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Reply to the Editor:

Kirschner's comment focuses on two points: the use of a chest tube after pneumonectomy and the similarity of our case to one recently reported in another journal.[1]

Regarding the use of a chest tube after pneumonectomy, it is worthwhile to repeat the commentaries of Nazari and Cooley cited in Kischner's letter.[2,3] It is a personal choice of the surgeon, and no evidence exists supporting one option or the other. However, we would like to remind Kischner that after pneumonectomy, the chest tube is not attached to continued suction and does not "court disaster produced by excessive mediastinal shift," as he says, but is connected to an appropriately balanced pneumonectomy drain set.[4] This is completely different from the one-way valve used after partial lung tissue resection. It is designed to achieve both positive and negative pressure control avoiding any abnormal mediastinal shift.

We reply to the second point of Kischner's comment stressing the role played by induction chemotherapy in our case, as emphasized both in the title and in the discussion of our article. In the past 5 years we performed 208 pneumonectomies, 47 with intrapericardial vessel ligation. The pericardial defect was closed directly by stitches in 35 cases and by a pericardial patch in 12 cases in which the defect was too large and a direct suture would have been under tension. The reported case of cardiac herniation was the first in our experience. We are sure that the closure we performed in the first operation was not under any abnormal tension, that the pericardium was fibrotic because of the previous chemotherapy, and that this played a major role in its disruption.

The message we would like to give is not that cardiac herniation may happen after intrapericardial pneumonectomy—this is widely known—but that it is more likely if the patient has undergone previous induction chemotherapy. In these cases, the use of pericardium to close small defects can also be recommended.

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Intussusception and spontaneous amputation of the esophagus

To the Editor:

I read with interest the case of a patient with acute leukemia and segmental esophageal necrosis reported by Salo and associates.[1] The authors imply that intussusception led to ischemia, and circumferential full-thickness loss of the esophagus was repaired "without interruption." Both assumptions may be challenged. The patient had no risk factor for gastroesophageal intussusception,[2] in which stomach is the intussusceptum, not esophagus, and perforation, rather than necrosis, a reported complication. Fungal infection of the esophagus in leukemia, however, is known to result in full-thickness necrosis and esophageal expulsion.[3] Their final comment relating to *Aspergillus* infection of the esophageal wall points to the likely cause of necrosis.

A complete loss of all esophageal layers, no matter how short, is expected to result in cicatricial stricture that may have been delayed in this case by cytotoxic therapy. Indeed, dysphagia later developed, necessitating further intervention. The mediastinal soft tissues may resemble the esophageal lumen so that perforation is not obvious. However, bare of epithelium and exposed to esophageal contents, the mediastinum provides an entry to systemic infection, particularly in an immunosuppressed state. Esophageal resection with immediate or delayed reconstruction should be considered even in this patient population to prevent mediastinitis and restore swallowing.

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Reply to the Editor

We thank Gaissert for his very valuable comments.

Spontaneous amputation of the esophagus is a serious and rare event, and its exact pathophysiology is not well understood. There were several reasons that led us to believe that intussusception, followed by ishemic necrosis, may be an etiologic factor. Our patient had a hiatal hernia, as previously stated. Hiatal hernia is a widely known risk factor for intussusception, as well as dysphagia, a condition that developed after severe vomiting.[1] On careful inspection of Figure 1, one can see that the inner circular muscle layer of the esophagus is located superficially as a result of intussusception.

Abildgaard and associates[2] have described total expulsion of the distal esophagus in a severely immunocompromised patient with *Candida* esophagitis. Multifactorial pathogenesis, including intussusception, is presumable inasmuch as their patient had also had regurgitation, vomit-

ing, and nausea for at least 6 days before esophageal expulsion. Fungal necrosis of the esophagus may also have appeared secondarily. As we stated in our article, concomitant infections of the esophageal wall with *Aspergillus* may have contributed to the pathogenesis in our patient as well. There was no sign of perforation or esophageal fistula at esophagography, after the amputated segment was removed, and the patient was able to eat and drink for the next 4 weeks.

Gaissert recommends that esophageal resection, with immediate or delayed reconstruction, be performed to prevent mediastinitis and to restore swallowing, even in patients as seriously ill as ours. We use the previously described procedures[3] in cases with delayed perforation and rupture when the basic disease has a reasonable prognosis. Nevertheless, in our case, stricture and dysphagia were successfully palliated, at endoscopy,[4] with dilatation and the use of a self-expandable metallic stent. After this procedure, the patient's ability to swallow was almost normal until her death, a result of leukemic relapse, 8 months after the primary event.

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