SELECTED TECHNIQUES IN INTERVENTIONAL RADIOLOGY

by

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1 Goldthorne Avenue
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Apartado 26370—Cedro 512
Mexico 4, D.F., Mexico

Rua Coronel Cabrita, 8
Sao Cristovao Caixa Postal 21176
Rio de Janeiro, Brazil

9 Waltham Street
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PREFACE

During the past decade, enormous changes have occurred in the practice of diagnostic radiology. The development of computerized tomography and ultrasound technology have led to a proliferation of new imaging procedures. These improvements in noninvasive imaging have resulted in a reduction of some diagnostic angiographic procedures.

Angiographers have not been idle during these years, and the development of therapy using catheter techniques has expanded significantly. The extension of angiographic techniques to therapy has almost as many applications outside the circulation as within it, including the genitourinary, gastrointestinal and biliary systems. All these new approaches to diagnosis and therapy provide an opportunity for greater contact between the patient and the radiologist, which we view as a healthy development in our specialty. This monograph covers in detail some of the more common procedures in interventional radiology.

We wish to acknowledge in particular the manager of the cardiovascular diagnostic laboratory, Ms. Patricia Bader, and our chief cardiovascular radiology technologist, Ms. Charlene Campbell, for their support. Mr. Carter Huff, our chief cardiopulmonary technologist, and Ms. Barbara Lowery, our cardiovascular radiology nurse-in-charge, have also contributed to the success of interventional radiology at The Johns Hopkins Hospital. Patient care before and after the interventional radiology procedure has been improved substantially by our cardiovascular radiology physician assistants, Ms. Patricia Adams and Mr. Frank Wenham.

The illustrations throughout the text were drawn by Mr. Gary Lees and Mr. Timothy Hengst, and the artist's perception of interventional radiology was produced by Mr. Leon Schlossberg after watching several procedures. Photography was under the direction of Mr. Henry Hessels and Mr. William Ragsdale. We also appreciate the secretarial assistance of Mrs. Deloris Deinlein, Mrs. Henrietta McCall and Ms. Diane Rizer. Ms. Lisette Bralow and the staff of W. B. Saunders have been most helpful in the preparation of the text.

Finally, all of the technologists, students, residents and fellows are acknowledged for tolerating us during the learning phase of this new technology. We also owe a debt to our patients and their referring physicians who allowed us to pursue these new methods of therapy when the results were uncertain.

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Chapter 1

OVERVIEW

Successful interventional radiology depends on four components of each procedure: consultation, performance, interpretation and follow-up.

CONSULTATION. As in diagnostic angiography, the consultation between the referring physician and the interventional radiologist is always important and sometimes crucial. For example, indications for iliac balloon angioplasty may be quite straightforward with favorable anatomy and appropriate symptoms; indications for splenic embolization are less clear and the risk of complications is greater. In many instances, the referring physician has not yet decided with certainty about the need for a specific procedure and is seeking the interventional radiologist's opinion on the merits of the therapeutic intervention. Thus, it is imperative that we be particularly familiar with the underlying disease processes as well as with the reported experience with each procedure.

Consultation should always include a review of the patient's past radiologic studies. In patients with obstructive jaundice, it is important to review the previous ultrasound or computerized tomographic examination as part of the evaluation. A patient with a large pancreatic mass will need skinny needle biopsy as part of the biliary decompression, whereas the patient without an obvious pancreatic mass may need diagnostic angiography to determine resectability if a distal common duct obstruction is felt to be of neoplastic origin.

Finally, it is necessary to interview the patient, obtain a pertinent history and perform a physical examination. No procedure is free of complications, and by taking time to explain these to the patient, we establish a rapport that will allay some of the patient's fears during the procedure and also provide the patient with a realistic expectation of what can be accomplished. We believe an informed consent can be obtained without alarming the patient to the point of resisting therapy or developing a central nervous system–mediated reaction during the procedure.[1]

PERFORMANCE AND INTERPRETATION. Interventional radiology is not an exact science and it is still developing. During each procedure, two interventional radiologists are scrubbed; often a fresh hand or idea from an assistant will provide just the right twist, guidewire or catheter to get us by a difficult part of the procedure. We have to guard against letting our egos get in the way.

Since the patient is lightly sedated and under local anesthesia, every effort to maintain one's "cool" is required, even when the procedure is going poorly. Unprofessional behavior is simply not tolerated and can terrify or create hostility in the patient. William Osler defined imperturbability as "coolness and presence of mind under all circumstances," and this is an attribute that all of us practicing interventional radiology must attempt to develop.[2]

FOLLOW-UP CARE. The interventional radiologist sees the patient during the remainder of the hospital stay as often as necessary.[3] This may be two or three times daily during the management of a difficult gastrointestinal hemorrhage with vasopressin or once a day in the postangioplasty patient. Outpatient follow-up may entail repeated visits for exchanging or cleaning biliary stents or a semiannual visit to see the vascular surgeon and the interventional radiologist for follow-up Doppler pressure measurement after angioplasty.

We have developed a cardiovascular radiology "physician assistant" position, which is shared by two senior cardiovascular radiology technologists (CVRTs). The CVRTs have become a valuable part of the interventional radiology team. By virtue of their prior training and patient orientation, they are extremely effective in spending additional time with an anxious patient to explain a procedure. Our physician assistants

accompany all angioplasty, biliary and chemotherapy patients back to their rooms. They make sure that external drainage of bile is done or heparin is started in an orderly fashion. They provide lectures to the nursing staff on these newer procedures and the associated management problems. They accompany us on rounds, returning later to measure Doppler pressures or adjust heparin dosages. Finally, they give instructions to the patient and answer any questions the patient may have before discharge.

Record keeping is an important part of interventional radiology, and the CVRT physician assistant starts a folder on each patient during his or her inpatient stay. Duplicate x-ray reports, Doppler pressure records, bile culture analyses and similar data are placed in the folder. Duplicate notes from outpatient visits are always placed in this folder along with the chart. It is particularly important to refer to this abbreviated patient folder when the patient calls and needs care.

IMPACT OF INTERVENTIONAL RADIOLOGY ON DEPARTMENTS OF RADIOLOGY

Interventional radiology, like surgery, is a physician-intensive therapy. Procedures take from two to five hours, and the consultation and follow-up may add another two to three hours to the time spent with a patient. While the use of diagnostic angiography has declined during the past decade, the use of interventional radiology has increased, and the time required for an interventional radiology procedure is easily twice that required for a diagnostic angiogram.[4,5] The physical and mental stresses associated with interventional radiology are present to a lesser extent in diagnostic angiography, and there is an added radiation hazard associated with the long fluoroscopy times in interventional radiology.

The interventional radiologist and the radiology departmental chairman must come to grips with the issues of professional time spent with the patient and the intensity of the effort. This often translates into additional staff in interventional radiology along with proper cost accounting and education of third-party payers. These issues were addressed in a recent editorial.[6]

If the productivity of radiology departments is to be assessed only by the percentage increase in the number of examinations performed annually, a large part of the measure clearly will have been missed. With proper selection of procedures and appreciation of the physician-intensive examination, one might expect a relative stabilization or even a decline in the number of procedures but more professional time spent performing each examination or therapy.

One additional issue that needs discussion is who is responsible for interventional radiology procedures outside the vascular system. This is a much less important issue in private practice, but in academic institutions there are potential conflicts between radiologists practicing by organ systems — e.g., gastrointestinal (GI) and genitourinary (GU) systems — and those practicing by technique — ultrasound, computerized tomography, nuclear medicine and angiography. Who should perform genitourinary and biliary stenting: the GU or GI radiologist or the angiographer-turned-interventional-radiologist? This is an issue for which there is no easy answer. In many departments, it will depend on the training and patient commitment of the various sections. In large departments where there is equal interest, commitment, and training among the groups, an equitable sharing of procedures may be condoned as long as enough procedures are performed annually to maintain competence. In smaller departments, it is probably in the best interest of the patient that the physicians most experienced in special procedures take primary responsibility for interventional radiology outside the vascular system.

REFERENCES

1. Lalli AF: Contrast media reactions: data analysis and hypothesis. Radiology 134:1–12, 1980.
2. Osler W: *Aequanimitas*, 3rd Ed. P. Blakiston's Son & Co., Philadelphia, 1932.
3. Baum S: The radiologist intervenes. N Engl J Med 302:1141–1142, 1980.
4. Whalen JP: Caldwell lecture. Radiology of the abdomen: impact of new imaging methods. Am J Roentgenol 133:585–618, 1979.
5. Levin DC, Millan V, Widrich WC, et al: Impact of CT on cardiovascular radiology: implications for future manpower needs. Am J Roentgenol 135:200–203, 1980.
6. White RI Jr: Interventional radiology in the 1980s (editorial). Am J Roentgenol 134:859–860, 1980.

Chapter 2

GASTROINTESTINAL BLEEDING

HISTORICAL REVIEW

The first application of diagnostic angiography to the management of gastrointestinal bleeding was in 1959.[1] One year later, intraoperative arteriography was used for the localization of unexplained gastrointestinal bleeding.[2] Subsequently, in 1963, experimental studies established that bleeding at a rate of 0.5 ml/min could be visualized by selective arteriography.[3] In 1967, transcatheter arterial infusions of vasoconstrictive agents were used successfully for the reduction of portal pressure in experimental portal hypertension and subsequently, in 1968, for the management of bleeding from esophagogastric varices and portal hypertension in humans.[4, 5]

Initially, transcatheter vasoconstrictive therapy was used for those patients whose clinical condition precluded surgical intervention. However, it was realized that such therapy not only was advantageous for the initial management but also offered a definite alternative to surgery. In other cases, angiographic localization was essential to provide a "road map" for surgical resection.[6]

Intravenous injection of vasoconstrictors for the control of upper gastrointestinal hemorrhage, which had already been used in 1956, was associated with significant systemic side effects.[7-9] The theoretic rationale for intraarterial infusion was that the systemic side effects could be avoided. Thus, the first intraarterial infusion of vasoconstrictive agents was in the superior mesenteric artery (SMA) for the control of variceal bleeding.[10, 11] The success achieved by this method prompted its application to other sources of bleeding in the gastrointestinal tract.[12, 13] However, limitations of this form of therapy for intestinal bleeding were recognized early and led to the introduction of transcatheter embolization techniques.[14, 15]

Initially, intraarterial epinephrine was used alone or in combination with propranolol.[13, 16] The autoregulatory effect and reactive hyperemia following discontinuation of the epinephrine infusions was found to be disadvantageous. Vasopressin, on the other hand, did not demonstrate such effects and was shown to be a safe drug, even for long-term infusions.[10]

The intravenous administration of vasopressin at lower doses was rediscovered after continuous intravenous infusions were found to be equally effective (as intraarterial infusions) for the control of bleeding of capillary and venous etiology (e.g., hemorrhagic gastritis and variceal bleeding) and in certain cases of arterial bleeding from the colon.[12, 17, 18] Thus, pharmacologic management of arterial, capillary and variceal bleeding from the gastrointestinal tract with intraarterial and intravenous vasopressin became well established.[18-21]

VASOPRESSIN

Vasopressin (Pitressin) is a purified antidiuretic hormone preparation. A vasopressin injection contains 20 pressor units for every oxytocic unit per milliliter. It is assayed for pressor rather than oxytocic activity, but the antidiuretic activity for both types (Pitressin and oxytocin) is equivalent. Vasopressin tannate is a water-insoluble preparation for antidiuretic use. It is suspended in oil and should not be confused with the intravenous, water-soluble hormone preparation.

The pressor effects of the posterior pituitary extract on the circulatory system were first described by Oliver and Schäfer in 1895.[22] Vasopressin causes contraction of the smooth muscles of the vasculature, especially in the capillaries and venules.[23] The smooth muscles of larger vessels are

less responsive. The effect is direct upon the contractile elements of the vessels and is neither antagonized by adrenergic blocking agents nor prevented by vascular denervation. The arteriolar and capillary constriction is evidenced by blanching of the skin and by decreased limb, splanchnic and coronary blood flow.

In the gastrointestinal tract, vasopressin has a twofold effect. It constricts the smooth muscles of both the blood vessels and the intestinal wall. This latter action was the basis for administering posterior pituitary extract (5 to 10 units intramuscularly) to relieve intestinal paresis and distention.[23] Vasopressin has also been used prior to cholecystography and ultrasonography to eliminate bowel gas. Smooth muscle contraction of the bowel is elicited only by larger doses (5 to 20 units). Intestinal wall motility and peristaltic activity (rather than tonus) are increased, which often results in defecation by the patient following intraarterial infusion of vasopressin. The effect on the large intestine is greater than on the small intestine.

The cardiac effects of vasopressin are indirect, resulting from a decrease in coronary blood flow and alterations in vagal and sympathetic activity.[23] Blood pressure elevation and symptoms of myocardial ischemia are observed. Although large doses are necessary to induce coronary ischemia in the majority of patients, some patients with coronary insufficiency may experience anginal pain even after relatively small doses.

In most cases, intravenous and SMA infusions of vasopressin are equally effective in reducing portal venous pressure for the control of variceal bleeding.[24-28] Under experimental conditions, intraarterial infusion of vasopressin into the SMA causes a more abrupt decrease and, in addition, a reduction in the SMA flow that is 15 to 20 per cent more effective.[24, 26, 28, 29] This may be the reason that intraarterial vasopressin infusions are more successful than intravenous infusions for the control of nonvariceal gastrointestinal bleeding.[30]

Following administration of vasopressin, reduction in portal inflow results from vasoconstriction of the prehepatic splanchnic vessels. This occurs at the small artery-to-vein level and possibly in submucosal arteriovenous shunts.[31-33] However, reduction in portal blood flow may not be accompanied by a corresponding fall in portal pressure.[34] Thus, in some patients portal venous pressure drop may be minimal, probably reflecting direct transmission of arterial pressure through presinusoidal hepatic artery–portal vein communications.[35] In such patients, vasopressin therapy may still be effective in controlling variceal bleeding, possibly by constriction of submucosal veins and by the pinchcock effect on the transmural veins.[36]

Reduction in SMA blood flow during intravenous vasopressin infusion is dose-related.[29, 37] In experiments on dogs, the optimal dosage is 0.3 mU/kg/min, which is equivalent to 0.2 U/min in the adult human.[29, 37] A higher dosage does not offer any significant additional reduction in SMA flow.[29] Maximal reduction is seen 20 to 30 minutes after infusion is begun, and the onset of maximal flow reduction is also dose-dependent (i.e., more rapid with a higher dose).[29] Rebound increase in blood flow (about 15 to 30 per cent above the baseline value) has been observed after termination of SMA vasopressin infusion.[29] Therefore, it is essential that the vasopressin infusion not be terminated abruptly, but rather that the dose be gradually decreased.

The success of intravenous infusion therapy may be due in part to higher systemic levels of circulating vasopressin achieved by the intravenous infusion.[29, 30] With intraarterial infusions, on the other hand, there is a gradual increase in circulating vasopressin levels, which remain below those achieved by intravenous infusions.[29, 38]

The effects of vasopressin on blood vessels other than the mesenteric arteries are variable.[24, 37, 39] During celiac or hepatic artery infusion, hepatic blood flow decreases initially but later increases by about 45 to 50 per cent.[31, 37, 39, 40] SMA vasopressin infusions increase hepatic arterial blood flow by 96.5 per cent. A dose-related decrease in coronary blood flow occurs.[14] In one experimental study, there was a 25 per cent reduction of coronary artery blood flow at 0.2 U/min and a 35 per cent reduction at 0.4 U/min.[41] Similarly, the cardiac output falls around 26 per cent at 3.0 mU/kg/min (equivalent to an average adult infusion rate of 0.2 U/min).[37]

Peripheral vasoconstriction may manifest

with decreased dorsal pedis and posterior tibial pulses and decreased skin perfusion. Systemic blood pressure rises initially (about 20 to 60 mm Hg, systolic).

INTRAVENOUS VERSUS INTRAARTERIAL VASOPRESSIN. Clinical experience shows that mucosal, variceal and, occasionally, low-grade arterial (especially colonic) bleeding may respond to intravenous infusions of vasopressin.[18, 21] At present, intravenous vasopressin infusion for reduction of portal venous pressure is the treatment of choice for documented variceal bleeding.[24-28]

SMA infusion of vasopressin at 0.2 U/min abruptly reduces portal venous pressure by about 50 per cent.[5, 28] Intravenous vasopressin effects a gradual decrease to almost equivalent levels.[28] Therefore, in patients in whom systemic vasopressin is not successful in controlling documented variceal bleeding, intraarterial infusion into the SMA could bring about an additional 15 to 20 per cent reduction in SMA flow, and the abrupt reduction in portal venous pressure may thus control the bleeding.

Vasopressin infusion into the left gastric artery reduces blood flow to the stomach by 90 per cent, whereas a reduction of only 55 or 60 per cent is achieved by intravenous or celiac infusion, respectively.[42] Celiac artery infusion initially decreases blood flow to 62 per cent of the baseline value, but subsequently an escape phenomenon is apparent due to increase in hepatic blood flow, leading to re-establishment of the flow at 90 per cent of the baseline value.[16] Failure of celiac infusions to control gastric bleeding may be due to vasopressin infusion beyond the origin of the left gastric artery, so that the resultant reduction in left gastric blood flow resembles an intravenous rather than an intraarterial infusion. Thus, in patients with gastric bleeding not responding to intravenous or celiac infusions of vasopressin, the left gastric artery should be infused. Left gastric artery infusion may be more appropriate than a trial intravenous infusion (see Fig. 2–3).

Intraarterial or intravenous vasopressin infusion above 0.4 U/min does not significantly increase the therapeutic effect; on the contrary, it is associated with significant morbidity and therefore should not be used. Intravenous vasopressin should be administered only through a central venous catheter, because of the danger of skin vasoconstriction and sloughing associated with a peripheral infusion.

METHODS OF DIAGNOSIS

Endoscopy

Angiography should always be preceded by endoscopy, unless the bleeding is massive and endoscopic identification difficult or unlikely. Endoscopy will not only identify those patients in whom the bleeding source is easily accessible (pharynx, upper esophagus, anorectal area) but also facilitate the angiographic procedure by serving as a guide for selective catheterization.

In patients with upper gastrointestinal bleeding, the esophagus, stomach and duodenum should be evaluated by endoscopy. Endoscopy should also be performed in patients with a previous documented ulcer or other potential source of bleeding. In approximately 40 per cent of affected patients, acute upper gastrointestinal bleeding may be from an unsuspected or hitherto undiagnosed lesion.[43]

In patients with lower gastrointestinal bleeding, endoscopy is less helpful. Nevertheless, it should be performed to evaluate the rectosigmoid region. A source of bleeding in the distal rectum will often be overlooked unless this region is specifically evaluated. Occasionally, endoscopy may be confusing if there is reflux of the blood from a rectal source into the sigmoid colon, where it may be mistaken for more proximal bleeding.

In addition to evaluation of the rectosigmoid area with a flexible instrument, the perianal region must be inspected. Commonly overlooked sources of bleeding in this region are rectal ulcers, lacerations and hemorrhoids. Hemorrhoids are occasionally the source of massive lower gastrointestinal bleeding in patients with portal hypertension.

Barium studies must be avoided during an acute episode of bleeding, since they not only delay definitive localization and therapy by endoscopy and arteriography but also do not contribute to the management of the acute episode. Thus, an ulcer detected by upper gastrointestinal barium examination may not be the source of bleeding. Further-

more, the presence of barium in the intestine can obscure arteriographic demonstration of the location of the bleeding.

Nasogastric Tube

A nasogastric tube should be placed in all patients with upper gastrointestinal bleeding. It serves to evaluate intensity of the bleeding and response to therapy following vasopressin infusion or embolization.

Even in some patients with lower gastrointestinal bleeding, a nasogastric tube should be inserted to evaluate gastroduodenal contents. Occasionally upper gastrointestinal bleeding with accelerated intestinal passage or a gastrocolic fistula may manifest as lower gastrointestinal bleeding. In such cases, the nasogastric aspirate may help to localize the bleeding.

Radionuclide Studies

In patients with intermittent or low-grade gastrointestinal bleeding from areas not easily accessible to the endoscopist (distal duodenum to ascending colon), arteriography may not be revealing if the bleeding is slow or if the study is done at a time when the patient is not bleeding actively. Such patients may be studied by nuclear medicine techniques using technetium-99m sulfur colloid or indium-111-labeled red blood cells.[44, 45] The techniques employed initially (e.g., technetium-99m sulfur colloid), like arteriography, were positive only if the patient was bleeding actively. Newer techniques using labeled red blood cells are more promising.

Angiography

For effective participation in the management of gastrointestinal bleeding, the angiography service should be available on an emergency basis. The study must be performed rapidly by an experienced angiographer and a specialized radiologic technologist.

Prior endoscopy helps to direct the angiographer to the vessel to be catheterized (e.g., left gastric artery for gastric bleeding, gastroduodenal artery for duodenal bleeding). If endoscopy is unrevealing, the initial effort is directed to other possible sources (e.g., if the esophagus and stomach appear normal by endoscopy, look for a duodenal or proximal small intestinal source).

Patient selection for angiography. In the majority of patients, gastrointestinal bleeding will stop on conservative therapy (bed rest, sedation, blood and fluid replacement, correction of coagulation deficit). Therefore, the number of patients requiring angiography is relatively small. When the conservative therapy fails to control gastrointestinal bleeding, arteriographic methods may be tried before surgery.

Angiographic demonstration of a bleeding source requires that the patient be bleeding actively at the time of arteriography. Although experimental studies have shown that bleeding at a rate of 0.5 ml/min (1.5 U/24 hrs) may be seen at angiography,[3, 46] clinical experience shows that in most patients more brisk bleeding is usually necessary for arteriographic demonstration (usually > 3 U/24 hrs).

Thus, arteriography is indicated in patients who require blood transfusions, who are hypotensive and hypovolemic because of bleeding and who present clinical evidence of continued bleeding, e.g., nasogastric aspirate (persistent red or pink return on gastric irrigation), falling hematocrit and continued blood requirement.

The number of blood transfusions required to stabilize the hematocrit does offer a clue to the severity of the bleeding. Thus, low-grade bleeding (requiring 1 to 3 U/24 hrs) is much less likely to be detected by angiography than is bleeding requiring more than 4 U/24 hrs. On the other hand, in patients with nonvariceal bleeding requiring more than 6 U/24 hrs, arteriography should not be delayed unnecessarily. With an increasing number of transfusions, coagulation defects will occur, making intravenous or intraarterial vasoconstrictive therapy ineffective.

Patients with intermittent, massive gastrointestinal bleeding (duodenal ulcer, diverticular bleeding) should be studied at the time of acute bleeding. This may not always be possible, however, since the acute episode may last for only a short while. If arteriography is performed and no bleeding source is localized, the catheter

should be removed prior to returning the patient to the ward, unless the selective catheter placement was extremely difficult. Usually, selective catheter placement requires only a few minutes; therefore, the potential risks (e.g., groin bleeding, thrombosis) and added expense (i.e., intensive care unit) are not warranted. Alternatively, radionuclide studies for the detection of intermittent gastrointestinal bleeding may be utilized.

Patients with chronic, low-grade bleeding (e.g., tumors, vascular malformations) should first undergo endoscopy and barium studies. Arteriography can be performed electively.

An orderly progression from the medical ward to the angiographic suite is desirable. If bleeding is not controlled by angiographic methods, the patient should proceed to surgery. It is in the best interests of the patient that an inordinate amount of time not be spent in the angiography suite. Similarly, returning the patient to the medical ward without successful intervention only delays surgery and makes the patient a poorer surgical risk.

ARTERIOGRAPHIC METHODS. In general, the femoral artery route is used. The axillary approach is used in patients who lack femoral pulses or have other contraindications to femoral artery puncture (e.g., new graft, infection, recent surgery). The axillary approach may also be useful in certain patients in whom the anatomy does not permit subselective catheterization.[47]

A catheter is placed into the artery supplying the source of bleeding, which was localized by endoscopy or suspected on the basis of the clinical presentation, as follows:

Distal esophagus — left gastric artery (if negative, left inferior phrenic artery);

Stomach — left gastric artery;

Duodenum — gastroduodenal artery;

Hemobilia — hepatic artery;

Small intestine — superior mesenteric artery (if negative, celiac artery);

Right hemicolon — superior mesenteric artery;

Left hemicolon/sigmoid colon — inferior mesenteric artery;

Anorectal area — inferior mesenteric artery, internal iliac artery.

The choice of catheter is determined by the vessel to be catheterized. Reinforced-wall catheters are necessary for selective catheterization, and both the Cobra and Sidewinder catheters may be used. The Cobra catheter is useful particularly for subselective catheterization (gastroduodenal, left hepatic artery). In addition, the loop catheter technique may be used for craniad-directed vessels (left gastric, inferior pancreaticoduodenal, right hepatic arising from the superior mesenteric or inferior phrenic arteries).[48]

Once the bleeding has been detected and the intraarterial vasopressin infusion has been successful in controlling it, the patient is returned to the intensive care unit for appropriate care of the arterial catheter and for infusion therapy (Table 2–1). The leg is restrained, if necessary, to prevent excessive motion and catheter dislodgment. Periodic evaluation is necessary as follows:

1. Examination of the groin for hematoma or bleeding from the arterial puncture site.
2. Monitoring of the femoral, dorsal pedis and posterior tibial artery pulses to evaluate vasoconstriction and possible complications (e.g., thrombus formation).
3. Monitoring of nasogastric aspirate or lavage to evaluate the success of treatment.
4. Monitoring of urine output to detect the antidiuretic hormone effects of vasopressin.

TABLE 2–1. Vasopressin Infusion Schedule

1. Localize bleeding by angiography.
2. Infuse vasopressin at 0.2 U/min for 20 to 30 minutes via constant infusion pump.
3. Repeat arteriogram.
4. If bleeding has stopped continue vasopressin infusion at 0.2 U/min for 12 to 24 hours.
5. If bleeding is not controlled by 0.2 U/min of vasopressin, infuse 0.4 U/min for 20 to 30 minutes and then repeat arteriogram.
6. If bleeding stops, continue vasopressin infusion over the next 24 to 48 hours as follows:
   a. At 0.4 U/min for the first 6 to 12 hours;
   b. At 0.3 U/min for the next 6 to 12 hours;
   c. At 0.2 U/min for another 6 to 12 hours; and finally
   d. At 0.1 U/min for the last 6 to 12 hours.
7. Then infuse D_5W or NS at 15 to 20 ml/hr for 4 to 6 hours to keep the catheter open, and observe the patient for signs of renewed bleeding (nasogastric aspirate, central venous pressure, hematocrit and transfusion requirement).
8. If bleeding has stopped, remove catheter. If bleeding persists at 0.4 U/min, consider transcatheter occlusion or surgery.

5. Cardiac monitoring for detection of arrhythmias and coronary ischemia.

Care of the arterial catheter is most appropriately performed in the intensive or intermediate care unit. Whenever possible, patients with arterial catheters should not be returned to the general ward.

APPROACH TO UPPER GASTROINTESTINAL BLEEDING

If endoscopy has demonstrated bleeding from the stomach or distal esophagus, the left gastric artery is studied. If no bleeding site is demonstrated, a celiac arteriogram is obtained to evaluate the anatomy and look for another source of bleeding. Other vessels that should be evaluated are the left inferior phrenic and left hepatic arteries, since both may give off several branches to the stomach. If a gastric antral source is detected by endoscopy but the bleeding is not demonstrated on the left gastric arteriogram, a common hepatic or gastroduodenal arteriogram, or both, should be obtained to evaluate antral branches arising from the gastroduodenal artery.

For duodenal bleeding, a gastroduodenal or common hepatic arteriogram is obtained. If the celiac artery is occluded or severely stenotic, an inferior pancreaticoduodenal arteriogram is obtained. If no bleeding source is demonstrated, a celiac arteriogram is obtained (to evaluate the dorsal pancreatic) or a superior mesenteric arteriogram is obtained (to look for aberrant supply).

If endoscopy can not be performed or has failed to localize a bleeding source, a celiac arteriogram is obtained first. If all branches (including left hepatic and left gastric arteries) are visualized adequately, the gastroduodenal artery is then injected. This is followed by a superior mesenteric arteriogram. If the left gastric artery does not opacify on the celiac arteriogram, it should be selectively catheterized. Often, during a celiac arteriogram, the catheter tip lies beyond the left gastric artery orifice; therefore, bleeding from the left gastric artery may not be demonstrated.[49]

If the source of bleeding is suspected to be distal to the duodenum, a superior mesenteric arteriogram is obtained first. If no bleeding is demonstrated, the celiac artery is injected to look for an aberrant blood supply or an unrecognized gastroduodenal source.

It is essential that the patients undergoing angiographic management have a nasogastric tube in place. This not only helps to evaluate the success of therapy but will prevent gastric overdistention and emesis.

APPROACH TO LOWER GASTROINTESTINAL BLEEDING

Patients with acute lower gastrointestinal bleeding should proceed to angiography following proctosigmoidoscopy. Endoscopy for lower gastrointestinal bleeding must include proctoscopy to exclude low rectal or perianal causes of the bleeding such as internal and external hemorrhoids, posttraumatic bleeding (as from trauma induced by a thermometer, Bardex balloon, or fecal disimpaction) and rectal ulcers.[50]

For acute colorectal bleeding, endoscopy can occasionally be confusing, since retrograde reflux of the blood may mimic a more proximal colonic source. Nevertheless, endoscopy is essential for evaluation of low rectal or perianal sources of acute lower gastrointestinal bleeding that would not necessitate arteriographic evaluation—e.g., hemorrhoids and fissures.

Because bleeding colonic diverticula are most commonly located in the ascending colon, a superior mesenteric arteriogram is obtained first, especially if the stool is maroon-colored, indicating a proximal source.[51] The splenic flexure and small intestine must be adequately visualized, especially when part of the intestine lies in the pelvis. Such a situation may require two SMA injections with high and low radiographic positioning. If a bleeding source is not identified on the superior mesenteric arteriogram, the inferior mesenteric artery (IMA) is injected. It is important to include the sigmoid colon and rectum in the arteriographic evaluation. If the IMA is occluded, an internal iliac arteriogram may be obtained to evaluate rectal bleeding.

Contrast medium in the bladder may obscure a rectal bleeding site unless a urinary catheter is present. In such a situation the result of the endoscopy is used as a guideline with respect to probable location (rectosigmoid or proximal) of the bleeding. If clearly no sigmoid or rectal bleeding source is identified or if the patient has a urinary

catheter, a superior mesenteric arteriogram is obtained first. Alternatively, an inferior mesenteric arteriogram should be obtained first. Occasionally the IMA is difficult to catheterize, owing to tortuosity of the aortoiliac vessels. In such cases one has to proceed to the SMA without wasting too much time at attempting to catheterize the IMA. After the SMA has been cleared, IMA catheterization may be attempted again.

Patients with chronic, intermittent gastrointestinal bleeding should undergo endoscopy and barium examination to exclude a tumor or inflammatory lesions as the source of the bleeding. Exploration laparotomy without a prior arteriographic search for an occult arteriovenous malformation or vascular tumor should not be performed. Such explorations have a very high incidence of "negative" or "indeterminate" findings.[52] Multiple explorations or repeated exploration can be avoided if angiography is used as a method of preoperative localization of the abnormality.[53] Intraoperative methylene blue or indigo carmine injection via a preoperatively placed subselective catheter facilitates resection of intestinal arteriovenous malformations.[54, 55]

VASOCONSTRICTIVE THERAPY FOR GASTROINTESTINAL BLEEDING

Esophagogastric Junction Bleeding (Fig. 2–1)

Bleeding from a Mallory-Weiss tear, the cause of upper gastrointestinal bleeding in 7 to 14 per cent of patients, is demonstrated as a linear collection of contrast extravasation in the region of the esophagogastric junction.[56, 57] If the tear is located anteriorly, extravasated contrast medium may collect in the gastric fundus or may outline the esophageal mucosa if it runs cephalad.[58] Occasionally, these may be difficult to differentiate from gastric erosions or ulcers at

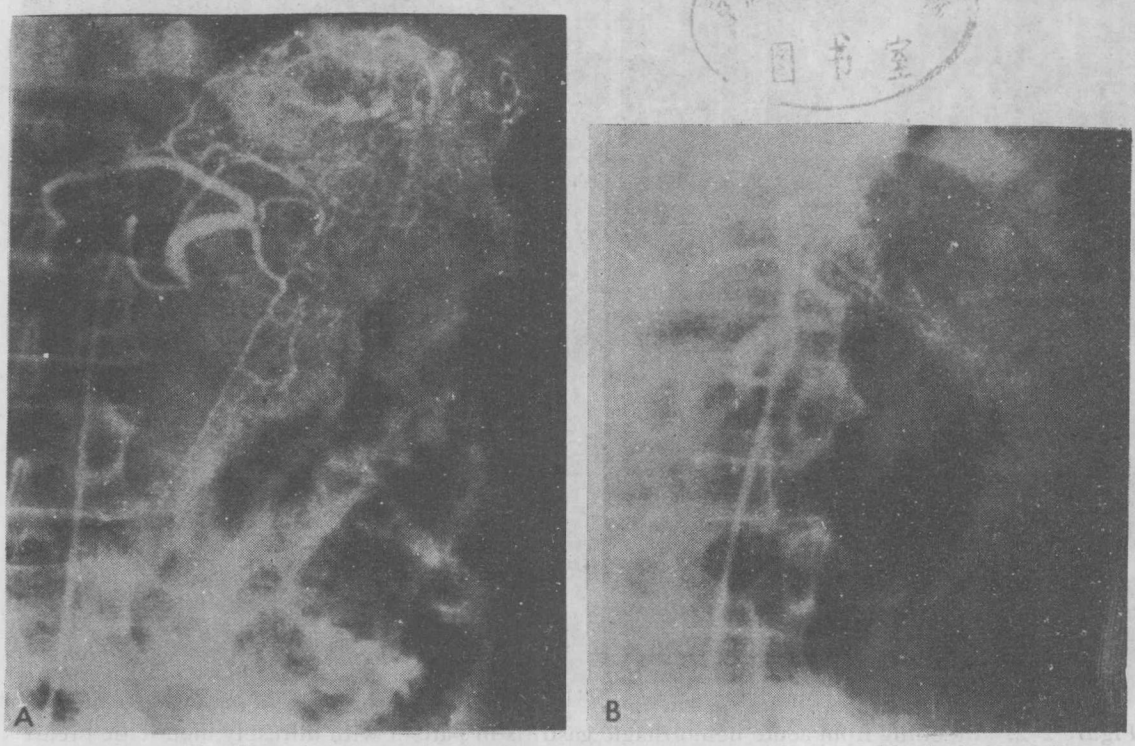

Figure 2–1. Bleeding from anterior Mallory-Weiss tears in a 41-year-old alcoholic following vomiting and hematemesis after a recent alcoholic binge. Endoscopy showed normal gastric mucosa and two distal esophageal mucosal tears. *A*, Left gastric arteriogram shows contrast extravasation from the gastroesophageal junction, which is seen in the gastric cardiac folds. *B*, Arteriogram following intraarterial administration of vasopressin shows control of the bleeding.

the esophagogastric junction. Bleeding from these tears is usually self-limited, and intraarterial vasopressin is the treatment of choice.[56, 57] In the occasional patient in whom intraarterial vasopressin does not control the bleeding, transcatheter embolization will be necessary.

Selective infusion into the artery supplying the bleeding source (left gastric, left inferior phrenic) is important for successful intraarterial vasopressin therapy. Success in controlling the bleeding with nonselective infusion (celiac) may be less assured, since the catheter tip may lie past the orifice of the artery from which the bleeding originated.

Other causes of arteriocapillary bleeding from the distal esophagus include esophagitis, esophageal ulcers, tumors and trauma (such as that induced by bougienage or a Sengstaken-Blakemore tube). Bleeding from esophagitis, ulcers and post-traumatic bleeding will often respond to intraarterial vasopressin.[17]

Gastric Mucosal Bleeding (Fig. 2–2)

Massive bleeding from acute hemorrhagic gastritis (superficial gastric erosions or petechial mucosal hemorrhage) may occur without an antecedent precipitating event as well as in critically ill or severely traumatized patients (e.g., following major surgery, burns, or alcohol or drug ingestion).[59-61] Acute gastritis is the cause of massive upper gastrointestinal bleeding in 17 to 27 per cent of patients.[61-63]

Hemorrhagic gastritis occurs predominantly in the gastric fundus.[61] Angiographically, two patterns are recognized: discrete contrast extravasation at one or several

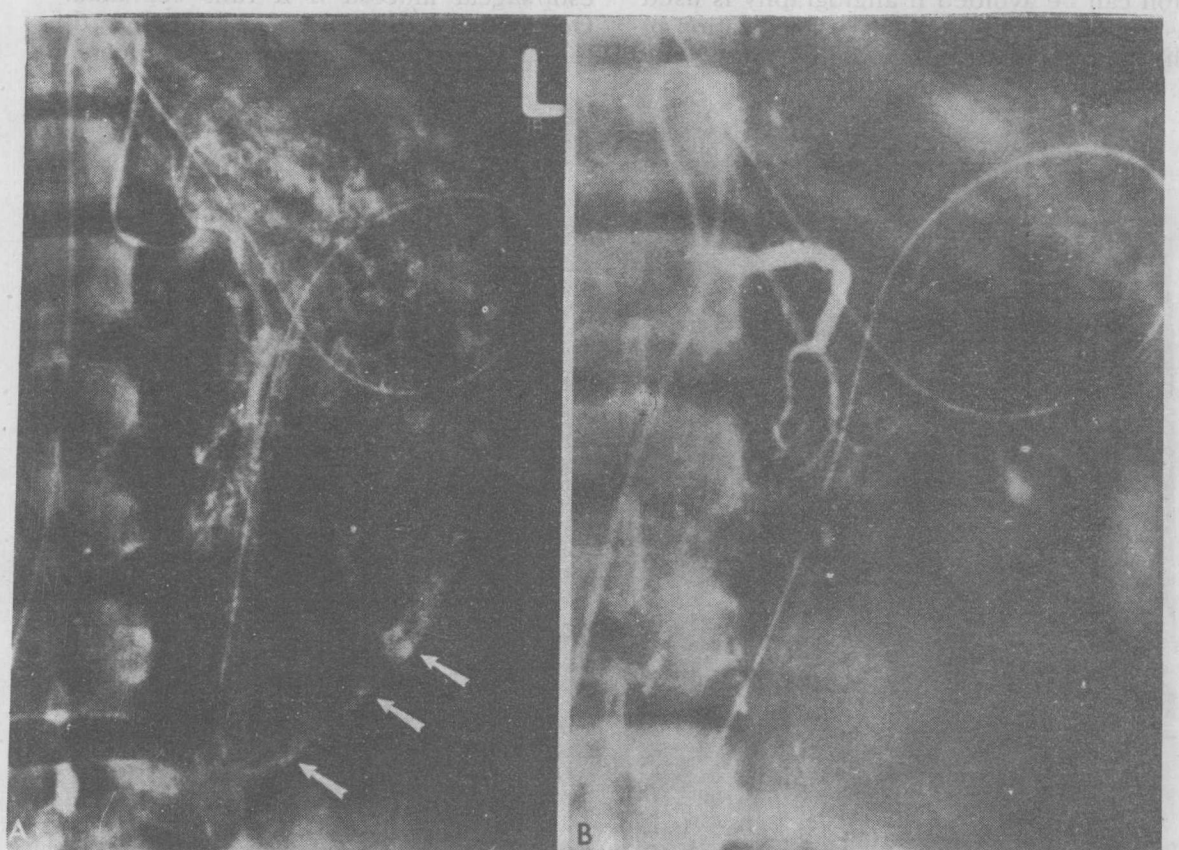

Figure 2–2. Bleeding from acute hemorrhagic gastritis in patient with diffusely metastatic breast carcinoma. *A*, Radiograph from late phase of the left gastric arteriogram shows diffuse gastric mucosal hyperemia. Areas of punctate contrast extravasation are seen from superficial erosions along the greater curvature (arrows). *B*, Following administration of vasopressin at 0.2 U/min for 20 minutes into the left gastric artery, there is excellent vasoconstriction and cessation of the bleeding.

points and diffuse hyperemia with or without extravasation. Surgical management for such patients, which involves subtotal gastrectomy (with or without vagotomy), may be unsuccessful in controlling the bleeding in up to 26 per cent of patients and carries an overall mortality rate of 50 per cent.[62, 64]

Conservative management will control bleeding from acute hemorrhagic gastritis in 80 to 85 per cent of patients.[16] The incidence of hemorrhagic gastritis has been reduced owing to the effectiveness of cimetidine and antacid prophylaxis in stressed patients.[63] In one series of patients who failed to respond to conservative management, intraarterial vasopressin successfully stopped the bleeding in 84 per cent (31 of 37 patients). Renewed bleeding was observed in 7 per cent (2 of 31 patients) 7 to 19 days later.[18, 21] Temporary control of bleeding can be achieved in the presence of coagulation defects by means of effective arteriolar and capillary vasoconstriction.[66] More permanent results are dependent upon an intact coagulation system.[16] Selective infusion into the left gastric artery is

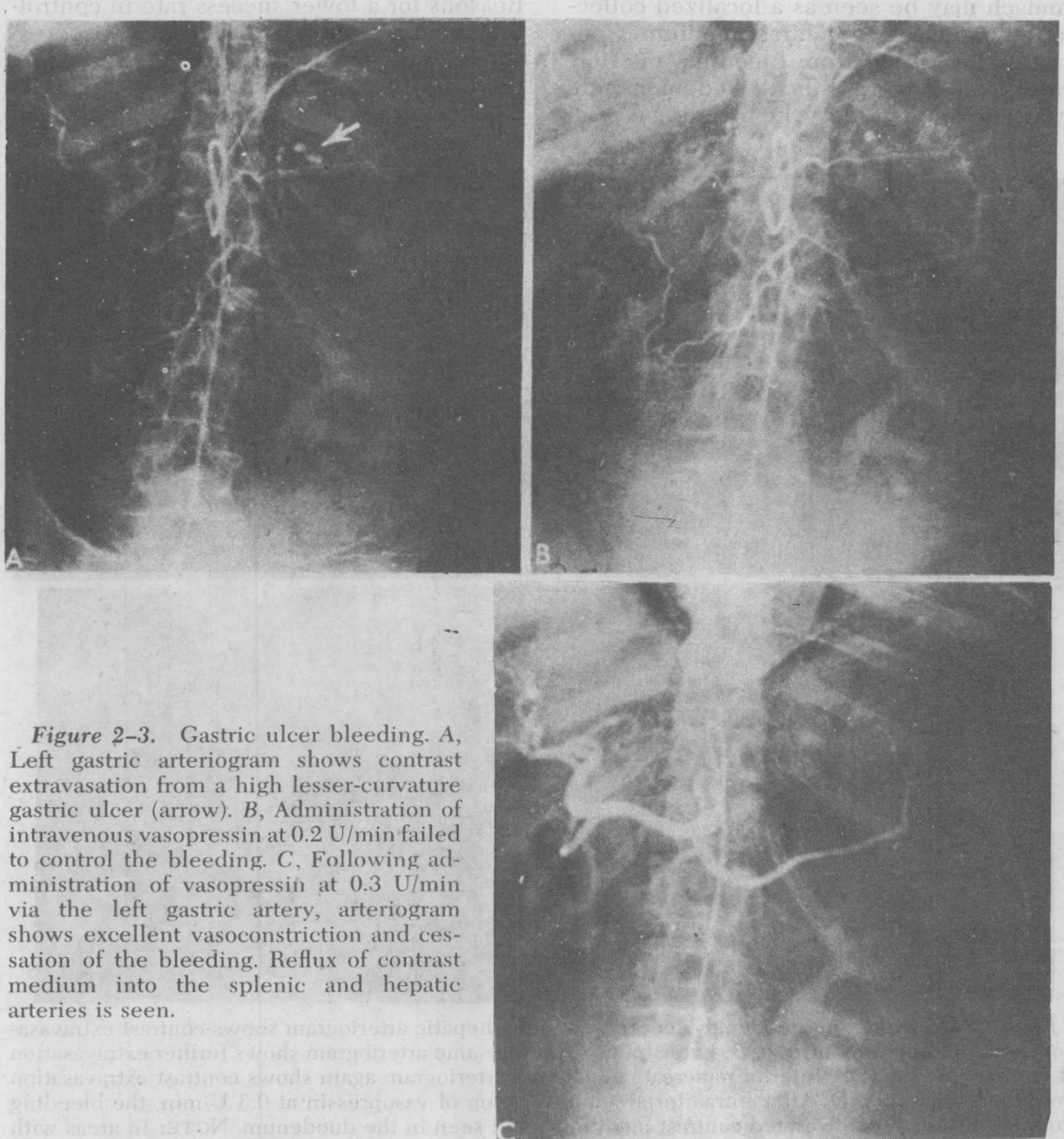

Figure 2–3. Gastric ulcer bleeding. *A*, Left gastric arteriogram shows contrast extravasation from a high lesser-curvature gastric ulcer (arrow). *B*, Administration of intravenous vasopressin at 0.2 U/min failed to control the bleeding. *C*, Following administration of vasopressin at 0.3 U/min via the left gastric artery, arteriogram shows excellent vasoconstriction and cessation of the bleeding. Reflux of contrast medium into the splenic and hepatic arteries is seen.

more successful in controlling the bleeding than are nonselective (celiac) infusions. On the other hand, intravenous or celiac artery vasopressin may stop the bleeding in some patients. If the left gastric artery anatomy is unfavorable for catheterization such an infusion should be tried before surgery is performed.

Gastric Ulcer Bleeding (Fig. 2–3)

On arteriography, bleeding from a gastric ulcer located on the posterior wall of the stomach may be seen as a localized collection of extravasated contrast medium. Contrast extravasation from bleeding anterior-wall ulcers is more difficult to demonstrate by arteriography unless the bleeding is relatively brisk,[58] in which case it may be demonstrated as curvilinear extravasation along the lesser curvature. Slow bleeding from anterior gastric wall ulcers may be demonstrated better when the stomach is not distended.

Treatment of a bleeding peptic ulcer is surgical for both control of bleeding and prevention of a recurrence. In poor surgical-risk patients or in patients in preparation for surgery, intraarterial vasopressin infusions may be used to stop the bleeding. The success rate for this form of treatment is between 50 per cent and 70 per cent.[17, 67, 68] Reasons for a lower success rate in controlling ulcer bleeding by infusion of vasopressin include the presence of inflammatory reaction at the ulcer base and bleeding from a large arterial branch. In some medical

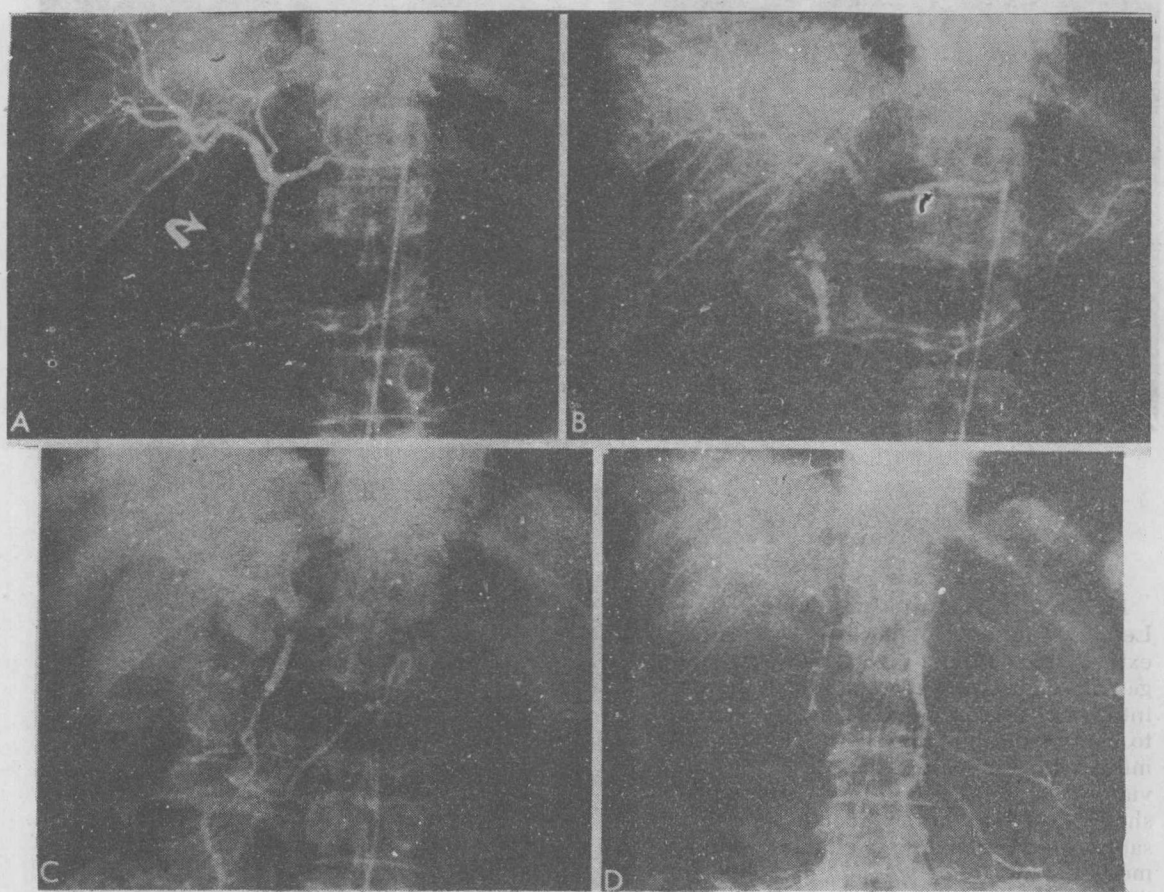

Figure 2–4. Bleeding duodenal ulcer. *A*, Common hepatic arteriogram shows contrast extravasation in the duodenum (arrow). *B*, Later frame from the same arteriogram shows further extravasation of contrast medium. *C*, Inferior pancreaticoduodenal arteriogram again shows contrast extravasation from the duodenum. *D*, After intraarterial administration of vasopressin at 0.3 U/min, the bleeding stopped. Residual extravasated contrast medium is still seen in the duodenum. NOTE: In areas with multiple sources of blood supply, all vessels should be evaluated for appropriate therapeutic infusion.