Deaths associated with insertion of nasogastric tubes for enteral nutrition in the medical intensive care unit: Clinical and autopsy findings

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ABSTRACT

It is generally assumed that blind insertion of nasogastric tubes for enteral nutrition in patients admitted to medical intensive care units is safe; that is, does not result in life-threatening injury. If death occurs in temporal association with insertion of a nasogastric tube, caregivers typically attribute it to underlying diseases, with little or no consideration of iatrogenic death due to tube insertion. The clinical and autopsy results in three recent cases at Baylor University Medical Center challenge the validity of these notions.

KEYWORDS Enteral nutrition; iatrogenic death; nasogastric tube

For patients in the medical intensive care unit (ICU) who cannot eat, enteral nutrition is preferred over parenteral nutrition because it maintains intestinal integrity and causes less infectious complications.¹ Because ICU patients are critically ill and often require aggressive resuscitation and monitoring, it is usually not practical to transport them to radiology or endoscopy departments for insertion of feeding tubes under fluoroscopic or endoscopic guidance. In our hospital, and probably also in many others, it has therefore become standard care to insert nasogastric tubes blindly (i.e., without fluoroscopic guidance) at the ICU bedside.² Although it is well recognized that tube misplacement can occur with this procedure,^{3,4} blind insertion is assumed to be safe—that is, not result in life-threatening complications—if a portable x-ray is obtained to confirm correct tube position in the stomach before feeding formula is infused. This assumption of safety from life-threatening injury is so widely accepted that blind insertion of feeding tubes is routinely performed by nurses who are not required to obtain specific certification for this procedure. This article describes clinical and autopsy findings in three patients in the ICU where trauma from blind insertion of nasogastric tubes was the proximate cause of death. The definition of *proximate cause*, as used in this article, is as follows: that which produces an effect, without which the result would not have occurred at that time, and that which is nearest in the order of responsible causation.⁵ This determination was based

on both the clinical and autopsy findings. *Table 1* summarizes some of the main clinical and autopsy findings in these three patients.

CASE 1

A 75-year-old woman was transferred to Baylor University Medical Center (BUMC) after experiencing cardiac arrest (pulseless electrical activity) at a nearby emergency department. Medical personnel regained a pulse after 3 minutes of cardiopulmonary resuscitation (CPR). While en route to BUMC, her sons provided her past medical history, which included prior ischemic stroke without residual deficits, atrial fibrillation, and hyperlipidemia. Warfarin was the only medication she had been taking.

At BUMC, she was stabilized on vasopressors and mechanical ventilation while hypothermia was induced. A 16 Fr Salem Sump tube was inserted through the nose into the stomach, with confirmation of gastric placement by roentgenogram. Subsequently, enteral nutrition was provided via this tube. Cardiac catheterization revealed extensive multivessel disease with >90% occlusion of left and right coronary arteries. Stenting of these coronary occlusions was not performed. Over a 10-day period, her cardiogenic shock resolved. On day 13, the endotracheal tube was removed with transition to bilevel positive airway pressure. She was alert and eager to have the nasogastric tube removed.

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Table 1. Clinical and autopsy findings in three women dying after blind insertion of Salem Sump nasogastric tubes in intensive care units

Patient characteristics	Case 1	Case 2	Case 3
Age (years)	75	81	52
Primary admission diagnosis	Myocardial infarct	New-onset aphasia	Pulmonary embolism
Oropharyngeal dysphagia	Yes	Yes	No
Prior stroke	Yes	Yes	No
Days of endotracheal intubation prior to NGT injury	13	0	14
Tracheostomy	No	No	Yes
Coagulopathy	Yes; warfarin	Yes; warfarin	Yes; heparin
Blood transfusions	No	Yes	Yes
Proximate cause of death	Bronchial perforation by NGT	Nasopharyngeal hemorrhage from NGT trauma	Nasopharyngeal hemorrhage from NGT trauma

NGT indicates nasogastric tube.

A bedside swallowing evaluation revealed pharyngeal dysphagia and decreased hyolaryngeal excursion. The nasogastric tube was therefore left in place for continued enteral nutrition. Warfarin was also delivered through the nasogastric tube. Repeat bedside swallow evaluation on day 21 revealed the same swallowing deficits. On day 26, a dysphagiagram was performed with nectar-thickened contrast. Decreased pharyngeal peristalsis and cricopharyngeal relaxation were noted. In addition, moderate vallecular and pharyngeal residuals were visualized in the pyriform sinuses along with decreased laryngeal elevation. Follow-up swallows resulted in aspiration of the residual contrast in her valleculae. She was deemed to be at high risk for aspiration when she ate food, and it was recommended that enteral nutrition be continued through the existing nasogastric tube.

After returning from the radiology department, it was discovered that the existing nasogastric tube was obstructed, and the attending physician ordered a replacement. A nurse attempted to insert a 16 Fr Salem Sump tube through the left nostril but the tube exited the patient's mouth. Therefore, a second attempt at placement was performed by another nurse. With accompanying explanation to the patient, a Salem Sump tube was inserted without any appreciated resistance until the location of "the tape" suggested full advancement into the stomach. Several minutes later, "blood returned at the proximal tip" of the tube. At the same time, the patient experienced discomfort and coughing and demanded that the tube be removed.

Fifteen minutes after removal of the nasogastric tube, the patient abruptly became unresponsive to command, developed frank hematemesis, and then lost a palpable pulse. Her code status was "do not attempt resuscitation" but, given the witnessed events leading up to her loss of pulse, her sons requested that CPR be performed. Despite CPR, the patient died 65 minutes after attempting to replace the nasogastric tube. A "chest-only" autopsy was requested by her sons.

Gross examination revealed bloody mucous within the nares. The upper airway and hilar bronchi were patent, and pulmonary vascular structures were free of thrombo-emboli. There was 650 mL of liquid and clotted blood in the right pleural cavity. All lobes of both lungs contained intraparenchymal, intrabronchial, and intraalveolar hemorrhages. Upon dissection, a distinct 4-cm hematoma was present within the lower lobe of the right lung (*Figure 1*), which expressed blood through a small pleural defect with applied pressure. The lateral basal segmental bronchus extended directly into this hematoma, and the bronchial wall was thin, friable, and perforated at this site. Multiple rib fractures were present (consistent with attempted resuscitation), but none were adjacent to the disrupted pleura. Microscopically, within the lower lobes there were multinucleated giant cells, neutrophils, and vegetable material, consistent with previous aspiration.

Radiographic and autopsy measurements in this patient revealed that there was a 46-cm distance from this patient's

Figure 1. (a) Autopsy findings in case 1. The segmental bronchus of the right lower lobe of the lung was perforated (white arrow) with an adjacent pleural perforation and hematoma (blue arrow). **(b)** Reference drawing, courtesy of Kenhub (<https://www.kenhub.com>), illustrator Yousin Koh. Accessed March 19, 2018.

nares to the furthest extension of the right lower end of the lung. Conversely, the nares-to-body-of-the-stomach distance measured 55 cm. Based on these measured anatomical lengths, if a feeding tube inserted through the nose was misplaced into the trachea and advanced without coiling to the calculated distance into stomach (as indicated by "the tape" placed on the tube), the tip of the tube would perforate the right bronchus and extend well into the pleural space.

The heart weighed 425 g. The anterolateral and posterior portions of the left ventricular free wall showed foci of subendocardial necrosis and fibrosis.

It was concluded that the proximate cause of death was bronchial perforation by a nasogastric tube that was inserted for enteral feeding, with hemorrhage into the parenchyma of the right lung and right pleural cavity. Subsequent respiration caused blood to aerosolize throughout the airways, causing asphyxiation. A contributing cause of death was pharyngeal dysphagia.

CASE 2

An 81-year-old woman presented to the emergency department with new-onset expressive aphasia. Her past medical history included atrial fibrillation, chronic kidney disease, chronic lymphocytic leukemia, and a stroke with residual pharyngeal dysphagia. Twelve months prior to admission, a dysphagia-gram revealed delayed swallowing of contrast with residual contrast visualized within the valleculae and piriform sinuses. Since that time, she had been on a nectar-thickened liquid diet with no progression of the dysphagia or aspiration pneumonia.

The timeline of her newly developed aphasia placed her outside the treatment window for thrombolytic therapy. Because of atrial fibrillation, she was on therapeutic doses of warfarin with an international normalized ratio of 3.6. Laboratory studies revealed a hemoglobin of 10.7 g/dL, a white blood cell count of 2.4 K/ μ L, and creatinine of 4.4 mg/dL. Computed tomography of the brain with angiography revealed patency of intracranial vessels with no occlusion or aneurysm.

A bedside swallow study revealed bilateral buccal muscle weakness with spillage of liquids, decreased hyolaryngeal elevation, and immediate coughing with thin and nectar-thickened liquids. She was deemed to be at high risk for aspiration, and a 16 Fr Salem Sump tube was placed blindly through her nostril for enteral feeding and medication administration. Proper gastric placement was confirmed by roentgenogram. The patient received enteral formula for 24 hours but, on the following day, she intentionally removed her nasogastric tube. At that time, her acute kidney injury had resolved, but a decision was made to reinsert a nasogastric tube for enteral nutrition prior to transfer to a skilled nursing facility.

On day 3, blind placement of a Salem Sump tube was attempted through the left nostril but failed after the tube exited the patient's mouth. Two additional attempts were made without successful passage through the nasopharynx. On a fourth attempt, the tube was passed into the right nostril, down the esophagus, and subsequently into the stomach. Proper gastric position was confirmed by roentgenogram. Forty minutes later, the nurse discovered heavy bleeding from the left

nostril. Pressure and phenylephrine nasal spray were ordered by the attending physician. However, bleeding continued, with the development of large blood clots extending into the nasopharynx. The nasogastric tube was removed and suctioning of the blood along with placement of nasal packing was performed. Her international normalized ratio was 3.2 so warfarin administration was discontinued. On day 4, her respiratory status became compromised with hypoxia, and endotracheal intubation was required. While directly visualizing the vocal cords, the critical care physician witnessed passage of copious amounts of blood from the posterior nasopharynx into the trachea. At this time, a 16 Fr Salem Sump tube was inserted through her mouth by nursing staff for subsequent enteral nutrition or gastric decompression. Her hemoglobin concentration dropped to 6.2 g/dL, so she received 2 units of packed red blood cells and fresh frozen plasma. Subsequently, her blood counts stabilized, but she continued to require mechanical ventilation. On day 6, she went into cardiac arrest. An autopsy was requested by her daughter.

A well-positioned orogastric tube was present along with two nasal packs. An oral endotracheal tube was well positioned. Anatomical cuts at the base of the skull to expose the sphenoid and ethmoid sinuses and upper nasal cavity revealed liquid blood and clots contiguous with the sphenoethmoidal recess at the upper posterior aspect of the nasal cavity (*Figure 2*), consistent with nasal mucosal hemorrhage.

The brain revealed acute hypoxic ischemic changes and hypertensive vasculopathy. The lung parenchyma appeared edematous and hemorrhagic. Microscopically, there was patchy bronchopneumonia. The heart weighed 445 g with biatrial dilatation and mitral annular calcium deposition. The coronary arteries were patent and there were no myocardial lesions.

The liver weighed 1475 g. Microscopically, there was chronic portal tract inflammation and mild periportal fibrosis. The kidneys had severe arteriolar nephrosclerosis. Small bilateral renal cell carcinomas were also present. A small gastrointestinal stromal tumor was present in the appendix.

The spleen weighed 30 g with no evidence of leukemic infiltrates. There was mild enlargement of peripancreatic lymph nodes, but no evidence of lymphoma or leukemia was seen in these nodes or the bone marrow.

It was concluded that the proximate cause of death was nasal cavity injury from insertion of a nasogastric tube for enteral nutrition, which led to hemorrhage, aspiration of blood, respiratory distress, hypoxic ischemic brain injury, cardiac arrest, and death. Contributing causes of death were warfarin therapy for atrial fibrillation, pharyngeal dysphagia, and the patient's decision to remove the first nasogastric tube, which led to a physician order for reinsertion.

CASE 3

A 52-year-old woman with a past medical history of obesity, asthma, and sarcoidosis presented to an outside emergency department with dyspnea. Computed tomography with angiography of the chest revealed bilateral pulmonary thromboembolism and right ventricular dysfunction. Tissue plasminogen

Figure 2. (a) Autopsy findings in case 2. Transverse section of the anterior cranial fossa (looking down into the skull) reveals the abnormal presence of blood clots within the upper nasal cavities (white arrow), indicating nasopharyngeal hemorrhage. (b) Reference drawing for orientation, courtesy of Basicmedical Key (<https://basicmedicalkey.com/the-musculoskeletal-system/>). Accessed March 19, 2018).

activator was administered intravenously. However, she went into cardiac arrest with pulseless electrical activity. CPR was initiated, and return of spontaneous circulation was achieved after 25 minutes. Tracheal intubation was performed, and a 16 Fr Salem Sump tube was inserted through the mouth into the stomach. Correct placement of both tubes was confirmed by roentgenogram. Over the next 24 hours, she developed acute respiratory distress syndrome and disseminated intravascular coagulation. Subsequently, she was transferred to BUMC.

At BUMC, she was continued on a heparin drip for pulmonary embolism, vasopressors for hypotension, and mechanical ventilation for respiratory failure. In addition, she was started on venoarterial extracorporeal membrane oxygenation (ECMO) for further cardiovascular and respiratory support along with continuous renal replacement therapy for acute renal failure. Laboratory studies revealed a hemoglobin concentration of 8.5 g/dL. Serum concentrations of liver markers were as follows: aspartate aminotransferase, 681 U/L; alanine aminotransferase, 130 U/L; and bilirubin, 2.0 mg/dL.

The orogastric tube remained in place on low to intermittent suction for 2 days but was then converted to a gastric feeding tube with enteral formula. On days 4 through 12 of the patient's hospitalization, she remained sedated on mechanical ventilation, ECMO, and continuous renal replacement therapy. Elevated liver enzymes normalized and disseminated intravascular coagulation resolved. The intravenous heparin drip was continued without evidence of bleeding. However, blood cultures grew *Candida albicans*, and micafungin was initiated. After decannulation of the ECMO catheters, skin injury and necrosis occurred in the groin area. This was managed with local wound care and antibiotics.

On day 14, a tracheostomy was placed and the orogastric tube was removed during this procedure. Thereafter, a 16 Fr Salem Sump tube was inserted blindly through the right nostril, with confirmation of gastric placement by roentgenogram. This Salem Sump tube was to be used for gastric

decompression. A styletted 10 Fr Dobhoff feeding tube was inserted through the left nostril and into the stomach, using a Contrak imaging device, but the tip of the tube could not be maneuvered through the pylorus. On day 15, a Contrak device was used again to assist the placement of a Dobhoff feeding tube into the duodenum. The tube passed successfully through the nasopharynx without reported issues, but the tip would not pass through the pylorus. This Dobhoff tube was left in the stomach (confirmed by roentgenogram) and enteral feeding was initiated through it. Several hours later, epistaxis was noted in both nostrils and bright red blood was visualized in gastric fluid aspirated through the Salem Sump tube. Her hemoglobin concentration was 8.4 g/dL. Over the next 3 days, epistaxis and bloody return from the Salem Sump tube increased. The nasal passageway was packed while the Dobhoff tube was left in the stomach. Melena occurred over the next 24 hours, and 7 units of packed red blood cells were infused intravenously. Vasopressors were reinitiated for blood pressure support. On day 20, her hemoglobin level dropped to 6.5 g/dL and upper endoscopy revealed bright red blood in the esophagus along with 1200 mL of dark red blood in the stomach without evidence of a bleeding gastrointestinal source. She received additional blood transfusions. An echocardiogram revealed normal systolic function and grade 1 diastolic dysfunction. On day 21, she required an increased level of vasopressor support. Her aspartate aminotransferase concentration was 6377 U/L, alanine aminotransferase was 1701 U/L, alkaline phosphatase was 212 U/L, and bilirubin was 23.2 mg/dL. Her hemoglobin concentration was 5.2 g/dL. On day 22, she developed fatal cardiac arrest. Despite antibiotics, blood cultures obtained 6 hours prior to her death grew *Klebsiella pneumoniae*.

On autopsy, there were bloody gauze packs within the nares bilaterally. A 16 Fr Salem Sump tube was present within the right naris with its terminus in the stomach. A 10 Fr Dobhoff tube was positioned through the left naris and also terminated in the stomach. A tracheostomy tube was well positioned

Figure 3. (a) Autopsy findings in case 3. Transverse section of the anterior cranial fossa reveals extensive blood and clots throughout the upper nasal cavities indicating nasopharyngeal hemorrhage (white arrow). (b) Similar view of the anterior cranial fossa in a patient without nasopharyngeal hemorrhage, demonstrating the normal absence of blood within the sphenoid sinus.

in the trachea. On sectioning the base of the skull, extensive hemorrhage was observed within the sinuses and upper nasal cavities (Figure 3). Microscopically, there was mucosal and submucosal disruption and hemorrhage of the nasal mucosa adjacent to the sphenoid sinus (Figure 4).

The stomach contained 1625 mL of liquid and clotted blood. However, the gastric mucosal surface did not have any focal ulcerations, polyps, or tumor masses. The small bowel mucosa appeared dusky but without evidence of ulceration or perforation. Microscopically, the small bowel mucosa was focally hemorrhagic and inflamed, consistent with ischemic injury. The liver revealed marked steatosis without inflammation or necrosis. The gallbladder contained multiple black gallstones. The common bile duct was not dilated or obstructed.

The larynx, trachea, and bronchi were patent. The lung parenchyma was edematous and congested. The pulmonary hilar vasculature was patent, but peripheral vascular structures contained organizing thrombi. Sections of a large pelvic vein near the aortic bifurcation revealed an organizing thrombus. The heart weighed 290 g with increased subepicardial adipose tissue. The right atrial and ventricular cavities were dilated but no valvular or myocardial lesions were seen. The coronary arteries and aorta were free of significant atherosclerosis. Although there were large calcified mediastinal lymph nodes consistent with sarcoidosis, active granulomatous inflammation was not seen. There were multiple areas of cutaneous ulceration involving the thighs. Postmortem cultures grew *C. albicans* and *K. pneumoniae* from the lungs and *K. pneumoniae* from blood within the heart.

It was concluded that the proximate cause of death was nasal cavity injury from insertion of nasogastric tubes for enteral nutrition, which led to hemorrhage and irreversible hypovolemic shock. A contributing cause of death was anticoagulation for pulmonary thromboembolism.

DISCUSSION

All three patients had comorbidities that made them vulnerable to acute pathophysiological derangements, but these are an accepted feature of hospital ICU practice and, in our opinion, they do not mitigate the tragedy of iatrogenic injury due to insertion of nasogastric tubes for enteral nutrition.

Moreover, in cases 1 and 2, the patients were recovering from the illnesses that prompted their ICU admission, and their autopsies revealed no underlying residual disease that would soon have claimed their lives (such as organ failure, malignancy, or infection).

In case 1, blind insertion of a nasogastric tube for enteral nutrition resulted in intubation of the trachea rather than the stomach. Three factors probably predisposed this particular patient to nasogastric tube misplacement: (a) a previous stroke that impaired her swallowing center in the medulla of the brain; (b) prolonged endotracheal intubation, which can impair closure of the glottis^{6,7}; and (c) advanced age, which is associated with calcification of the epiglottis.⁸

Inadvertent placement of a nasogastric tube into the trachea does not usually result in death, and there are at least two ways in which death from tracheal misplacement in this patient could have been avoided. First, she was no longer critically ill and she could have been sent from the ICU to the radiology department and had the nasogastric tube inserted under fluoroscopic guidance. Second, a two-step x-ray protocol might have prevented injury from tracheal intubation during nasogastric tube insertion.^{9–11} In this protocol, 30 cm of the tube is

Figure 4. Case 3, microscopic examination. Upper nasal cavity mucosa adjacent to the sphenoid sinus was focally disrupted (black arrow) with submucosal hemorrhage (white arrow). Hematoxylin and eosin, original magnification $\times 100$.

Figure 5. Line drawing of the nasal and oral routes through which a feeding tube can be inserted into the stomach. The red dot denotes the sphenoid recess, and the blue dot denotes the sphenoid sinus. The green dot denotes the larynx. The black arrow denotes mucosa of the nasal cavity, and the red arrow denotes mucosa of the hypopharynx.

inserted, an x-ray is taken, and only when the tube is known to be in the esophagus is the tube inserted further. A second chest x-ray is obtained after further advancement to confirm proper location in the stomach before tube feedings are started. In this case, a nasogastric tube was blindly inserted to the full length calculated to place the proximal aperture of the tube below the esophageal-gastric junction. That distance exceeds the distance between the nares and the lower end of the bronchial tree. Therefore, when the tube was advanced to its predetermined distance, bronchial perforation, severe bleeding, and asphyxiation with blood occurred.

In case 1, the patient was not allowed to eat food because pharyngeal dysphagia made her likely to develop aspiration pneumonia. Her physicians therefore ordered blind insertion of a nasogastric tube so that enteral nutrition could be provided. In our opinion, any patient who is deemed unfit to eat because of pharyngeal dysphagia is unfit to have blind insertion

of a nasogastric tube because of the risk of tracheal, rather than esophageal, intubation.

In case 2 and case 3, feeding tubes were blindly inserted through the nose in patients with known coagulopathies, and both patients developed massive bleeding from traumatized nasal mucosa. *Figure 5* illustrates the anatomy of the nasal cavities and adjacent structures. Except for its most anterior portion, the nasal cavities are lined by a pseudo-stratified columnar epithelium that rests on delicate supporting connective tissue.¹² The epithelium is relatively thin and the submucosa is crowded with veins that can become rapidly dilated with blood.¹³ The nasopharynx is lined by a combination of pseudo-stratified columnar epithelium and stratified squamous epithelium. Both the oropharynx and the hypopharynx are lined continuously by a stratified squamous epithelium.¹⁴

To illustrate some of these well-known differences, we obtained tissue samples from the posterior roof of the nasal cavity and from the hypopharynx. Histological staining (*Figure 6*) revealed the delicate structure of the nasal epithelium, in contrast to the much thicker squamous epithelium of the hypopharynx. If blind insertion of a tube for gastric decompression or enteral nutrition is absolutely required in a coagulopathic patient, oral insertion might be less likely to cause trauma and severe bleeding than nasal insertion. The relative degree to which oral and nasal feeding tubes can be tolerated by patients in the ICU has not been carefully studied.

In case 2, a patient with longstanding pharyngeal dysphagia intentionally removed a functioning nasogastric tube. This led to multiple attempts to replace the tube, which in turn resulted in nasal cavity injury, massive bleeding, and death. Without belaboring the multitude of moral questions involved in this sequence of events, it seems to us that a long-term nasogastric feeding tube was not an acceptable method of delivering nutrition to this particular patient and that reinsertion of the tube should not have been attempted without obtaining formal written consent from the patient or from her daughter. Consideration of other options, including hospice, would have been preferable in this patient who had an international normalized ratio of 3.2. Furthermore, nurses who are ordered to insert nasogastric tubes by doctors need to be encouraged to stop when an inserted nasogastric tube keeps coming out of the mouth. When this happens, it should be interpreted as an indication of pharyngeal incoordination that

Figure 6. Tissue samples obtained from patients undergoing autopsy who had not required enteral feeding tubes or endotracheal tubes prior to death. (a) From the upper nasal cavity, adjacent to the sphenoid sinus. The pseudo-stratified columnar epithelium is thin and delicate. (b) From the oropharynx. The stratified squamous epithelium is relatively much thicker than mucosa from the upper nasal cavity. Hematoxylin and eosin, original magnification $\times 100$.

predisposes the patient to serious injury when a stiff tube is forced through the nose.

At our institution, the electronic order set for nasogastric tube placement contains only two options: (a) insert nasogastric tube (which is interpreted by the nursing staff to mean full-distance blind nasogastric insertion, followed by a single x-ray to establish proper placement in the stomach before infusing enteral formula) and (b) remove nasogastric tube. These limited options promote inappropriate intubation protocols for some patients and lack of safety precautions for others. In our opinion, physician orders should reflect careful consideration of swallowing function, coagulation status, and length of previous endotracheal intubation. In patients with coagulopathy, physicians should be able to specify that a nasogastric tube be inserted through the mouth rather than the nose. In patients with pharyngeal dysphagia or prior prolonged endotracheal intubation, physicians should be able to order either a two-step radiographic protocol^{9–11} or insertion under fluoroscopic guidance.¹⁵

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