Management of laryngeal mask airway induced hiccups using dexmedetomedine

Sir,

An 38-year-old female patient, weighing 55 kg, a known hypertensive under good control with medication, American Society of Anaesthesiologists (ASA) grade 2 was posted for fibroadenoma excision under general anaesthesia with laryngeal mask airway (LMA). She was pre-medicated with 100 µg fentanyl IV and 0.2 mg glycopyrrolate IV. Induction was with 120 mg propofol IV and 80 mg of succinvlcholine IV, followed by LMA (#3) insertion. Anaesthesia was maintained with 2-2.5% sevoflurane in 60% nitrous oxide and 40% oxygen, and spontaneous ventilation. Thirty minutes after induction, she developed hiccups, which continued for 5 min despite repeated boluses of propofol 10 mg, upto a total of 50 mg. Then, 50 µg of dexmedetomidine was given IV over 10 min, following which the hiccups ceased. There was fall in heart rate and blood pressure, but not more than 20% of baseline. Thereafter, the surgery continued uneventfully and the patient was shifted to the post-operative care unit. There was no delay in recovery. She had no hiccups in the post-operative period. When enquired, she gave no history of hiccups in the past.

Hiccups are involuntary contractions of the diaphragm and intercostal muscles. They occur with a sudden inspiration, immediately followed by active closure of the glottis.[1] The hiccup reflex is comprised of afferent pathways-vagal, phrenic, and sympathetic (T6-12) branches. The efferent pathways are composed of the phrenic nerve to the diaphragm and nerves to the glottis and the external intercostal muscles. The central connection is the spinal cord (C3-5), possibly controlled by supraspinal pathways.^[2,3] A stimulation at any of the above afferent pathways can trigger hiccups. In our case, the stimulus for hiccups may have been stimulus caused by cuff inflation, pain at the surgical side, or decreased depth of anaesthesia. The precise mechanism(s) of action of dexmedetomidine in the suppression of hiccups in our case is not known. Nevertheless, we postulate a possible mechanism of action for the suppression of hiccups by dexmedetomidine. α_{a} adrenoreceptors are found in the central and peripheral nervous systems and in the autonomic ganglia at both pre- and post-synaptic sites. Stimulation of pre-synaptic receptors in sympathetic nerve endings inhibits release of norepinephrine, while central post-synaptic receptor

stimulation inhibits sympathetic activity. Stimulation of α_2 adrenoreceptors in the spinal cord produces analgesia. This inhibition of sympathetic activity (one of the pathways of the reflex arc in the hiccups pathway) along with analgesic and sedative properties may have been the reason for suppression of hiccups. Because the other agents purported for use in hiccups, i.e., atropine and ephedrine, may cause unacceptable elevations in heart rate and blood pressure levels, we propose the use of dexmedetomidine. Also, dexmedetomedine does not promote gastroesophageal reflux, by itself, is an etiological factor for hiccups.

Thus, dexmedetomedine, by the virtue of its sympatholytic, analgesic, and sedative properties, could be beneficial for intraoperative hiccups. Because intraoperative hiccups are a rare event, a randomized controlled trial would be difficult to conduct. Although further clinical investigation is needed, we conclude that dexmedetomedine may be useful in the treatment of hiccups after LMA insertion. The dose used by us was empirical. The exact dose may be determined after larger studies, which may be difficult in view of the rare and unpredictable occurrence of this phenomenon.

Chethan Manohara Koteswara, Jitendra Kumar Dubey

Department of Anaesthsiology, AJ Institute of Medical Sciences, Kuntikan, Mangalore, Karnataka, India

Address for correspondence:

Dr. Chethan Manohara Koteswara, Department of Anaesthesiology, A J Institute of Medical Sciences, Kuntikana, Mangalore-575 004, Karnataka, India. E-mail: major.chethan@gmail.com

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