
Clinical Report

Complete atrioventricular block during anesthesia

Kikyo Mamiya MD,
Jun Aono MD,
Masanobu Manabe MD

Purpose: To describe a case of asymptomatic first degree atrioventricular block with a bifascicular block that progressed to complete atrioventricular block during anesthesia. This potentially fatal block was successfully treated with transesophageal ventricular pacing.

Clinical features: A 67-yr-old man was scheduled for microvascular decompression of the right trigeminal nerve under general anesthesia. His preoperative ECG showed first degree atrioventricular block with complete right bundle branch block and left anterior hemiblock, but he had experienced no cardiovascular symptoms. Anesthesia was induced with sevoflurane 5%, and maintained with isoflurane 1.5-2% in oxygen. Fifteen minutes later in the left lateral decubitus position, the systolic arterial blood pressure suddenly decreased from 80 mmHg to 0 mmHg. Then, the ECG abruptly changed from sinus rhythm to complete atrioventricular block. The heart was unresponsive to drug therapy such as atropine 1.3 mg and isoproterenol 0.5 mg, or transcutaneous pacing but transesophageal pacing was successful.

Conclusion: Asymptomatic first degree atrioventricular block with bifascicular block advanced to complete atrioventricular block during anesthesia. The block was successfully managed with transesophageal pacing.

Objectif : Décrire le cas d'un bloc auriculoventriculaire asymptomatique du premier degré qui s'est développé en un bloc auriculoventriculaire complet pendant l'anesthésie. Ce bloc d'issue fatale possible a été traité avec succès par l'entraînement ventriculaire transœsophagien.

Éléments cliniques : Un homme de 67 ans a été admis pour une décompression microvasculaire du nerf trijumeau droit sous anesthésie générale. L'ECG préopératoire montrait un bloc auriculoventriculaire du premier degré avec un bloc de branche droit complet et un hémibloc antérieur gauche, mais sans symptômes cardiovasculaires. L'anesthésie a été induite avec 5 % de sévoflurane et maintenue avec 1,5-2 % d'isoflurane dans de l'oxygène. Quinze minutes plus tard, en décubitus latéral gauche, la tension artérielle systolique a soudainement chuté de 80 mmHg à 0 mmHg. Le rythme sinusal de l'ECG a alors passé abruptement en bloc auriculoventriculaire complet. Le coeur n'a pas répondu aux médicaments, 1,3 mg d'atropine et 0,5 mg d'isoprotérénol, ou à l'entraînement transcutané, mais l'entraînement transœsophagien a réussi.

Conclusion : Le bloc auriculoventriculaire asymptomatique du premier degré avec un bloc bifasciculaire est devenu un bloc auriculoventriculaire complet pendant l'anesthésie. Le bloc a été traité avec succès par l'entraînement transœsophagien.

From the Department of Anesthesiology, Kochi Medical School, Kochi, 783-8505 Japan.

Address correspondence to: Kikyo Mamiya MD, Phone: 888-80-2471; Fax: 888-80-2475; E-mail: mamiyak@med.kochi-ms.ac.jp

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PATIENTS with potential complete-atrioventricular block (AVB) require temporary insertion of an intravenous pacemaker before anesthesia. Bifascicular block with first degree-AVB (I° -AVB) is considered to be an exception, because, when asymptomatic, it rarely advances to complete-AVB during anesthesia and the hazards of pacemaker insertion such as cardiac perforation must be considered.^{1,2}

In the present case, asymptomatic I° -AVB with a bifascicular block advanced to complete-AVB after a hypotensive episode during anesthesia. The heart did not respond to either drug therapy or to transcutaneous pacing, but did respond to transesophageal ventricular pacing.

Case Report

A 67-yr-old, 67-kg man (ASA 2), was scheduled for microvascular decompression of the right trigeminal nerve. He had been treated with carbamazepine (350 mg per day) and loxoprofen (180 mg per day) for trigeminal neuralgia.

His past medical history was unremarkable except for cervical laminectomy performed under general anesthesia at the age of 59 yr. Preoperative ECG showed I° -AVB (PR interval=0.22 sec) with complete right bundle branch block and left anterior hemiblock (Figure (A)). The patient had no cardiovascular symptoms and he was not taking any cardiovascular medication.

Preoperatively 10 mg diazepam *po* was administered one hour before induction of anesthesia. Upon arrival in the operating room, the heart rate was 75 bpm (sinus rhythm) and arterial blood pressure was 120/60 mmHg. Standard monitors, including ECG, temperature, pulse oxymeter, and end-tidal capnography, were used.

After 0.2 mg atropine *iv*, anesthesia was induced with inhalation of sevoflurane 5% in oxygen by a mask. Due to limited neck flexibility, with the help fiberoptic bronchoscopy, tracheal intubation was performed without muscle relaxants. After placement of the tracheal tube, anesthesia was maintained with isoflurane 1.5-2% in oxygen. A catheter was placed in the right radial artery to monitor blood pressure continuously. The patient was placed in the left lateral decubitus position for surgery with the noninvasive transcutaneous cardiac pacing electrodes on the chest and back.

Fifteen minutes later, the systolic arterial blood pressure suddenly decreased from 80 to 50 mmHg. Within one minute, the arterial pressure wave disappeared from the monitoring screen. The ECG abruptly changed from sinus rhythm (55 bpm) to complete-AVB (Figure (B)).

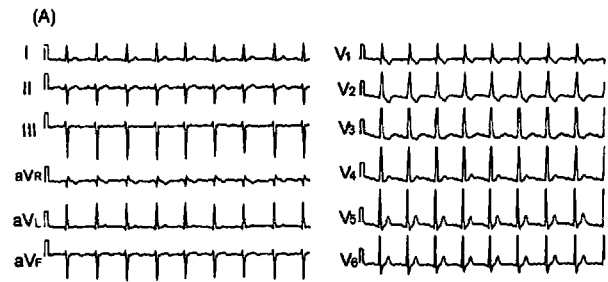


FIGURE (A) Preoperative ECG showing first degree atrioventricular block (PR interval = 0.22 sec) with complete right bundle branch block and left anterior hemiblock.

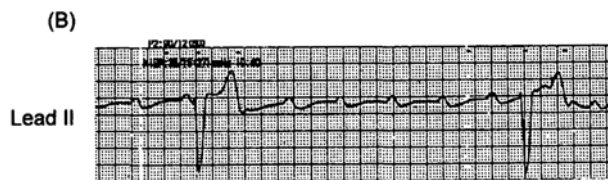


FIGURE (B) ECG showing complete atrioventricular block.

Isoflurane was terminated. The heart did not respond to 0.3 mg atropine or 12 mg ephedrine *iv*. The heart was unresponsive to external transcutaneous pacing, at 80 bpm, with a threshold of 60 mA and then, 80 mA (Cardiopac® 3M33, NEC medical systems, Japan) and there was no response to 1 mg atropine or 0.5 mg isoproterenol *iv*.

The patient was placed in the supine position, chest compression was started. About two minutes later, transesophageal ventricular pacing was commenced, at 70 bpm, threshold of 25 mA, and a depth of insertion of 37 cm (Dual Chamber Temporary Pacemaker, Model 5388, Medtronic, Inc., USA). This restored the arterial blood pressure to 130-150/70-80 mmHg. The transesophageal pacemaker was replaced with a transvenous pacer, to obtain reliable ventricular pacing with less electric energy.

Three hours after the episode, the patient had recovered from anesthesia without further complication. The ECG showed I° -AVB, and he did not require further pacing support.

Discussion

Asymptomatic I° -AVB with bifascicular block rarely progressed to complete-AVB perioperatively.³ Therefore, the routine prophylactic insertion of a tem-

porary pacemaker has been questioned in such patients.^{1,2} Our case, however, suggests that hypotension and an increase in vagal tone can cause complete-AVB in a patient with asymptomatic I°-AVB combined with bifascicular block. Sudden hypotension may have caused ischemia in the AV node in this case. The left lateral decubitus position may have enhanced vagal tone,⁴ slowed conduction of the AV node. This combination of ischemia at the AV node and increased vagal activity may be caused the complete-AVB.

In this case, transcutaneous pacing had been prepared, but it was ineffective. A previous report suggested that failure to capture with transcutaneous pacing may be related to electrode placement or to a barrel-shaped chest.⁵ However, we placed pacer electrodes correctly and this patient did not have barrel-shaped chest. It is uncertain why transcutaneous pacing was ineffective. Drugs such as atropine and isoproterenol also were ineffective. Thus, transesophageal pacing was tried which was successful.

Transesophageal pacing is usually recommended only for atrial pacing, although previous reports have demonstrated that it may be applied for ventricular pacing.⁶ The present case showed that this pacing method may be applicable in acute complete-AVB occurring during anesthesia. The technique is minimally invasive and the catheter is easy to insert, and may be useful in the perianesthetic care of patients with asymptomatic but potentially complete-AVB.

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