thetized, paralyzed and sedated.[1,2] Ozer and Benumof have found that the most common sites of impaction of orogastric and nasogastric tubes are pyriform sinuses and arytenoids cartilages, rendering its coiling in the oropharynx.[3] In our experience, in addition to the impaction of tube against these structures, the basic design of the tube contributes to tube coiling in the oropharynx. The distal 6 cm of the gastric tube has multiple holes that are weak points. Once the tube is impacted against the pyriform sinuses or arytenoid cartilage, bending of the tube occurs at these weak points, thereby promoting coiling and retarding its entry into the esophagus.

We explain a technique of digital assistance to facilitate the insertion of gastric tube (orogastric or nasogastric) in anesthetized and sedated patient. The gloved index finger of the left hand is introduced into the left side of the oral cavity of the patient. Once the gastric tube is negotiated into the oropharynx, it is pulled towards the lateral pharyngeal wall with the index finger, virtually grasping it between the index finger and the lateral pharyngeal wall. As the tube is pushed to the proximal end by the right hand, the left index finger simultaneously guides the tube along the lateral pharyngeal wall into the esophagus. The fingertip provides the buttress against the holes in the distal part of the gastric tube providing it the requisite sturdiness, preventing its bending and impaction with simultaneous steering into the esophagus.

Our method is akin to that reported by Bong and colleagues, which tends to keep the gastric tube adjacent to the lateral pharyngeal wall.[1] Our technique avoids some of the time consuming and technically demanding measures of failed gastric tube insertion. No lateral bending of the head, lateral neck pressure or anterior lifting of the thyroid cartilage is required.[1,3,4] These maneuvers may not be possible in patients with cervical spine trauma, cervical traction or in neck surgery, where our method can be used easily. Further, digital palpation of the feeding tube in the oral cavity almost obviates the need to check the entry of the gastric tube into the esophagus or its retention in the oropharynx, if any, by direct laryngoscopic examination. This technique has been used by us approximately 90 times over the past six months and was found to be successful approximately 83% of times it was used.

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Re-expansion pulmonary edema following laparotomy for volvulus

To the Editor:

Re-expansion pulmonary edema (REPO) is an uncommon life-threatening condition that results mostly from rapid drainage of long-standing pleural effusion or pneumothorax. We report yet another unusual cause of REPO in a surgical patient with acute abdomen.

A 24-yr-old autistic girl presented to the general surgeons with a two-day history of abdominal pain and distension. She deteriorated prior to surgery and was admitted as an emergency on the intensive care unit in extremis with severe hypoxemia and a tense grossly distended abdomen. Despite pre-oxygenation the SpO_2 did not rise above 85%. Tracheal intubation was successfully performed but lung compliance was extremely poor. Despite use of high inflation pressures with positive end-expiratory pressure, the SpO_2 deteriorated down to 60% on FIO_2 of 1.0. With the situation becoming desperate, a decompressive laparotomy was performed in the intensive care unit. Immediately the lung compliance improved pari passu with the SpO_2, which rose to 96%. However she developed pulmonary edema a few minutes later. She was immediately transferred to the theatre for a formal extended right hemicolectomy for a massive dilatation of transverse colon secondary to a volvulus.

She was electively ventilated postoperatively in the intensive care unit. Postoperative chest *x-ray* showed bilateral pulmonary shadowing. Gas exchange improved over the next 24 hr to allow ventilatory weaning and extubation.

Reported REPO from unusual causes include delayed repair of traumatic diaphragmatic hernia[1] and excision of extra-pleural lesions such as mediastinal tumours and giant hepatic cysts.[2,3] Clinical presenta-

tion ranges from asymptomatic chest radiological abnormalities to severe cardiorespiratory insufficiency and death. It is commonly ipsilateral (92.3%) and bilaterality worsens the prognosis. In about two thirds of reported cases REPO develops rapidly within an hour and typically occurs following lung collapse of three days duration or more.[4]

The pathophysiology is not completely understood. Mechanical and biological factors are probably involved in the pathogenesis of REPO as a consequence of ischemia of collapsed segments and their reperfusion. Using a rabbit model Sakao *et al.* have demonstrated an inflammatory process in segmental collapse and reperfusion.[5] Mechanically, lung re-expansion generates a negative perivascular pressure with a parallel rise in hydrostatic pressure due to vascular flooding with possible capillary damage.

We postulate in this patient that marked abdominal distension which gradually worsened over three days, resulted in significant bilateral lung collapse. On decompressing the abdomen the lungs expanded rapidly, akin to rapid drainage of a large pleural effusion, with consequential REPO.

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Pre-existing otitis media and hearing impairment after interscalene block combined with general anesthesia

To the Editor:

A 30-yr-old man presented with a traumatic injury of his clavicle. An upper respiratory tract infection was noted during the preanesthetic interview. Anesthesia was provided with a right interscalene plexus block, using a 22-G needle and nerve stimulation. A muscular response of the deltoid and biceps was easily obtained at 0.7 mA, and 30 mL of ropivacaine 0.75% were injected after negative aspiration. Horner's syndrome was not recorded and no complication occurred. Shoulder surgery was started after induction of general anesthesia using nitrous oxide in oxygen, propofol, sufentanil, and placement of a laryngeal mask airway. Upon emergence, the patient complained of a profound hearing loss of the ear ipsilateral to the block. This hearing loss was not associated with fullness in the ear, tinnitus, vertigo or headache. Immediate ear examination performed by an otorhinolaryngologist confirmed a bilateral serous otitis media with effusion and retracted tympanic membranes. Audiometric testing performed on the same day disclosed a conductive deficit (homogeneous decrease of 30 dB in all frequencies for air conduction with normal and symmetric bone conduction) and was normal for the opposite ear. Motor block lasted six hours, while hearing loss notably improved within a few hours after recovery of motor function and completely disappeared within four days. An audiogram repeated one month later showed complete recovery.

General anesthesia using nitrous oxide may have impacted on the movement of the tympanic membrane and thus the stapes.[1] However, we believe that regional anesthesia was the triggering factor, as hearing loss was experienced only ispislateral to the block while otitis media was detected bilaterally. One would have expected nitrous oxide to alter hearing bilaterally. Moreover, hearing recovered with the same time profile as the interscalene block. Rosenberg has described a case of hearing impairment after interscalene brachial plexus block anesthesia.[2] Sympathetic block-induced vasodilatation induced edema of the mucosal membranes of the Eustachian tube (ET) and the middle ear, thereby producing a hearing decrement on that side.

Otitis media also contributed to hearing impairment by obstructing the ET and by causing tympanic cavity mucosal edema.[3,4] In this patient with otitis media, vasodilatation of the ET and the tympanic cav-