Periodic Isorhythmic Dissociation during Enflurane Anesthesia in a Patient with Sinus Bradycardia

Toshiaki NISHIKAWA and Shuji DOHI

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Isorhythmic dissociation is not uncommon during inhalational anesthesia[1]. But a few reports are available, and a report described that during enflurane anesthesia isorhythmic dissociation occurred in 5 out of 12 subjects (42%)[2]. The etiology of this rhythm disturbance remains unclear, as does its treatment[3]. We experienced a case in which isorhythmic dissociation developed several times in a patient with sinus bradycardia during enflurane anesthesia. The conversion from sinus rhythm to isorhythmic dissociation occurred periodically in association with acceleration of heart rate due to surgical stimulation or following intravenous atropine. Although isorhythmic dissociation has been recognized to disappear spontaneously and reappear during anesthetic course[1], we believe that this periodical occurrence has not been detailed in the literature.

Report of a Case

A 64-year-old, 62.5 kg man was scheduled for removal of left parotid tumor. He was otherwise healthy except a previous history that he had once suffered from a syncopal attack in a bathroom more than 40 years before. Since then, however, he has been in a good health without such an episode. His preoperative electrocardiogram and ward records after admission revealed sinus bradycardia with a rate of 48–60 beats·min^{-1}. Other laboratory data were within normal limits.

Premedications consisted of diazepam 10 mg, per os, 90 min before arrival in the operating room. Before anesthesia induction, his blood pressure was 106/76 mmHg with a normal sinus rhythm of 50 beats·min^{-1} (fig. 1-A). Anesthesia was induced with intravenous thiamylal, 300 mg, the lungs were ventilated with pure oxygen, and the trachea was intubated with intravenous succinylcholine, 60 mg. The blood pressure rose to 172/124 mmHg immediately after tracheal intubation, whereas only a 10 beats increase in heart rate was noted. Thereafter, anesthesia was maintained with enflurane 1.5–2.5% inspired and 67% nitrous oxide in oxygen under controlled ventilation. During these procedures, normal sinus beats were confirmed by continuous monitoring of the electrocardiogram.

After the initiation of surgery, the P wave disappeared periodically in association with acceleration of heart rate due to surgical stimulation. Namely, the dissociated P wave approached the QRS complex and disappeared within it for 10–20 min (fig. 1-B). Conversely, the P wave that submerged within the QRS complex or T wave reappeared and preceded the QRS gradually on the electrocardiogram, thus regaining a previous sinus rhythm during the deceleration

Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Japan

Address reprint requests to Dr. Nishikawa: Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba City, Ibaraki, 305 Japan

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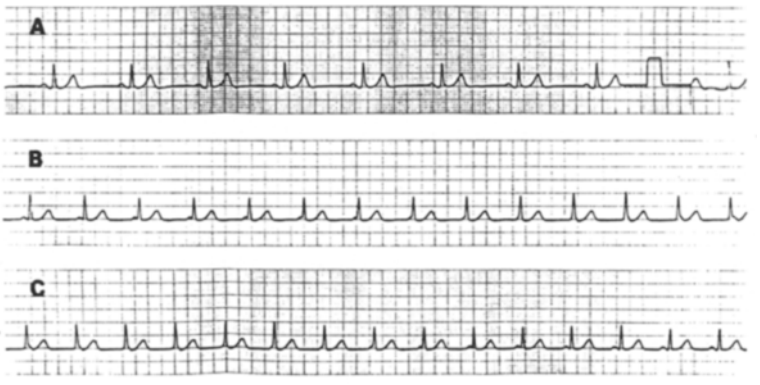

Fig. 1. Electrocardiograms before and during enflurane, nitrous oxide and oxygen anesthesia.

A: Sinus bradycardia before general anesthesia. B: Appearance of isorhythmic dissociation. C: Conversion to sinus rhythm.

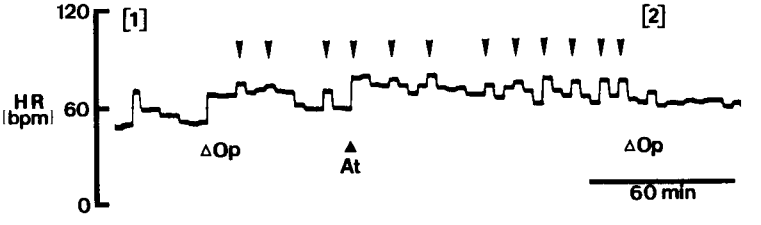

Fig. 2. Changes in heart rate (HR) during general anesthesia.

1: anesthesia induction and tracheal intubation. 2: emergence and extubation. Op: start and end of operation. At: intravenous atropine, 0.6 mg. Periodic occurrences of isorhythmic dissociation associated with acceleration of HR, indicated by arrows.

of heart rate (fig. 1-C).

During sinus rhythms, atropine, 0.6 mg, was given intravenously as a bolus in order to clarify the effect of vagal activity upon the heart rate and sinoatrial node as a pacemaker. Heart rate increased from 60 to 83 beats·min^{-1} following atropine injection (fig. 2), resulting in isorhythmic dissociation and an increase in blood pressure of 10 mmHg. Then, the heart rate gradually decreased to 76 beats·min^{-1} a few minutes later, and the heart resumed a normal sinus beat. Such a periodic withdrawal of sinoatrial nodal pacemaking rhythmicity was observed several times in association with acceleration of heart rate in the remaining anesthetic course (fig. 2). The period of oscillations in heart rate was approximately 10 to 20 min (fig. 2). After the surgery, the patient was widely awaked and extubated while breathing pure oxygen. Little changes in heart rate and blood pressure were found, and sinus beats persisted throughout this procedure. His postoperative course was uneventful.

Discussion

Atrioventricular dissociation is a relatively rare arrhythmia with an incidence of 0.48 per cent[4], and induced by a variety of autonomic influences, drugs, infections, and others[4]. But under general anesthesia with halothane or enflurane, isorhythmic dissociation occurs frequently (42%–50%)[2,5]. In addition to inhaled anesthetics, surgical or sympathetic stimulation[5], pancuronium[5], and atropine[4] are possibly responsible for the mechanism of this frequently encountered rhythm disturbance during general anesthesia.

Essentially in the isorhythmic dissociation, two pacemakers, i.e., the sinus and atrioventricular nodes, fortuitously discharge at the same or a nearly similar rate without antegrade or retrograde conduction across the atrioventricular node[1]. It has

been suggested that sympathetic or surgical stimulation exerts more influences on atrioventricular nodal cells than on sinus pacemaker cells[6]. Furthermore, the automaticity of sinoatrial nodal cells is depressed by inhaled anesthetics more than that of lower pacemaker cells[7]. Clinically, heart rate has been noted to increase on conversion from sinus rhythm to isorhythmic dissociation[3], as in our patient. Enflurane is known to consistently increase heart rate in healthy volunteers[2,8]; this may be relevant to the frequent occurrence of isorhythmic dissociation. Therefore, the acceleration of heart rate induced by sympathetic stimulation, vagal depression (by atropine), or both, seems to predispose to the development of isorhythmic dissociation in many circumstances.

In the present patient, isorhythmic dissociation occurred periodically with a cycle of approximately 10–20 min for quite long period of time during enflurane, nitrous oxide and oxygen anesthesia. Oscillations in blood pressure have been observed in experimental animals[8–12] and critically ill patient[13]. These cyclic variations in blood pressure with periods of second, minute, or hour time scales are accompanied by oscillations in heart rate[9], cardiac output[9], and vascular resistance[10]. However, the definite mechanism of the oscillations in hemodynamics remains unclear, although these phenomena have been known to be produced or augmented by metabolic acidosis[11], hypoxia[11], hypovolemia[11], and intracranial hypertension[12] or cerebral ischemia[13]. The oscillations in heart rate associated with isorhythmic dissociation observed in this case might be related to these hemodynamic rhythms of unknown, but probably central origin.

The treatment for isorhythmic dissociation is rarely necessary[6]. Formerly, the Valsalva maneuver had been proposed as a method to restore normal sinus rhythm from isorhythmic dissociation[14]. In recent reports, propranolol[6] or calcium chloride[15] has been demonstrated to be an effective drug for isorhythmic dissociation. However, isorhythmic dissociation of this case did not reappear in the postoperative period, indicating no requirement of special care for this arrhythmia, since isorhythmic dissociation is considered to be fundamentally benign and have little clinical significance, if the hemodynamic change is trivial, or underlying heart disease does not exist[16].

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References

1. Sethna DH, Deboer GE, Millar RA: Observations on "junctional rhythms" during anaesthesia. Br J Anaesth 56:924–925, 1984
2. Calverley RK, Smith NT, Prys-Roberts C, Eger EI II, Jones CW: Cardiovascular effects of enflurance anesthesia during controlled ventilation in man. Anesth Analg 57:619–628, 1978
3. Sosis M, Cooper PS, Herr G: The diagnosis of "junctional rhythms" with halogenated anesthetics. Anesthesiology 63:233–234, 1985
4. Marriott HJL, Schubart AF, Bradley SM: A-V dissociation: A reappraisal. Am J Cardiol 1:586–605, 1958
5. Haldemann G, Schaer H: Haemodynamic effects of transient atrioventricular dissociation in general anaesthesia. Br J Anaesth 44:159–162, 1972
6. Breslow MJ, Evers AS, Lebowitz P: Successful treatment of accelerated junctional rhythm with propranolol: Possible role of sympathetic stimulation in the genesis of this rhythm disturbance. Anesthesiology 62:180–182, 1985
7. Hauswirth O: Effects of halothane on single atrial, ventricular, and Purkinje fibers. Circ Res 24:745–750, 1969
8. Levesque PR, Nanagas V, Shanks C, Shimosato S: Circulatory effects of enflurane in normocarbic human volunteers. Can Anaesth Soc J 21:580–585, 1974
9. Shimada SG, Marsh DJ: Oscillations in mean arterial blood pressure in conscious dogs. Circ Res 44:692–700, 1979
10. Killip T III: Oscillation of blood flow and vascular resistance during Mayer waves. Circ Res 11:987–993, 1962
11. Ferretti R, Cherniack NS, Longobardo G, Levine OR, Morkin E, Singer DH,

Fishman AP: Systemic and pulmonary vasomotor waves. Am J Physiol 209:37–50, 1965

12. Guyton AC, Satterfield JH: Vasomotor waves possibly resulting from CNS ischemic reflex oscillation. Am J Physiol 170:601–605, 1952
13. Moorthy SS, Stoelting RK, King RD: Delayed cyclic variations (oscillations) in blood pressure in a critically ill patient. Crit Care Med 11:476–477, 1983
14. Schubart AF, Marriott HJL, Gorten RJ: Isorhythmic dissociation: Atrioventricular dissociation with synchronization. Am J Med 24:209–214, 1958
15. Gottlieb A, Satariano P, Sethna D, Millar RA: Isorhythmic dissociation. Anesthesiology 64:407, 1986
16. Chung EK: Electrocardiography: Practical Applications with Vectorial Principles, Second ed. Philadelphia, Harper & Row, Publishers, Inc., 1980, pp. 208–217