A Sudden Increase in Expired Nitrogen: Diagnosis and Management of a Bronchial–Gastric Fistula

David J. Murray, M.D.*

Sudden changes in nitrogen concentrations in inspired or expired gases can provide important diagnostic information.1,2 When end-tidal nitrogen concentration increases, an immediate search for a source of air entrainment is imperative. Venous air embolus, the most life-threatening cause of an increase in end-tidal N₂ concentration, must be distinguished from other sources of air.2,3 Increased expired N₂ can also originate from vaporizers, humidifiers, or air entrained from leaks in anesthesia circuits or gas sampling equipment.

In this patient, a sudden increase in end-tidal N₂ concentration was caused by an undiagnosed bronchial–gastric fistula. The presence of end-tidal N₂ established a definitive diagnosis of a fistula, helped localize the fistula, and later confirmed endoscopic closure of the distal bronchial–gastric fistula.

Case Report

A 17-yr-old, 56-kg girl presented for bronchoscopy, esophagoscopy, and gastroscopy during general anesthesia to evaluate the cause of a persistent left lower lobe lung infiltrate. She had been discharged from the hospital less than 3 weeks previously after a prolonged hospitalization for esophageal anastomotic leak/stenosis after esophagegastrostomy.

Her medical history was complicated. She was born with the most common type of tracheoesophageal fistula (a blind esophageal pouch and a distal tracheoesophageal fistula). The patient had required multiple complex surgical procedures to repair the tracheoesophageal fistula that was associated with distal esophageal atresia. Four months before the current procedure, an esophagogastric anastomosis was performed, which resulted in a prolonged hospitalization associated with a prolonged esophageal leak and persistent left pleural effusion. Eventually, the patient tolerated clear liquid feeds and was discharged from the hospital, but fewer than 3 weeks later, she was readmitted with a persistent left lower lobe bronchopneumonic process. She returned to surgery for a diagnostic bronchoscopy, esophagoscopy, and gastroscopy to evaluate the cause of the persistent left lower lobe infiltrate. She had a respiratory rate (RR) of 16 breath/min, heart rate (HR) of 76 beat/min, and blood pressure (BP) of 130/70 mmHg.

After anesthesia induction with intravenous propofol, isoflurane in O₂ was administered via a semiclosed circle system. Endotracheal intubation (6.0 cuffed tube) was performed after neuromuscular blockade had been achieved with vecuronium. Anesthesia was maintained with a combination of isoflurane in O₂ and a propofol infusion. Rigid bronchoscopy was followed by fiberoptic bronchoscopy to search for a cause of the persistent left lower lobe infiltrate. No evidence of a fistula was observed during either examination. During gastroscopy via the gastrostomy site, the use of positive pressure ventilation combined with repeated sustained increases in airway pressures during endoscopy did not help identify an air leak between either the esophagus or stomach and bronchial tree. During gastroscopy, intermittent increases in nitrogen were observed in expired gas using a Raman Scanning Analyzer (Rascal II, ® Ohmeda, Salt Lake City, UT) (fig. 1). Endoscopy was discontinued, and end-tidal N₂ disappeared. The absence of changes in end-tidal CO₂, BP, and HR suggested a problem with gas monitoring equipment or entrainment from around the endotracheal tube. These potential sources of air were excluded. When endoscopy resumed, intermittent increases in N₂ occurred. Air insufflation during gastroscopy was determined to be the cause of N₂ in expired gas. The presence of end-tidal N₂ suggested a bronchial–gastric fistula. Over the next hour, a probing catheter inserted via the gastroscope was used to inject air to explore for potential fistula sites. The source of the fistulous communication was localized to a segment of fundus of the stomach.

Twenty-four hours later, a repeat endoscopic procedure was planned to seal the bronchogastric fistula using “fibrin glue.”4 Anesthesia induction was similar to the initial procedure, but during this procedure, intubation (6.0 cuffed tube) was conducted during deep isoflurane and propofol anesthesia. The patient was allowed to breathe spontaneously after endotracheal anesthesia. During gastroscopy, air insufflation was associated with increased expired N₂ concentrations. The fistula site was identified, and “fibrin glue” was prepared and injected via the gastroscope into the fistula site.

Five minutes after the initial “fibrin glue” injection, end-tidal N₂ was still observed after air insufflation. The injections via the gastroscope were repeated three times. Ten minutes after the last injection, N₂ was no longer observed in expired gas during air insufflation. The...
most serious cause of increased end-tidal $N_2$, venous air embolus, was the differential diagnosis that needed to be rapidly excluded. The presence of elevated expired $N_2$ caused initial concern about the possibility of venous air (fig. 1). In operative settings not usually associated with venous air emboli, an increase in expired nitrogen warned of an air embolus before cardiovascular collapse. $^1$-3 Venous air was believed an unlikely source of the increased nitrogen concentrations in this case for a variety of reasons. First, expired nitrogen exceeded 5% and 10% of expired gas concentrations. In animal studies, a bolus or infusion of 1.5 ml per kg of air produced serious cardiovascular signs, but less than a 1.0% increase in end-tidal $N_2$ concentrations. $^1$-5 An expired $N_2$ concentration of 10% would also have been expected to cause a large increase in dead space ventilation and cardiovascular collapse if venous air emboli had been the source of expired $N_2$. The increases in end-tidal $N_2$ with venous air emboli are usually small and sustained for more than 5–10 min even when air enters the venous circulation as a bolus. $^1$-5

Brief, often large, increases in inspired and expired $N_2$ concentrations occur when the source of air is from anesthesia equipment such as a leak in a gas sampling line. The initial absence of $N_2$ in inspired gas ruled out anesthesia equipment, a leak from around the endotracheal tube, or a gas sampling problem as the cause of the increased nitrogen concentration in this patient (fig. 1). The presence of air in expired gas during gastroscopy made this a unique differential diagnostic problem in this patient. The inability to detect an air leak from the distal bronchus during gastroscopy even with controlled ventilation and sustained increases in intrathoracic pressure suggest the fistula must have been small. This leak might also have not been detected during endoscopy because of the distal location of the involved bronchus, the persistent left lower lobe consolidation, or dense adhesions in the left chest from a previous esophageal anastomotic leak. During the second procedure to occlude the fistula, end-tidal $N_2$ was used to determine whether the fistula had been effectively sealed using “fibrin glue.” In this patient, multiple previous thoracic operations and prolonged esophageal leak had made thoracotomy to manage the fistula an unsatisfactory alternative to an endoscopic approach.

In summary, in this unusual case report, nitrogen monitoring provided important diagnostic and therapeutic information in a patient with an undiagnosed bronchial–gastric fistula. The report illustrates some of the important differential diagnostic features of in-
creased expired nitrogen concentration and how this information was used to diagnose a bronchial–gastric fistula.

References