrhagic necrosis of the bowel in man, however, remains to be determined.

**Summary**

Seventy-five cases of hemorrhagic necrosis of the gastrointestinal tract were analyzed and compared with the entire autopsy population as control. The disease occurred most frequently in association with shock, which was present in 80 per cent of the cases with the gastrointestinal lesions.

The incidence of heart disease was higher in the cases of hemorrhagic necrosis than in the control group. The role of cardiac dysfunction in the etiology of hemorrhagic necrosis is emphasized as are the roles played by the bacterial toxins and shock. All three conditions have been shown to result in marked reduction of the intestinal blood flow and venous stasis.

Hemorrhagic necrosis of the gastrointestinal tract probably represents a conglomeration of microinfarcts of the mucosa secondary to markedly decreased local blood flow. Vasopressor agents have produced no beneficial effect; the effect of vasodilators remains to be determined.

**References**


HEMORRHAGIC NECROSIS


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Circulation. 1965;32:332-341
doi: 10.1161/01.CIR.32.3.332
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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