



Editorial

Surgical Treatment of Bleeding Duodenal Ulcer:

A Plea for Caution

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DURING recent years, many surgeons have championed the use of pyloroplasty with vagotomy for the early treatment of acute bleeding duodenal ulcer. There is little question that this procedure can effectively control immediate active hemorrhage and that it has the added advantage of obviating the problem of the difficult duodenal stump. The true test of the success of an operation for bleeding duodenal ulcer, however, is not only how effectively the procedure controls immediate life-threatening hemorrhage but also how effectively it protects the patient from recurrent bleeding in the early and remote postoperative periods. Our experience with several cases of recurrent and severe bleeding occurring within a few weeks after pyloroplasty and vagotomy has led to a painstaking reappraisal of the entire problem.

The grave complication of early recurrent bleeding following pyloroplasty and vagotomy occurred in patients with large, deep, posterior, penetrating duodenal ulcers that had eroded the gastroduodenal artery itself, or one of its major branches. These patients had active, serious hemorrhage during or just a few hours before the primary pyloroplasty and vagotomy. Non-absorbable sutures were placed deeply in the ulcer bed in the prescribed manner

and were sufficiently effective at operation to control completely all bleeding from the eroded artery. Vagotomy was judged to be complete by the usual means in the postoperative period in all instances. There is of course no way to prove unequivocally that all suture-ligations were performed properly or that vagotomies were in fact complete. Yet we have encountered postoperative massive bleeding from deep posterior ulcers following Billroth II gastrectomies in which the callous ulcer bed with sutured vessels was allowed to remain within the duodenal stump, even when the gastroduodenal artery was ligated and divided in undiseased tissue.

That such ulcers treated in this manner develop recurrent bleeding in the markedly alkaline medium of the duodenal stump provides some slight solace for our experiences with rebleeding after pyloroplasty and vagotomy. One is forced to consider the possibility that the common denominator in these cases of recurrent hemorrhage is that a *major* vessel, such as the gastroduodenal artery, has been eroded by and involved in the inflammatory reaction of the ulcer. It is not surprising that suture-ligation of this major vessel in diseased tissue is not successful, since only ligation or bypass in undiseased normal tis-

sue can prevent repeated hemorrhage, a lesson well learned long ago for many other areas of the body.

Recurrent bleeding from these large chronic ulcers has not occurred in our experience after complete exclusion of the ulcer bed from the gastro-intestinal tract.

Since the Civil War, it has been a principle of wound surgery that side-wall injuries in arteries do not stop bleeding as readily as transections because the artery cannot display its normal hemostatic mechanisms of constriction, retraction, and withdrawal. Recurrent bleeding is much more likely. It is important to emphasize that bleeding from the gastroduodenal artery is usually due to a side-wall erosion in the artery and is, therefore, unlikely to cease spontaneously regardless of the acidity of the medium.

A review of the reports favoring pyloroplasty and vagotomy for the management of bleeding duodenal ulcers has brought to light several interesting facts. A surprisingly large number of patients (almost half in one series) treated in this manner had at operation an *anterior* bleeding ulcer that was excised with the pyloroplasty, had no demonstrable ulcer, or simply had a scar without a definite crater. In many of the reported cases, the episode of bleeding that led to surgical intervention occurred several months before the actual operation; thus the procedure was being carried out to control the ulcer diathesis rather than to treat exsanguinating hemorrhage. A similar setting exists when an actively bleeding

anterior duodenal ulcer can be excised during the pyloroplasty, a circumstance strangely infrequent in our experience.

One must close any such discussion with a platitude that the anatomic procedure must be suited to the needs of the pathologic situation. Our point is merely that when treating patients with large callous, posterior, penetrating duodenal ulcers (and a side-wall opening in a large artery) it is not enough simply to regulate the acidity and assist pyloric emptying. Some sort of confrontation must be made with the artery itself. Actual circumferential dissection with removal of the ulcer and ligation of the artery appears to be essential, despite its hazards. The rest of the anatomic procedure must depend upon the patient's history and the surgeon's conviction and experience as to whether a standard subtotal resection, subtotal resection with vagotomy, antrectomy with vagotomy, or some other procedure is advisable. The surgeon performing such a difficult dissection, especially under emergency circumstances, should remind himself that this is where most of the mortality arises in duodenal ulcer surgery today. Drainage, duodenal decompression by catheter, and extreme conservatism in postoperative management are essential. Because of these hazards, any acutely bleeding patient should be given the benefit of an intelligent conservative trial for 24-48 hours with the hopes that operation can be performed in an elective phase.