Thoracic surgeons are familiar with identifying gastric distension on portable chest radiographs early after major lung resection. This finding typically resolves spontaneously during the ensuing postoperative days. The aetiology of postoperative gastric distension is unclear. When present on the immediate postoperative film, it is likely attributable to air insufflation into the stomach during bag-mask ventilation upon anaesthetic induction or while changing from a single-lumen to a double-lumen endotracheal tube. Ineffective bag-mask ventilation due to difficult patient characteristics (e.g. obesity, obstructive sleep apnoea, etc.) or poor technique is more likely to result in this finding.

A separate group of patients exhibits maximum gastric distension on postoperative day 1, and this is not likely to be attributable primarily to air insufflation by the anaesthesiologist. Instead, it is more likely a result of swallowed air that has not passed further into the gastrointestinal tract. That such distension is not usually evident in patients who are electively left intubated overnight after lung resection (usually without a nasogastric tube) for planned early extubation the following morning raises the important question of whether impaired gastric motility is a necessary precondition for such gastric distension.

The paper by Renaud et al. [1] published in this issue is the first to quantify the extent of gastric distension present after major lung resection, determine clinical factors that are associated with its development, and suggest an aetiologic relationship between the presence of gastric distension and the development of postoperative pneumonia. It is likely that in their patient population gastric distension was due to swallowed air rather than insufflated air: patients routinely had a nasogastric tube placed during their operations, and, importantly, the authors studied the development of gastric distension that occurred between postoperative days 0 and 1.

Some study design choices the authors made may have had a direct bearing on the outcomes of the study. The categorization of ratios for gastric distension apparently was arbitrary. Rather than using cutoffs of 1.3 and 2.3, other options might have been 1.5 and 2.0, or 1.5 and 2.5. Whether these values were chosen prior to the data analysis is unclear. In addition, the selection of postoperative day 5 as the determinant of whether pneumonia developed was also arbitrary. In an ideal study, any pneumonia attributable to the operation would have counted as an infectious complication.

The authors logically attribute gastric distension to vagal nerve injury that occurs as a result of mediastinal nodal dissection. It is less obvious why gastric distension was more pronounced after pneumonectomy than after lesser operations. The anatomic rationale provided by the authors is not immediately convincing. It is possible that the authors' efforts to compensate the problem with the other vagus nerve. I really like the idea of an irritation of the vagus nerve that can cause gastric distension. For me, it is hard to understand or to believe that irritation of the vagus nerve on one side leads to the consequences you have shown. There is an old operation which was done in the era before we had all the drugs to block acid production, which was selective proximal vagotomy, and we learned during these operations that there is a huge network of the vagus nerve on both sides of the oesophagus and also of the stomach. So irritation of the vagus nerve on one side should not really lead to these severe consequences. So for me it is difficult to believe that this is really the main reason for the problem.

Concerning the dilatation of the bowel, if we had a doubt between the stomach or the bowels, these patients were excluded. For us, we ensure that all the measures that have been done are measured down on the stomach. And concerning the measure, of course, if it is distension with liquid, we were not able to measure it.
hemidiaphragm. As the authors correctly state, the use of parenteral opioid is known to slow gastrointestinal motility. However, it is not clear whether the epidurals used for postoperative pain control employed opioids alone, local anaesthetics alone or some combination for all patients. Further, the effectiveness of the analgesia is not quantified. Pain, too, is known to slow gastric emptying.

The rationale the authors provide for why gastric distension is associated with an increased risk of postoperative pneumonia is based on microaspiration related to gastroesophageal reflux. While this is reasonable, no data are offered to support this hypothesis. It is entirely possible that gastric distension is a surrogate for other factors that might influence the development of pneumonia. Poor oesophageal motility resulting from vagal injury might promote microaspiration in the absence of gastroesophageal reflux. Placement of a nasogastric tube in patients who were found to have more profound degrees of gastric distension may have promoted aspiration during tube placement, or may have interfered with pulmonary toilet exercises after its placement. The presence of gastric distension may have reduced functional residual capacity, resulting in closure of small airways and trapping of secretions.

In summary, the study by Renaud et al. describes an important radiographic observation related to clinical outcomes in patients after major lung resection. It offers an intriguing hypothesis regarding the aetiological relationship of postoperative gastric distension to the observed increase in postoperative pneumonia. As is common with seminal studies of this sort, more questions are raised than are answered by the findings. The study provides ample opportunity for other investigators to confirm the observed relationship between gastric distension and postoperative pneumonia, and to explore the mechanisms that underlie this observation. We anticipate that this will be a fruitful area for future clinical research.

Conflict of interest: none declared.

REFERENCE