Letters to Editor

An urgent brain computed tomography (CT) revealed no intracranial hemorrhage so, a diagnosis of acute ischemic stroke was made and intravenous thrombolysis was instituted. At 7 h after the attack, the patient became alert and oriented. Physical examination revealed right hemiparesis regression; a repeated brain CT scan revealed signs of ischemic edema localized to the left frontoparietal apical regions. The patient was re-scheduled for surgery six days later. As in the first instance, femoral and sciatic nerve block was induced with 600 mg of plain mepivacaine. Approximately 20 min later, the heart rate suddenly increased to 120 bpm and the blood pressure to 220/120 mm Hg. The patient became confused, with slurred speech and slow response to simple verbal commands, but remained conscious. Neither tonic-clonic activity nor major neurologic deficits were noted. Cardiovascular parameters and neurological signs normalized within 1 h after the incident.

According to available data, plain mepivacaine seems quite safe even at doses exceeding the maximum recommended. [1] Although the close temporal relationship between the onset of stroke signs and symptoms and the anesthetic procedure had prompted suspicions, we initially ascribed the stroke to a perioperative event and we attributed them to the unconsciousness and the highly elevated blood pressure.[2] It was only after the second instance that we realized we faced two episodes of mepivacaine toxicity. In retrospect, we speculate that the ischemic stroke was a consequence of a hypertensive crisis due to systemic absorption of a potential mepivacaine toxic dose.[3]

Local anesthetic intoxication, mepivacaine in particular, can increase both, blood pressure and heart rate as consequence of sympathetic nervous system activation.[4] In this setting, metoprolol could have behaved as a non-selective ß-adrenergic blockade resulting in unopposed α-adrenergic activity producing vasoconstriction and increase in systemic vascular resistances.[5] Pre-existing uncontrolled hypertension could have acted as a superimposed factor and all mechanisms together might explain the rapid onset of exceptionally high blood pressure. Unfortunately, serum mepivacaine concentrations, which could have provided clearer answers about the possible mepivacaine overdosage, were not measured in our patient.

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Manufacturing defect of endotracheal tube connector: A cause of airway obstruction

Sir,
Preuse check of the endotracheal tube (ETT) is an integral part of anesthesia practice. Despite this common practice, some of the manufacturing defects not perceptible on visual inspection can go unnoticed, leading to complete or partial airway obstruction.[1] Such errors can be catastrophic if corrective steps are not instituted timely, especially in pediatric patients.

We report one such case of ETT connector defect recently encountered in the pediatric operation theater, which resulted in inadequate ventilation. A 7 month old, American Society of Anesthesiologists physical status 1 child, weighing 8 kg was posted for bilateral inguinal herniotomy. Following induction of anesthesia, the airway of the patient was secured with a 4.0 mm single use ETT (Sterimed Medical Devices Ltd., Bahadurgarh, Haryana, India). On connecting to the anesthesia circuit, the ventilation was difficult, and the compliance of the bag was poor. The air entry was grossly diminished. There
was an increase in the peak pressures and the end-tidal carbon dioxide (EtCO₂). To rule out any mechanical obstruction as the plausible cause, the anesthesia circuit was checked for any kink or obstruction. The problem was diagnosed when a 10 F suction catheter could not pass through the ETT. The distal end of the connector was found to have an extremely narrow orifice, in comparison to the standard 4 mm ETT connector [Figure 1]. The connector was removed and replaced with another same size ETT connector. There was immediate improvement in ventilation. The peak pressures and the EtCO₂ came down to normal limits. The rest of the surgery was uneventful.

Difficult ventilation following successful endotracheal intubation could be due to acute bronchospasm, malfunction or obstruction of breathing circuit, kinking, obstruction of ETT by foreign body or malfunctioning defects of different parts of ETT e.g., inflation line, ETT connector.[2-5]

In spite of the several case reports of manufacturing errors, such mishaps continue to occur. Reporting of such critical events re-emphasizes the need for thorough check of each and every part of the anesthetic equipment prior to its use and highlights the role of a vigilant clinician in timely detection of such errors and thereby avoiding anaesthetic mishaps.

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**Figure 1**: The narrow orifice endotracheal tube connector and the standard connector

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Sir,

Nasogastric tube (NGT) insertion is done routinely in abdominal surgeries. However, it can be really difficult in some cases such as head injury and facial trauma, esophageal narrowing, heavy built patients, and intubated patients.[1] Various complications such as endotracheal insertion, intracranial insertion, blockade, and nasal bleeding have been reported.[2] It is not uncommon for NGT to coil around into a knot in the pharynx; but, we came across a case where malpositioned NGT coiled around the endotracheal tube (ETT).

We report a case of a 60-year-old male posted for perforation peritonitis for which NGT was inserted, which coiled around the ETT and formed a knot. Literature on the same reveals that it is a rarest of the rare complication, which can be life-threatening.

A 60-year-old male, American Society of Anesthesiologists physical Status II was admitted as a case of perforation peritonitis.