GASTROINTESTINAL BLEEDING
Diagnosis and Management

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SERIES PREFACE

During the past decade remarkable progress has been made in our understanding of many basic physiological processes related to liver and gastrointestinal tract functions. Much of this information has led to significant improvements in our understanding of clinical diseases that alter normal hepatic and intestinal function and in the therapy of these diseases. Innumerable examples can be cited. For instance, the application of basic principles of physical chemistry has clarified considerably the manner in which cholesterol is solubilized in bile. Related studies have identified the causes of cholesterol gallstones in several large groups of patients, and specific forms of therapy for the prevention or the dissolution of such stones are now available. Other experimental work that relies heavily on basic techniques of immunology and electron microscopy has identified specific infectious agents affecting liver function. These studies, in turn, have provided considerable insight into the different clinical syndromes included under the general heading of viral hepatitis, raising the possibility that effective immunization against these organisms may soon be available. Equally impressive advances have been made in our understanding of the control of gastric secretion and peptic ulcer disease, in the causes of intestinal malabsorption, and in radiographic and endoscopic methods for examining the liver and gastrointestinal tract.

This explosion of knowledge in gastroenterology poses a particularly difficult problem for those interested in the dissemination of new medical information to students, house officers and medical practitioners. Often advances have come so quickly that the information presented in standard textbooks is outdated before the books become available. Also, it is difficult to revise such texts rapidly because of the large number of authors involved and the long production time necessary for these books. Finally, the space available to authors for extensively reviewing both the basic physiological concepts and their clinical implications is limited in most texts and in more rapidly published medical journals.

This series of volumes published under the general title "Clinical Gastroenterology Monographs" was conceived and designed to overcome many of these difficulties and to bring to the medical practitioner the most current information on the pathophysiology and treatment of major areas of disease affecting the liver and gastrointestinal tract. Each volume covers an important group of related disorders and is sufficiently long to allow for extensive discussion of their basic pathophysiological, clinical, and therapeutic aspects. New volumes will appear regularly, and a special effort will be made to identify areas for inclusion in the series in which there is a rapidly expanding body of information relevant to the

care of patients with a particular gastrointestinal disorder. Existing volumes will be updated and republished frequently where continued advances in information justify such rapid revision.

It is hoped that this series will provide a continuously evolving and current reference source for the broad spectrum of physicians who deal with patients with diseases of the liver and gastrointestinal tract.

John M. Dietschy, M.D.

PREFACE

The patient presenting with gastrointestinal hemorrhage poses a difficult diagnostic and management problem for the physician. During the past 15 years major advances have been made in these fields. The developments in both fiberoptic endoscopy and selective angiography have provided the physician with the means to make a precise and specific diagnosis as to the origin of bleeding. A greater understanding of the physiologic and biochemical consequences of acute hemorrhage has created a basis for better management of patients with major hemorrhage. New surgical approaches have also been developed. These diagnostic and treatment modalities and pathophysiological insights have now been available long enough to permit an assessment of their contribution. On the basis of such an evaluation, we offer a logical and practical approach to the problem.

A problem-oriented format is utilized. We indicate where decisions are based on hard data and where they are made on the basis of clinical impressions. Where appropriate, we review the physiological and biochemical changes that are important to an understanding of the clinical disorder or its management.

Our aim is to present important basic concepts with particular emphasis on the more common and major diseases. We do not attempt to be all-inclusive; readers should refer to specific texts for detailed descriptions of disease entities. Finally, and perhaps most importantly, the book presents the views of an internist, gastroenterologist, and surgeon, a team that we believe is crucial for the optimal care of patients with gastrointestinal bleeding.

JOHN A. BALINT, M.D.

Albany, New York
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GENERAL CONSIDERATIONS

The proper management of the patient with gastrointestinal bleeding involves a series of important decisions, each based on the accurate clinical assessment of the problem at hand. Many of these steps are based on sound physiologic or epidemiologic principles. Others, however, are based more on art, or, if you will, dogma, than on hard scientific fact. Thus the management of the patient presenting with bleeding from the gastrointestinal tract involves both the science and the art of medicine. Our purpose in this book is to present our view of the principles that should guide the physician in caring for such a patient. We indicate where these principles are based on hard data and also where they are derived from clinical impressions. Whenever possible, we present the approach to patients with gastrointestinal bleeding in a problem-solving format and, when appropriate, indicate alternative positions that have been recommended.

The year 1935 marked a major turning point in the management of patients with gastrointestinal hemorrhage. In that year two reports appeared that laid the foundations for our present mode of treatment of these patients. Meulengracht (1) reported on his experience with 251 patients treated with a regimen of pureed diet, antacids, sedation, and oral iron replacement starting within 24 hours of admission to the hospital. There were only three deaths (1.5%). By contrast in a comparable group of 289 patients treated in a neighboring Copenhagen hospital with the old regime of ice chips only, the mortality was 7.9%. Most other investigators at that time using the old regime reported mortality rates of 11 to 25% (1). Meulengracht listed his reasons for trying diet therapy as follows:

1. Exhausted patients were likely to die.
2. Bleeding sometimes seemed to stop with feeding.
3. Ambulant patients with melena often stopped bleeding on their own.
4. It seemed to him unreasonable to starve patients who needed food *and* fluids.

This requirement for fluid and blood replacement presented a major problem until in the same year Marriott and Kekwick (2) reported on a method for the continuous drip infusion of blood. Intravenous infusion of electrolyte solutions had first been used 100 years earlier by Drs. O'Shaughnessy and Latta to treat the dehydration of cholera, but it had fallen into disrepute because of problems with infections. Blood transfusions had been used early in this century when blood

grouping was first established in 1901 by Landsteiner so that this form of therapy could be safely undertaken. However, until 1935 blood transfusions involved direct intravenous injection of freshly drawn blood. This limited the usefulness of transfusion. Marriott and Kekwick (2) described a method for controlled, prolonged infusion of large amounts of blood by a method that in its essentials is still in use today. They described their experience with 17 patients given between 2.6 and 6.6 liters of blood. Their description of the change in their first patient is impressive testimony to the benefits of this new treatment modality. "His demeanor changed from that of a dying man to an optimistic invalid." The patient had ulcerative colitis.

As illustrated by this case, gastrointestinal bleeding is always a potentially life threatening condition. When the patient presents with a massive hemorrhage and shows evidence of circulatory embarrassment, this threat is evident. However, even a relatively small bleeding episode may presage a later massive hemorrhage. Alternatively, even mild or occult bleeding, which is not in itself a threat to the patient's life, may be the presenting symptom or sign of a major illness, such as gastric carcinoma. Yet, again, a bleeding episode may complicate a relatively benign condition, such as acute peptic ulcer. Each of these general situations requires different approaches to therapy. Therefore, appropriate management decisions will depend on precise diagnosis as to the site and pathologic nature of the bleeding lesion, the magnitude of the hemorrhage, and the presence or absence of complicating conditions. Thus the problem is predictably greater in older patients than in younger individuals. It is only when all the necessary facts are assembled and carefully reviewed, that correct decisions about treatment can be made. Since gathering the necessary data will usually involve the internist, the endoscopist (gastroenterologist), and the radiologist, although treatment may well be surgical, a team approach is basic to good management, and it should be instituted at the earliest possible time.

DEFINITIONS

Gastrointestinal hemorrhage may be massive, overt, or occult, and may come from any part of the digestive tract. *Massive bleeding* may be defined quantitatively or functionally. The presence of signs of circulatory embarrassment indicates massive blood loss. These signs include hypotension, tachycardia, peripheral vasoconstriction, and oliguria. Alternatively, a patient requiring 2500 ml or more of blood replacement in the first 24 hours is considered as having had a massive hemorrhage. *Overt bleeding* is defined as the occurrence of hematemesis (i.e., the vomiting of blood), melena (i.e., the passage of black, tarry stools), or of bright red rectal bleeding, not requiring massive blood replacement, and not associated with signs of circulatory embarrassment. The presence of positive tests for occult blood in the stools with or without associated anemia is evidence of *occult gastrointestinal bleeding.*

SITE OF BLEEDING

Hemorrhage may originate from any part of the digestive tract. As a first step in diagnosis, it is helpful to determine from which general area of the gastrointestinal tract the bleeding is coming. Thus the upper gastrointestinal tract is taken to include the esophagus, stomach, and duodenum. *Upper gastrointestinal bleeding* usually pres-

ents with melena with or without hematemesis which may be massive. Bleeding from the upper digestive tract is often associated with symptoms of the causative lesion such as peptic ulcer, cirrhosis of the liver or esophagitis. *Lower gastrointestinal bleeding* usually presents with passage of red blood per rectum, since it originates in the large bowel. Such bleeding is rarely massive and may be otherwise asymptomatic, unless it is due to inflammatory bowel disease. *Bleeding from the small bowel* is uncommon compared with hemorrhage from the upper or lower gastrointestinal tract. The bleeding is rarely massive and is often occult. It is often unassociated with symptoms other than those due to blood loss.

ETIOLOGIC CONSIDERATIONS

Within each of these three areas of the gastrointestinal tract certain lesions are common causes of overt bleeding, but others rarely result in such events. Thus, acute superficial mucosal lesions are responsible for about 25% of upper gastrointestinal bleeds (3,4), while carcinoma of the stomach accounts for only about 2% (5). Armed with this type of data, examination can be directed specifically to elicit information relative to the likely cause of bleeding. This information is important not only in providing the basis for therapeutic decisions but also in indicating the special investigative procedures most likely to help in establishing the diagnosis. Furthermore, the preliminary diagnosis provides information on which to base the prognosis and the proper management.

MAJOR DECISIONS

The most important decision in any patient with gastrointestinal bleeding is whether treatment is to be medical or surgical. The importance of this decision is well illustrated by the results reported by Gordon-Taylor in the first Lettsomian Lecture to the Medical Society of London in 1946 (6). Even in those early days of the surgical treatment of bleeding peptic ulcer, he was asked to see about 20% of such patients during their admission. He reported that he was able to operate successfully, with a 5.5% mortality, if he was asked to see the patient early. When he was asked to see the patient late, and to operate as a desperate measure to save the patient's life, the mortality rate rose to 36%. The timing of the decision on whether a patient is to be treated medically or surgically is thus very important. This decision will depend on the answers to several questions:

    1. Is the lesion causing bleeding amenable to surgical management?

    2. Is the patient an acceptable surgical risk? The patient's age, clinical condition, and the presence of complicating associated diseases are major determinants of the answer to this question.

    3. What is the magnitude of the blood loss? Estimates of volume depletion depend on clinical judgement, supplemented when necessary by special investigation such as measurements of central venous pressure.

    4. Does the patient require intensive resuscitative measures? Stabilization of the patient's circulatory status is a prerequisite to establishing a precise diagnosis and to all other therapeutic undertakings.

The answers to these questions and the decisions based on them require a team approach by the internist, endoscopist, radiologist, and surgeon. The role of these members of the team have been significantly altered in recent years. The internist has been provided with the means to provide adequate fluid and blood replacement, and the surgeon has at his disposal a variety of surgical procedures developed during the past 20 years. The role of the endoscopist was dramatically changed by the advent of the fiberoptic instruments (7). In 1952 Avery Jones (8) felt that gastroscopy was no substitute for radiologic examination. However, that statement was based on experience with the semirigid instruments then available. The new fiberoptic instruments together with newer analgesic drugs, such as Diazepam, make for greater patient comfort and much better visualization of the esophagus, stomach, and duodenum in a single evaluation. Thus, endoscopy now offers complete evaluation of the upper gastrointestinal tract with the ability to make precise diagnoses (9). Radiologists have also been provided with a new diagnositc and, indeed, therapeutic, method by the advent of selective abdominal angiography (10). This procedure provides not only a method for localizing the site of bleeding but also a means for its control in selected instances. Thus we now have the means of making precise diagnoses and of controlling hemorrhage. As a result we gain time for the resuscitation of the patient before undertaking definitive surgical treatment of the problem if this is indicated.

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2

PRESENTATION AND CLINICAL DIAGNOSIS

In this chapter, we deal with the four major presentations in gastrointestinal bleeding (i.e., hematemesis, melena, red rectal bleeding, and occult bleeding) separately. Although the approaches to the patient with each of these presentations will overlap, there are differences that warrant separate discussion of each.
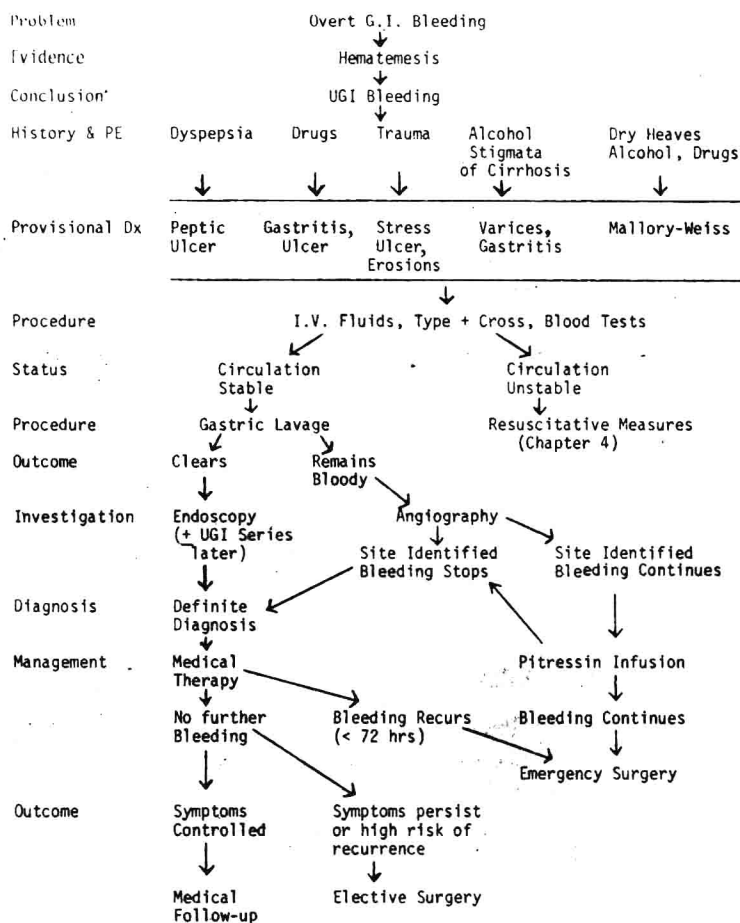
UPPER GASTROINTESTINAL BLEEDING

Jean Cruveilhier in his "Anatomie Pathologique du corps Humaine" (Paris, 1830–42) first fully described peptic ulcer and presented a classic description of a young man who died of hemorrhage from a gastric ulcer in the antrum (1). The patient was a muscular young carpenter of 29 years with a long history of alcoholic excess. Five years earlier he had bled repeatedly but recovered. He then did well in the interval, although he returned to drinking. On April 15, 1830, burning epigastric pain developed. On the 30th he began to vomit blood and was admitted to the Charite. Here he was found to be in shock (small pulse, easily compressed and severe anemia) so that he could not be bled! Next day he was a little better, but on May 2 he had a massive further hematemesis and died. At autopsy a large, chronic ulcer was found with an artery in its base. This case history is indeed a classical presentation, and it illustrates many of the problems in clinical diagnosis and decision making that face the physician when he first sees a patient with hematemesis.

Hematemesis (Scheme 2-1)

In this patient the problem was presented as overt bleeding evidenced by hematemesis. As shown in Scheme 2-1, this presentation allows the conclusion that bleeding has occurred from the upper gastrointestinal tract, that is, from a point above the ligament of Treitz. While it is true that bleeding from this area of the gastrointestinal tract may not result in hematemesis, bleeding beyond the ligament of Treitz rarely will result in vomiting of blood. Cruveilhier's patient had a history of excessive alcohol intake, but also complained of burning epigastric pain. Thus, on historical grounds a diagnosis of esophageal varices, gastritis, or of peptic ulcer must be considered. However, more importantly, examination showed the patient to be in an unstable circulatory state. This finding mandates immediate steps to correct

Problem   Overt G.I. Bleeding
                                       ↓

Evidence   Hematemesis
                                         ↓

Conclusion*   UGI Bleeding
                                     ↓

History & PE	Dyspepsia	Drugs	Trauma	Alcohol Stigmata of Cirrhosis	Dry Heaves Alcohol, Drugs
	↓	↓	↓	↓	↓
Provisional Dx	Peptic Ulcer	Gastritis, Ulcer	Stress Ulcer, Erosions	Varices, Gastritis	Mallory-Weiss

                                ↓

Procedure   I.V. Fluids, Type + Cross, Blood Tests

Status   Circulation   Circulation
                  Stable   Unstable

Procedure   Gastric Lavage   Resuscitative Measures
                                         (Chapter 4)

Outcome   Clears   Remains
                       Bloody

Investigation   Endoscopy   Angiography
                  (+ UGI Series
                  later)   Site Identified   Site Identified
                                  Bleeding Stops   Bleeding Continues

Diagnosis   Definite
                  Diagnosis

Management   Medical   Pitressin Infusion
                  Therapy

                  No further   Bleeding Recurs   Bleeding Continues
                  Bleeding   (< 72 hrs)

                                         Emergency Surgery

Outcome   Symptoms   Symptoms persist
                  Controlled   or high risk of
                             recurrence

                  Medical   Elective Surgery
                  Follow-up

SCHEME 2-1

the volume deficit before further diagnostic evaluation is undertaken. The evaluation of circulatory status and the considerations involved in correction of this situation are discussed in Chapter 4.

Provisional clinical diagnosis

If the patient's circulatory status is stable, a full history and physical examination is undertaken. The history should include careful inquiry for symptoms suggesting peptic ulcer, previous gastric surgery, drug ingestion, alcoholic excess, recent trauma or infection, retching, blood disorders, and family history. The relative frequency of major causative lesions will vary somewhat from country to country. Table 2-1 has been constructed from data presented by Palmer based on his prospective study of 1400 patients over 23 years in the United States (2) and by Schiller, Truelove, and Williams from a study of 2149 patients studied in Oxford, England (3). In both series, duodenal ulcer was thought to be the source of bleeding in about 30% of the patients. Gastric ulcer was the bleeding site in 12% to 15% of

Table 2-1
Major Causes of Upper Gastrointestinal Bleeding

Disease	Cardinal Features	Age	Frequency (%)	
			(2)*	(3)*
Duodenal ulcer	Periodic bouts of pain			
Gastric ulcer	Relief from food or antacids	> 20	27.7	29.0
	Relief of pain with bleeding		12.6	15.1
Gastritis	Salicylates, steroids, alcohol, dyspepsia	Any age	12.0	—
Esophageal varices	Alcohol, previous liver disease, hepatomegaly, stigmata of cirrhosis	Any age	18.7	2.4
Mallory-Weiss	Dyspepsia, dry heaves, alcohol	Any age	5.7	—
Carcinoma of stomach	Anorexia, weight loss	> 40	—	2.2
Other			23.3	51.3†

*Based on data from references 2 and 3.
†Includes 26.2% undiagnosed sources of bleeding.

instances. Esophageal varices, secondary to hepatic cirrhosis, were diagnosed as causing bleeding in 18.7% of Palmer's patients (2), but only in 2.4% of the British series (3). This difference presumably reflects the different prevalence of alcoholic liver disease in the two countries. Mallory-Weiss syndrome (4), that is, esophageal mucosal tears, was reported to be the cause of bleeding in 5.7% of patients by Palmer (2), who used esophagogastroscopy regularly. This lesion was not noted by Schiller et al. (3) who did not use endoscopy frequently. Others have reported esophageal tears in some 10% of patients with upper gastrointestinal bleeding (5). It is important to note that carcinoma of the stomach, esophagus, or duodenum was found to account for only about 2% of bleeding episodes (2,3). The converse of this is also true, in that only about 2% of gastric carcinomas bleed overtly. Thus malignant lesions of the stomach are rare causes of major, overt bleeding. As we discuss later, such lesions commonly give rise to occult bleeding. Hence the major causes of hemorrhage listed in Table 2-1 account for about 80% of all cases. Another lesion that is being recognized with increasing frequency is the so-called stress ulcer (6,7,8,9). This lesion occurs in the stomach, and also in the duodenum. It develops in patients who have suffered massive trauma, cerebral injury, severe burns, or major infections and is a dreaded complication in these situations. It is most commonly encountered in hospitalized patients, with bleeding occurring 4 to 15 days after admission.

Peptic ulcer. Overt hemorrhage is a common complication of peptic ulcer, with an incidence of about 1% per annum, as reported in most series (10,11). Peptic ulcer has its peak prevalence rate in the fourth and fifth decade of life. Bleeding from this cause may be suspected on clinical grounds in a majority of instances. The patient often has a very helpful history of periodic indigestion and antacid ingestion. Typically, the dyspeptic symptoms, especially pain, increase for several days before

the onset of bleeding and promptly subside once hemorrhage has occurred. Sometimes, these events follow a period of stress. The bleeding episode may present as hematemesis, melena, or both.

There are no specific physical findings of peptic ulcer other than epigastric tenderness and rarely a distended stomach. Physical examination is important, however, in confirming the absence of physical findings of other causes of bleeding or of associated disease. Finally, the history and physical examination are most important in determining the circulatory status of the patient, as is described in the discussion that follows.

Gastritis.   This diagnosis should be suspected in patients who present with upper gastrointestinal bleeding following recent heavy ingestion of alcoholic beverages, salicylates, or perhaps adrenocorticosteroid medication, and may affect patients of all ages. The association of gastritis with alcohol abuse is well recognized. It is, however, difficult to differentiate this lesion as a cause of bleeding from that caused by esophageal varices on clinical grounds alone, and endoscopic confirmation will be needed. The association of salicylate ingestion with gastritis and, indeed, peptic ulcer and hemorrhage has become accepted in recent years (12,13). Since salicylates appear in many over-the-counter medications, inquiry for their use has to be rather searching, or the patient may well not recognize that a medication contains salicylates. As in the case of ulcer disease, the physical examination is crucial in evaluating the circulatory state of the patient and excluding other causes of bleeding.

Esophageal varices.   Hemorrhage from esophageal varices may vary from massive to mild and recurrent. There is usally a history of alcohol abuse or previous liver disease. In children, with the possibility of extrahepatic portal obstruction, the question of umbilical infection should be investigated. Physical examination should be directed to finding evidence of portal hypertension such as splenomegaly, dilated abdominal wall veins, and ascites. The liver is often enlarged, and there may be spider angiomata on the skin. Since, however, the spleen will contract in response to hemorrhage, splenomegaly may not be appreciated. In addition, the patient is often jaundiced. The physical findings in patients with esophageal bleeding not only help to suggest the diagnosis but also indicate whether the patient is an acceptable candidate for surgical therapy. Thus, the presence of marked jaundice, ascites, bruising, and evidence of hepatic encephalopathy make the patient a poor surgical risk.

Mallory-Weiss syndrome.   This condition, which may occur at any age, classically presents with a history of repeated retching or dry heaves followed by hematemesis (4). Alcohol abuse is a frequent precipitating factor. However, other causes of gastritis may also lead to esophageal mucosal tears. Usually there is no clear history of previous dyspepsia. Physical findings are unremarkable, other than those attributable to the hemorrhage.

Carcinoma of the stomach.   This condition, as indicated in Table 2.1, is a rare cause of massive gastrointestinal bleeding. However, ulcerating carcinomas may lead to major bleeding episodes. The patient is usually over 50 years of age, with a short history of dyspepsia that is often accompanied by anorexia and striking weight

loss. It should be remembered that giant ulcers (greater than 2.5 cm in diameter) are more often benign than malignant. Final diagnosis needs confirmation by endoscopy and by four-quadrant biopsy of the edges of the ulcer after hemorrhage has been arrested for several days.

Other causes of upper gastrointestinal bleeding. These are presented in Table 2-2. The so-called *stress ulcers* first described as an entity by Curling in 1842 are perhaps the most common in this category (6-9). These lesions develop in 2% to 3% of patients with massive trauma, sepsis, burns, or cerebral lesions (6-9). Most often bleeding from these ulcers, which are commonly multiple, occurs 2 to 15 days after hospitalization. This is a particularly grave situation, since it occurs in an already seriously ill patient. Beil et al (7) in 1962 reported an overall mortality of 71% in their 35 subjects. The outlook in this group of patients has been improved by the availability of intraarterial pitressin infusion.

Leaking aneurysms or from infected aortic grafts and mesenteric vascular occlusions. These are fortunately rare causes of gastrointestinal hemorrhage and tend to occur in older patients with vascular disease. Prompt diagnosis, however, gives the only hope of saving the patient's life. The diagnosis is often very difficult. A high index of suspicion is needed, especially in patients with evidence of arteriosclerotic cardiovascular disease, or cardiac arrhythmia. If there is a history of severe postprandial abdominal pain and weight loss preceding the bleeding episode, the level of suspicion should be even higher. The presence of abdominal or femoral arterial bruits, and a pulsatile mass in the abdomen are highly suggestive physical findings for aneurysm. Gastrointestinal bleeding in the face of a silent abdomen is very suggestive of mesenteric vascular occlusion, especially if a perforation can be ruled out.

Osler-Weber-Rendu syndrome (telengiectasia). This usually presents with a history of repeated bleeding, often occult, in a patient with no history of other sources of bleeding. The diagnosis can usually be established by careful examination of the mucous membranes for the telltale vascular lesions. It is important to examine the patient for these lesions after restoration of blood volume, since the telengiectases may not be apparent when the patient is first seen.

The other lesions listed with their cardinal features in Table 2-2 are even rarer. Many of these lesions can be readily recognized on careful examination of the patient as indicated. Some of them will be discussed further in relation to obscure gastrointestinal bleeding in Chapter 6.

Investigation

After a preliminary clinical diagnosis has been arrived at, a decision must be taken whether the patient is in a stable or unstable circulatory state. In the latter case, the patient will be treated as discussed in Chapter 4. If, however, the patient's status is stable, that is skin turgor is good, pulse pressure is 30 mm Hg or greater, diastolic pressure is >60 mm Hg and pulse rate not excessive, further investigation to ascertain the source of bleeding may be pursued. Before this is undertaken, an intravenous infusion of isotonic electrolyte solution should be started. At the same

Table 2-2
Less Common Causes of Upper Gastrointestinal Bleeding

Disease	Cardinal Features	Age
A. Diseases amenable to surgical treatment		
Stress ulcers and erosive gastritis	Follow 2 to 15 days after severe burns, surgery, trauma or brain injury, sepsis	Any age
Aortic aneurysm	Massive bleeding, but may have recurrent episodes	> 40
A-V malformations	Isolated episodes of x-ray negative hemorrhage	Any age
Mesenteric vascular occlusion	Acute abdominal catastrophy, bleeding with silent abdomen	> 40
Hemobilia	Biliary colic, jaundice, bleeding	Any age
Benign and malignant tumors of small bowel	Often have painless, recurrent bleeding May have cramping pain	Any age
B. Diseases requiring medical management		
Heredofamilial		
Osler-Weber-Rendu (Telengiectasia)	Often familial, history of nose bleeds recurrent bleeding; telengiectasia	Any age
Pseudoxanthoma elasticum	Tigroid (angioid) streaking of retina, Morocco leather skin, BP ↑	< 50
Ehlers-Danlos	Abnormal skin elasticity Hyperextensible joints	< 50
Hematologic diseases		
Hemophilia	History of bleeding disorder	
Von Willebrand's disease	Cutaneous or joint hemorrhages	Any age
Thrombocytopenia*	Purpura, splenomegaly	
Lymphoma*	Hematologic abnormalities, splenomegaly	
Disseminated intravascular coagulation	Often have liver disease or severe infection	Any age
Fibrinolysis	Abnormal clotting tests	
Drugs		
Anticoagulants Reserpine Salicylates	Absence of other causes, history of drug ingestion abnormal clotting tests	Any age

*Some forms of thrombocytopenia, and lymphomas if localized, may be amenable to surgical treatment.

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