Achalasia is a primary esophageal motility disorder of unknown etiology. It is a rare disorder (1/100,000) that affects males and females equally. Achalasia is characterized by progressive loss of peristalsis in the body of the esophagus and failure of a normal or hypertensive lower esophageal sphincter (LES) to relax in response to swallowing.\(^1,2\) No form of therapy returns esophageal peristalsis or LES function to normal. Therapy is aimed instead at relieving the functional obstruction at the gastroesophageal junction. Medical efforts to relieve the distal esophageal obstruction have been largely unsatisfactory. Surgical cardiomyotomy, introduced by Heller in 1913, provides excellent relief of dysphagia in 85–95% of patients with achalasia, with minimal complications.\(^1,4\) Thoracotomy or laparotomy, however, has been required, which results in significant pain and prolonged recovery period. Pressure-controlled balloon dilatation and, more recently, botulinum toxin (Botox) injection of the lower esophageal sphincter have therefore become the primary treatment for achalasia.\(^5,7\) This is in spite of lower success rate, frequent retreatment, and, for pneumatic dilatation, a perforation rate of 3–5%.\(^3,5,7\)

Laparoscopic cardiomyotomy for achalasia was first reported in 1991 by Shimi et al.\(^9\) The patient had complete relief of dysphagia and no untoward symptoms. Several reports on laparoscopic cardiomyotomy have been published since then.\(^2,6,8-11\) Today, laparoscopic cardiomyotomy has proven to be safe, effective, and associated with minimal discomfort.\(^2,6,8-11\)

### Preoperative Evaluation and Preparation

It is important to establish the correct diagnosis prior to treatment because other disorders can present in a similar fashion. These include malignant obstruction (especially when the sphincter is infiltrated), gastroesophageal reflux with stricture formation, diffuse esophageal spasms, and nutcracker esophagus. Diagnostic work-up therefore includes an endoscopic examination with biopsies as necessary, a barium swallow, and esophageal manom-
etry. If a peptic stricture is suspected, then a 24-hour pH study is indicated. A CT scan is obtained if a malignant obstruction is suspected.

Patients with proven achalasia who can tolerate general anesthesia are candidates for cardiomyotomy. Prior balloon dilatation and/or botulinum toxin injection is not a contraindication for cardiomyotomy. In these patients, however, the periesophageal dissection and the myotomy may become difficult because of scarring in the area.

The Procedure

The surgeon needs to stand between the patient’s legs, facing the monitors to maintain coaxial alignment with the gastroesophageal junction and the video-laparoscope (Fig. 10.1).

The hiatal exposure and the mobilization of distal esophagus and cardia is the same as that for laparoscopic fundoplication. The dissection begins by incising the avascular area of the gastrohepatic omentum above the hepatic branch of the vagus. This exposes the caudate lobe of the liver and the right crus. The right and left crura of the diaphragm are identified and separated from the esophagus. The posterior esophageal attachments are divided under direct vision. A Penrose drain is passed around the esophagus, and used for traction. The periesophageal dissection is completed by dissecting both crura free of all epiphrenic tissue, mobilizing an adequate length of the esophagus, and developing a posterior window large enough for a loose partial fundoplication (270-degree). The epiphrenic fat pad is dissected off the anterior surface of the gastroesophageal junction and the cardia.

The myotomy is started on the anterior surface of the esophagus, to the left of the anterior vagus nerve, just proximal to the gastroesophageal junction. The longitudinal fibers are separated using the twin action of a pair of scissors. The transverse fibers are separated from the underlying mucosa with blunt dissection, and then divided with hook-cautery or scissors (Fig. 10.2). Once in the submucosal plane, the mucosa bulges up. This is clearly seen in the magnified laparoscopic view. The myotomy is now carried proximally for 5–6 cm above the gastroesophageal junction. The myotomy is carried distally across the gastroesophageal junction and on to the stomach for about 1 cm. On the stomach site, the separation of the muscle layers from the mucosa is more difficult to achieve than it is on the esophagus. This may result in more bleeding than that encountered during the esophageal myotomy, as well as increased risk of perforation. Once the myotomy is completed, the muscle edges are separated from the underlying mucosa for approximately 40% of the esophageal circumference. The orogastric tube, which is placed at the beginning of the procedure, is pulled back into the distal esophagus, and about 100 ml of diluted methylene blue solution is infused down the tube. This will clearly