Hyperventilation syndrome after general anesthesia

To the Editor,

Hyperventilation syndrome (HVS) is defined as breathing in excess of that required to maintain normal blood PaO_2 and $PaCO_2$. The symptoms are dyspnea, carpo-pedal spasm, numbness of the extremities, tachycardia, and unconsciousness etc. HVS is an acute complication sometimes encountered in dentistry.[1] We experienced two patients of hyperventilation attack after awaking from general anesthesia in oral and maxillofacial surgery.

A 16-year-old healthy man was scheduled for extirpation of tongue benign tumor under general anesthesia. Preoperatively, he had had an extreme anxiety for operation. Anesthesia was induced with intravenous remifentanyl, propofol, and the trachea intubated after administration of rocuronium bromide. Anesthesia was maintained with oxygen, air, sevoflurane, and remifentanyl. Supplemental flurbiprofen axetil was administered before 30 min of extubation. The procedure was finished safely in 40 min. Anesthesia was uneventful. His respiration rate suddenly increased to 40 breaths/min after 20 min of extubation. His consciousness was clear, but seemed to be very anxious and complained of breathlessness and numbness of the extremities, but had no pain. Carpopedal spasms were also observed. Heart rate was about 90 beats/min. We diagnosed his condition as HVS and administered midazolam 1.0 mg. The hyperventilation disappeared, but his respiration was depressed.

The second patient, a 50-year-old woman, was scheduled for tumor extirpation of floor of the mouth under general anesthesia. She had had the medical history of HVS after general anesthesia 23 years ago. She was not asthmatic. Anesthesia was induced with intravenous remifentanyl,

propofol, and the trachea intubated after administration of rocuronium bromide. Anesthesia was maintained with oxygen, air, sevoflurane, and remifentanyl. The procedure was finished safely in 150 min. Anesthesia and operation were uneventful. Supplemental flurbiprofen axetil was administered before 30 min of extubation. After extubation, she awoke and stated that she had no pain. 5 min after extubation, her heart rate suddenly increased from 80 beats/min to 130 beats/min, and her respiration rate simultaneously increased to 50 breaths/min. Her blood pressure was 160/85 mmHg. She complained of breathlessness and numbness of the extremities. We diagnosed her condition as HVS, and administered a beta-adrenergic blocker, propranolol hydrochloride 1.0 mg intravenously. As soon as her heart rate fell to 80 beats/min, the hyperventilation stopped.

Anxiety is the main cause of HVS. Thus, benzodiazepines including midazolam or diazepam are used as the first choice drug for the treatment of HVS like the first case. However, propofol may be not effective for HVS owing to its excitatory effect.[2] Although postoperative pain also becomes the cause of HVS, these patients complained no pain owing to postoperative analgesia by the administration of flurbiprofen axetil and local infiltration anesthesia. We have previously reported three cases with HVS in dentistry that were improved or prevented by the administration of a beta-adrenergic blocker,[3] because HVS reflects the enhancement of sympathoadrenal tone.[4] The second case could be managed using propranolol irrespective of psychosedation. We believe that a beta-adrenergic blocker having no respiratory depression may be effective for HVS patients who extremely sensitive to sympathoadrenal tone. However, more clinical studies are required to confirm the usefulness of a beta-adrenergic blocker for HVS after general anesthesia.

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