Breath-holding in Vitamin D deficiency rickets: A dilemma for the anesthetist

Sir,

Vitamin D deficiency rickets presents profound anesthetic concerns ranging from impaired neuromuscular function, risk of fractures, difficult airway to electrolyte imbalance causing laryngospasm, seizures, arrhythmias, and tetany.[1] Breath-holding secondary to hypocalcaemia is rare and mostly seen in neonates or infants and hardly reported in older children.[2,3]

Two children of 3 months and 8 years, diagnosed previously as Vitamin D deficient rickets on daily calcium, phosphorus, and monthly Vitamin D$_3$ depot injections, were posted for herniotomy with orchidopexy. Both gave a history of breath holding spells associated with cyanosis and drowsiness but recovering immediately without active interventions. Records revealed need for hospitalization and intravenous calcium gluconate during one such episode in the older child at 4 months of age. His clinical assessment prior to surgery showed craniosynostosis, pigeon chest, genu varum, frontal and parietal bossing, serum calcium of 8.1 mg/dL (normal 8.8-10.8), and phosphate 4.8 mg/dL (normal 4.5-5.5). The infant’s examination was unremarkable but calcium and phosphate were 7.2 mg/dL and 4.0 mg/dL. In view of anticipated difficult airway, general anesthesia with endotracheal intubation for the child and laryngeal mask airway for the infant along with ilioinguinal, iliohypogastric nerve block was planned. The intraoperative period was uneventful. Twenty minutes into the postoperative period, the older child’s respiration became irregular with diminished chest expansion and brief apnea of 10-15 s, and drop in oxygen saturation to 91%; however, cyanosis, dyspnea, stridor, or loss of muscle tone was not observed. The episode repeated after 10 and 20 min. First, oxygen was supplemented via facemask at 6 L/min that improved the saturation to 98% briefly. Common causes of breath holding in postoperative period such as laryngospasm, opioid overdose, seizures, inadequate reversal, and analgesia were excluded. When subsequent episodes occurred and saturation dropped while still on oxygen, considering hypocalcemia as a probable cause 10 ml of 10% calcium gluconate was administered over 10 min. After discussing with pediatrician, Vitamin D$_3$, 6 lakh units was also given. After these interventions, saturation improved and there was no relapse. In view of the emergency, calcium gluconate was administered immediately without waiting for a serum calcium value. However, once the child was stable, blood investigation revealed serum calcium of 8.3 mg/dL. We accepted this almost low normal value as his symptoms had subsided, noting his preoperative baseline value of 8.1 mg/dL and keeping in mind his underlying disease pathology. Intubation was deferred as the episodes though recurrent, lasted for only 20 s. He was observed for 8 h, shifted after ensuring regular breathing. He was discharged the following day with oral supplements and a follow-up after 1 month revealed normal values of serum calcium. The infant had an uneventful hospital stay.

Breath-holding in rickets may be seen in infants as reported by Buchanan et al. where hypocalcaemia, accentuated by stoppage of calcium produced recurrent apnea that necessitated intubation and intensive care in a seven-week baby who later responded dramatically to intravenous calcium. [3] Bonnici added that this line of treatment with intravenous calcium alone produces transient recovery and should be supplemented with regular Vitamin D supplements. [2] The complex relationship between low calcium and apnea may be attributed to increase in production of sleep-regulating substances such as tumor necrosis factor-α and prostaglandin D$_2$ in Vitamin D deficiency. Airway hypotonia causing obstructive sleep apnea has also been postulated. [4] Knowledge of this complication is important in postoperative period to anesthetists especially in developing countries where rickets is endemic. [5] However, ruling out other causes of postoperative apnea is prudent. Early identification and initiation of treatment at young age may avoid problems as seen in the infant who had an uneventful perioperative period.

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Sir,

I sincerely appreciated reading “spinal anesthesia and direction of spinal needle bevel” written in response to my letter “subarachnoid space needle manipulations for a successful block.” The response adequately highlighted the differences between Quincke needle (cutting tip type) and Whitacre needle (pencil point tip type) in regard to the mechanics of exiting local anesthetics' flow from these needles. However, my letter was emphasizing about the position of the exit-point (eyelet in case of Whitacre needle and terminal end in case of Quincke needle) in respect to the overall lumbar cistern in the transverse plane. The flow and direction of exiting local anesthetics are secondary objectives because the primary objective is to ensure that the exit-point of blindly placed subarachnoid needle is in the center of the cerebrospinal fluid (CSF) filled lumbar cistern. This is to ensure high success rates of effective subarachnoid anesthesia as well as to avoid delivery of local anesthetics through needle's exit-point that could have been inadvertently/blindly placed near to/within the intrathecal/extradural nerve roots because this unwarranted proximity to these nerve roots may cause partial-to-no subarachnoid anesthesia with potential direct exposure of these nerve roots to high concentrations of local anesthetics that may present as postanesthesia complications of transient-to-long-term neurological deficits. As subarachnoid needle placement is a blind procedure and the exit-point can end up being in one of the dural-arachnoid side-walls of this lumbar cistern as visualized, it is important to realize that re-directioning when performed in calculated manner can provide better success rates by ensuring the exit-point of the needle and subsequent delivery of medications in lumbar cistern's central pool of CSF rather than away from it or outside it. There is a difference in diagnostic lumbar puncture versus subarachnoid anesthesia because the diagnostic lumbar puncture's goal is to just collect CSF irrespective of its flow rates whereas subarachnoid anesthesia requires that appropriate CSF flow rate/aspiration should be ensured before injecting medications, otherwise the goal of successful block will not be achieved. My letter had tried to focus on this aspect only. As mentioned in my letter at the beginning of the third paragraph, the subarachnoid needle manipulations advised in my letter are not meant for Quincke needle because the Quincke needle has cutting tip with no true “eyelet” and medication exits from its terminal end itself. Moreover, as the cutting edge of Quincke needle is completely open on one side with opening extending/slanting to both side-edges of needle’s exit-point, it becomes improbable to ensure the guidance of calculated re-directioning with Quincke needle as advised in my letter. Comparatively, the eyelet in Whitacre needle is rectangular and restricted to only one of the four “surfaces” of conical (approximately curvilinear rectangular) shaped pencil tip thus allowing the guidance aimed to be achieved by calculated re-directioning advised in my letter. In summary, the points raised by the respondent author are valid in regards to Quincke needle rotations while performing subarachnoid block; however, it is my limited understanding that calculated re-directioning can be performed with Whitacre needle only wherein the aim can be achieved by calculated re-directioning advised in my letter.