Cases of Esophageal Syncope during an Early Postoperative Period

Kenji Nishioka MD (Assistant Professor) a
Sadayo Niiya MD (Chief Anesthesiologist) b
Kanna Ogata MD (Staff Anesthesiologist) b
Kyoko Motokawa MD (Staff Anesthesiologist) b
Koji Sumikawa MD (Professor) a

aDepartment of Anesthesiology, Nagasaki University School of Medicine, Nagasaki 852-8501, Japan
bDepartment of Anesthesiology, Isahaya Health Insurance General Hospital, Isahaya 854-8501, Japan

Address correspondence to: Kenji Nishioka
Department of Anesthesiology, Nagasaki University School of Medicine,
1-7-1 Sakamoto, Nagasaki 852-8501, Japan
Phone: 81-95-819-7370, Fax: 81-95-819-7373
E-mail: k-24oka@nagasaki-u.ac.jp
To the Editor:

Esophageal syncope (ES) is the loss of consciousness upon swallowing or vomiting, which is considered to be due to hypersensitive vagal responses. Although mortality risk appears to be low, it is potentially a life threatening event [1, 2]. An increasing number of reports in the literature, and the diagnosis of 5 cases in one Canadian hospital over a 4-year period, suggest that ES may be more common than thought [2]. Postoperative ES was reported to have occurred in the relatively late postoperative period after cardiovascular surgery [3, 4]. We describe 4 cases of this type of neurally mediated syncope observed in the early postoperative period. This case series occurred in a community hospital from August 2003 to October 2008, with a total of 3400 patients receiving general anesthesia.

Case #1

A 56-year-old woman underwent mastectomy. She had a long-standing PQ prolongation (0.24 sec of PQ interval) in electrocardiogram (ECG) preoperatively, but had neither symptoms nor currently taking medications. Atropine, 0.5 mg, was given as premedication. General anesthesia was induced with thiamylal and maintained with sevoflurane, nitrous oxide, fentanyl (a total dose of 300 µg), and vecuronium, which was reversed with neostigmine and atropine. The operation was performed
uneventfully, and anesthesia time was 192 min. Approximately 9 hours after the operation, the patient vomited and then lost consciousness. She recovered consciousness after a minute in response to nurse’s calls and tapping. Her ECG revealed Mobitz II block with a short period of ventricular asystole after the vomiting. Three hours later, the same phenomenon was observed, and then temporary transvenous pacing was started (mode: VVI; rate: 40 bpm). On the postoperative day (POD) 1 and 2, a short period of pacing rhythm after vomiting was observed a few times a day. The serum potassium level ranged from 3.9 to 4.4 mEq/L during this period. Thus, an internal pacemaker (VVI; rate: 50 bpm) was implanted on POD 3. Ten days after the implantation, the pacing rhythm was 0.6 % of total beats. She was examined every 6 months for 3 years, and the incidence of pacemaker discharge was 2 - 3 % of the total beats with no syncopal episode.

Case #2

A 50-year-old woman underwent hysterectomy. She had a history of colectomy 5 years ago, when she had a single episode of AV block associated with vomiting during the night of POD 0, which we failed to notice before the current operation. After insertion of an epidural catheter for postoperative pain management, general anesthesia was induced with thiamylal and maintained with sevoflurane, remifentanil and rocuronium. The operation was uneventful, and anesthesia time was 122 min. She
received continuous epidural anesthesia with ropivacaine, 4 mg/hr, and fentanyl, 6 µg/hr, which controlled postoperative pain effectively. Postoperative nausea and vomiting (PONV) were not observed throughout POD 0. However, in the early morning of POD 1 (14 hours after operation), she complained of pain and nausea, for which flurbiprofen and metoclopramide were administered. She vomited despite the treatment. While a nurse cared for her emesis, she lost consciousness, and her ECG monitor gave the alarm of asystole. Ventricular asystole associated with Mobitz II block was observed for about 15 sec (Fig. 1a). Thus a temporary transvenous pacing was started (VVI; rate: 40 bpm). The epidural catheter was removed to rule out adverse effects of epidural anesthesia. She still experienced nausea and vomiting when she moved. Every vomiting episode was followed by artificial pacing (Fig. 1b), which was observed frequently on POD 1, and a few times a day from POD 2 to POD 4. The potassium level ranged from 3.8 to 4.5 mEq/L during this period. No pacing rhythm was recorded after POD 5, and the pacing catheter was removed on POD 7. She was followed up for 8 months without syncopal episodes.

Case #3

A 69-year-old man underwent radical prostatectomy. He had no history of cardiovascular disease or medication preoperatively. General anesthesia was induced and maintained with propofol, remifentanil and rocuronium. The operation was
uneventful, and anesthesia time was 205 min. He received fentanyl, 200 µg i.v., against postoperative pain at the end of operation. He had no PONV throughout POD 0. In the early morning of POD 1 (16 hours after operation), he received pentazocine for pain control. He then vomited when he sat up in bed. While a nurse cared for the emesis, ECG revealed Mobitz II block for a few seconds (Fig. 2). Metoclopramide and diphenhydramine were administered as antiemetics, and he did not complain of nausea thereafter. ECG monitoring was continued throughout POD1, followed by application of Holter ECG. The serum potassium level ranged from 3.9 to 4.1 mEq/L during this period. There were no abnormal findings in Holter ECG checked at his discharge on POD 10.

Case #4

A 74-year-old woman underwent gastrectomy. She had hypertension and hyperlipidemia controlled with medication preoperatively. After insertion of an epidural catheter for postoperative pain management, general anesthesia was induced with thiamylal and maintained with sevoflurane, remifentanil and rocuronium. The operation was uneventful, and anesthesia time was 244 min. Muscle relaxant was reversed with neostigmine and atropine. She received continuous epidural anesthesia with ropivacaine, 4 mg/hr, and fentanyl, 8 µg/hr, which controlled postoperative pain effectively. She suddenly lost consciousness after vomiting at 10 hours after the
operation. ECG revealed Mobitz II block, and a 20-sec period of missed beat (Fig. 3a). She recovered consciousness in a minute in response to nurse’s calls and tapping. After this event, she complained of slight headache without nausea. Dopamine was continuously infused to prevent further cardiac events. Another short period of Mobitz II block with syncope was observed after vomiting in the morning of POD 1 despite dopamine infusion (Fig. 3b). Since she did not complain of nausea thereafter, temporal pacing was not applied. The serum potassium level ranged from 3.8 to 4.4 mEq/L during this period. The epidural catheter was removed, and dopamine was discontinued on POD 4. Although she vomited several times with the increase in dietary intake after POD 8, she had no further syncopal episodes.

All of these patients had stable vital signs postoperatively, and there was no anemia or electrolytic abnormality. The events of AV block were observed frequently in the first two cases with difficulty in controlling PONV, whereas the other had only a few events. The ECG monitor gives the alarm with in 5 seconds after asystole, and any of the alarms did not precede the vomiting episodes. Vomiting occurred despite back-up pacing in cases #1 and #2. These examples confirm that the vomiting would be the cause but not the effect of AV block.

The mechanism of ES is not fully understood, and the present cases could not provide ample evidence to show that vomiting induced Mobitz II block. Nevertheless,
several lines of evidence suggest that certain central neurotransmitters, such as beta-endorphin and serotonin, may play a role in eliciting or facilitating these reflexes [1]. It seems possible that certain stressors, e.g., pain, anxiety and hypovolemia, and/or analgesics, e.g., opioids and epidural anesthesia, would affect the reflex arc in the central nervous system (CNS) in the postoperative period. Additionally, balloon inflation in the esophagus reproduced rhythm disturbances in most of the reported cases of ES [2], and there was also a case report that Mobitz II block was reproduced by aspiration of oral cavity during recovery from anesthesia [5]. In the present case series, nausea alone did not cause AV block, and thus the peristaltic activity of the esophagus during vomiting would have caused afferent vagal stimulus triggering the reflex arc.
References


Legends

Figure 1
a. The record of ECG in case #2 during the loss of consciousness after vomiting at 14 hours after the operation. It had been normal sinus rhythm before this event, and the ECG alarm went off when the staff had cared for the emesis.

b. A record of pacemaker rhythm just after vomiting in case #2. It shows the pace maker backed up the beat without delay.

Figure 2
The record of ECG just after vomiting at 16 hours after the operation in case #3. The alarm of ECG did not precede the vomiting episode.

Figure 3
The records of ECG during the loss of consciousness after vomiting at 10 hours (a) and 18 hours (b) after the operation in case #4. The marks of “?” indicate the tapping of the patient by a nurse to restore consciousness.
Figure 3