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Perioperative management of lobectomy in a patient with hypertrophic obstructive cardiomyopathy treated with dual-chamber pacing

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Introduction

In patients with hypertrophic cardiomyopathy (HCM), β-adrenergic blockers, calcium antagonists, and disopyramide have been used to decrease the degree of the left ventricular outflow tract (LVOT) narrowing and to effectively relieve symptoms, prevent complications, and reduce the risk of sudden death [1]. In some patients with severely symptomatic and drug-refractory hypertrophic obstructive cardiomyopathy (HOCM), dual-chamber (DDD) pacing is proposed as an alternative treatment to surgical myectomy [2]. By pacing the right ventricular apex, DDD pacing is believed to beneficially reduce the pressure gradient between the LVOT and aorta (LV-Ao PG) via dyssynchronous contraction of the septum [3]. The present report demonstrates the usefulness of DDD pacing in perioperative management of an HOCM patient who underwent right upper lobectomy for lung cancer.

Case report

A 59-year-old man (height, 163 cm; weight, 63 kg) was referred to our hospital for preoperative evaluation of HOCM and right upper lobectomy. The patient first noted chest pain and became out of breath following slight exertion such as climbing a flight stairs 4 years previously. A routine chest roentgenogram taken during an annual check-up showed a suspicious shadow. The patient was referred to another hospital for a complete medical evaluation. Left lung cancer was diagnosed. His electrocardiogram (ECG) showed a negative T wave in leads V5 and V6, indicating a pattern of old septal and inferior myocardial infarction with mild left ventricular (LV) hypertrophy. Furthermore, epicardial echocardiography (EE) revealed interventricular septal hypertrophy (ISH) and LV outflow hypertrophy, without deterioration of LV wall motion but with mild mitral regurgitation (MR). His cardiac index (CI), ejection fraction (EF), and EE-derived LV-Ao PG were 3.85 l·min^{-1}·m^{-2}, 86%, and 72 mmHg, respectively. Finally, HOCM accompanied with MR were diagnosed. The patient was scheduled for right upper lobectomy in the same hospital without any specific medical treatment for HOCM prior to surgery.

The patient was premedicated with intramuscular atropine (0.5 mg) and hydroxyzine (50 mg) prior to the induction of anesthesia. On arrival at the operating theater, his heart rate was 78 beats·min^{-1}, and his systolic and diastolic blood pressure showed 132/86 mmHg. After the placement of an epidural catheter via the thoracic segment T8/9, anesthesia was induced with bolus intravenous (iv) fentanyl (0.8 μg·kg^{-1}) and propofol (1 mg·kg^{-1}), as well as with continuous infusion of propofol (10 mg·kg^{-1}·h^{-1}), followed by vecuronium (0.03 mg·kg^{-1}). Hypotension (60 mmHg) and profound bradycardia (35 beats·min^{-1}) were observed after anesthesia was induced. Hypotension was controlled rapidly both by volume replacement and intermittent administration of bolus iv methoxamine (1 mg), whereas bradycardia persisted at less than 40 beats·min^{-1}. The anesthetists discontinued the induction of anesthesia and cancelled the operation. Ten minutes later, the heart rate of the patient gradually recovered to a level of 50 beats·min^{-1}. Oral atenolol (50 mg·day^{-1}) and disopyramide (300 mg·day^{-1}) were started immediately

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after this unanticipated critical event. He was immediately referred to our hospital for further preoperative evaluation for HOCM and for the scheduled lobectomy.

On admission to our hospital, the patient's heart rate was 60 beats·min^{-1} and systolic blood pressure was 150 mmHg. His respiratory function was within normal range. He was classified as New York Heart Association class II. Ambulatory Holter ECG changes consisted of multifocal premature ventricular contraction. The anaerobic threshold (AT) treadmill test showed that AT was 10.4 ml·min^{-1}·kg^{-1} (58% of the standard value) and peak oxygen intake (peak VO_2) was 20.2 ml·min^{-1}·kg^{-1} (63% of the standard value). These values suggested that the patient's exercise tolerance decreased to 60% of standard cardiac reserve. EE revealed asymmetrical ISH (18 mm in the interventricular septal thickness, 12 mm in the LV posterior wall thickness) and second-degree MR with abnormal anterior movement of the anterior mitral leaflet. The EF- and EE-derived LV-Ao PG were 56% and 178 mmHg, respectively. Physicians diagnosed this highly elevated LV-Ao PG to be derived from a sigmoid hypertrophic septum protruding into the LVOT. Because LV-Ao PG was not sufficiently lowered by atenolol and disopyramide, temporary DDD pacing was attempted to obtain a stable perioperative circulatory state. Cardiac catheterization was performed before pacing and the baseline data were as follows: pulmonary arterial pressure (PAP), 31/9 mmHg; pulmonary capillary wedge pressure (PCWP), 9 mmHg; left ventricular end-diastolic pressure (LVEDP), 16 mmHg; LV-Ao PG, 100 mmHg; and CI, 2.5 l·min^{-1}·m^{-2}. Temporary DDD pacing was then installed via the right internal jugular vein. Hemodynamic data were also obtained to define the optimum atrioventricular (AV) interval that gave the best reduction in LV-Ao PG without inducing a drop in mean aortic pressure and cardiac output (CO). The treadmill test was performed for 4 min until the patient developed dyspnea. His heart rate increased from 53 to 100 beats·min^{-1} and his systolic and diastolic blood pressure also increased from 115/57 to 162/78 mmHg. These results suggested that the patient would tolerate exercise well unless his heart rates exceeded 100 beats·min^{-1}.

A permanent DDD multiprogrammable pacemaker (Pacesetter, 2360L, St. Jude Medical, St. Paul, MN, USA) was placed subcutaneously under local anesthesia. The optimum AV interval and heart rate were programmed as 82 ms and 45 to 100 beats·min^{-1}, respectively, which had been confirmed in advance to be optimal. Subsequent EE showed asymmetrical interventricular septal movement resulting from DDD pacing. Cardiac catheterization also showed a profound reduction of LV-Ao PG to 6 mmHg. The AT treadmill test showed that AT VO_2 was 12.9 ml·min^{-1}·kg^{-1} (73%) and VO_2 was 19.5 ml·min^{-1}·kg^{-1} (61%), suggesting that the patient's exercise tolerance improved to 70% of standard cardiac reserve. Consequently, we decided to perform right upper lobectomy under general anesthesia.

A continuous cardiac output (CCO) catheter was inserted into the right subclavicular vein to obtain preoperative baseline values. On the following day, the patient was premedicated with iv famotidine 20 mg prior to anesthesia. On arrival at the operating theater, his heart rate was 55 beats·min^{-1}, his blood pressure 128/70 mmHg, CCO 4.8 l·min^{-1}, mixed venous blood oxygen saturation $S\bar{v}O_2$ 70%, and EE-derived LV-Ao PG 15 to 18 mmHg (Fig. 1). A test dose of 2 ml of 2% mepivacaine was given after placement of an epidural catheter via the thoracic segment T4/5. His pacing heart rate was set at 70 beats·min^{-1}. There was little change in CCO (5.4 l·min^{-1}), $S\bar{v}O_2$ (75%), and EE-derived LV-Ao PG (18 mmHg). Anesthesia was gradually and carefully induced with midazolam (0.05 mg·kg^{-1}), continuous infusion of fentanyl at 30 μg·kg^{-1}·h^{-1}, and inhalation of 1% to 2% sevoflurane, followed by vecuronium (0.16 mg·kg^{-1}). Because EE-derived LV-Ao PG increased transiently to 41 mmHg after anesthesia was induced, an acetate Ringer's solution was given rapidly, resulting in a marked decrease to 18 mmHg. After the intubation, a transesophageal echocardiography (TEE) probe was inserted immediately for continuous cardiac monitoring. Anesthesia was maintained with iv fentanyl (100 μg) and 1% to 2% sevoflurane inhalation, combined with 2% mepivacaine epidural administration. Epidural morphine (2 mg) was given once just after the start of the surgery. Continuous epidural infusion containing 8 mg of morphine and 60 ml of 1% mepivacaine at 2 ml·h^{-1} was started 30 min after the start of the surgery for postoperative pain relief.

TEE-derived LV-Ao PG increased to 55 mmHg after the start of the surgery, and then decreased rapidly to 30 mmHg. The PG also transiently increased again to 78 mmHg during one-lung ventilation (Fig. 2), but returned to 40 mmHg 10 min later. Apart from the LV-Ao PG, no marked hemodynamic changes were observed during surgery, as indicated by the following data: pacing heart rate set at 70 beats·min^{-1}; systolic blood pressure, 90–140 mmHg; PCWP, 14–18 mmHg; CCO, 4.2–5.2 l·min^{-1}; and SvO_2, 75%–80%. No marked abnormalities in blood gas analysis or serum electrolyte findings were observed during surgery. The TEE probe was extracted and the patient was extubated shortly after surgery when he had awakened sufficiently from anesthesia. He was then immediately transferred to the intensive care unit (ICU). The patient did not complain of an unbearable pain. His hemodynamic and respiratory status remained stable in the ICU, and data at 6 h after admission to ICU were as follows: paced heart

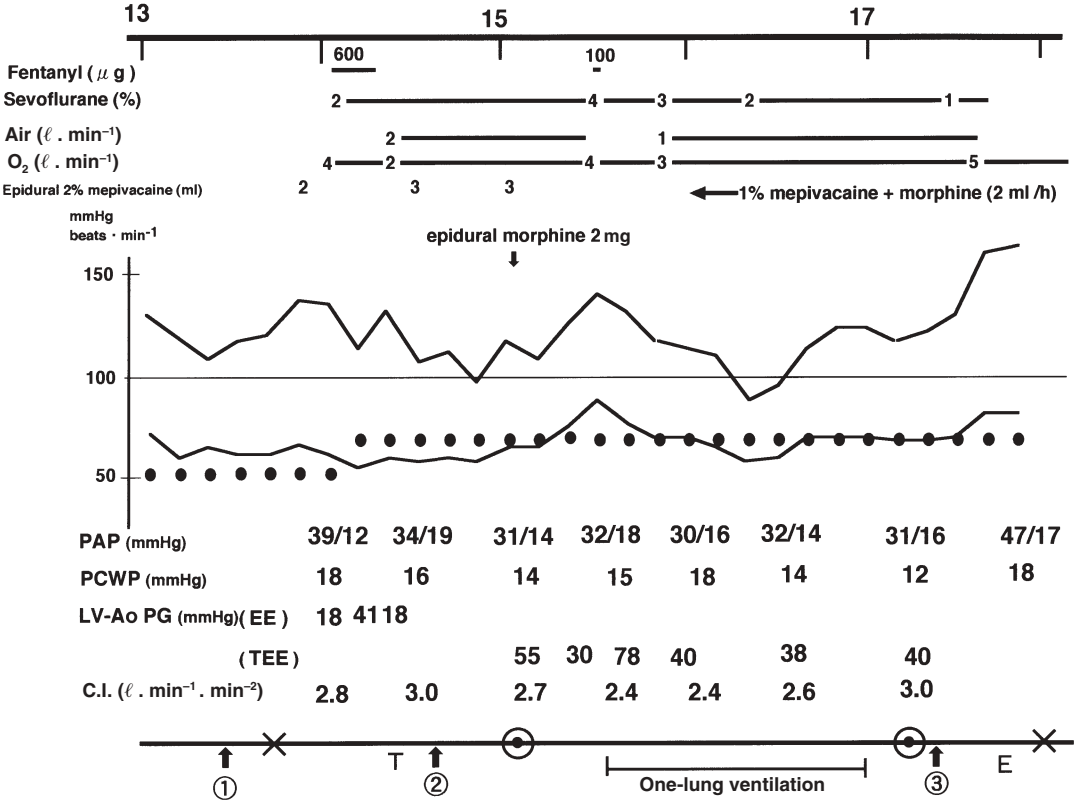

Fig. 1. Time course of anesthesia with sevoflurane and fentanyl in combination with epidural mepivacaine and morphine. *Solid line*, Systolic and diastolic blood pressure, *closed circles*, heart rate; *X*, beginning and end of anesthesia; *opencircles with center dot*, beginning and end of surgery; *T*, tracheal intubation; *E*, extubation; *1*, placement of the epidural catheter; *2*, insertion of the transesophageal echocardiography (TEE) probe; *3*, extraction of the TEE probe; *PAP*, pulmonary arterial pressure; *PCWP*, pulmonary capillary wedge pressure; *LV-Ao PG*, pressure gradient between left ventricular outflow tract and aorta; *EE*, epicardial echocardiography; *C.I.*, cardiac index

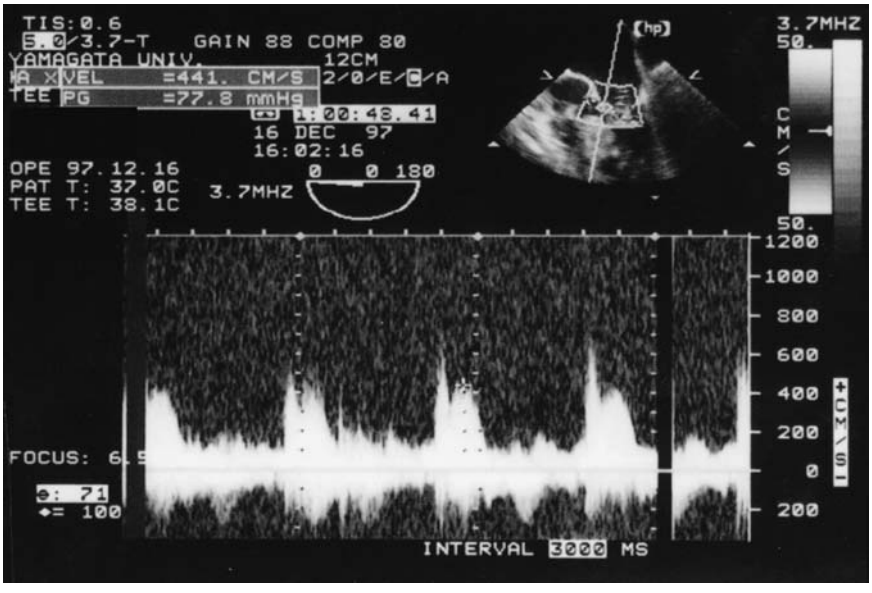

Fig. 2. The intraoperative continuous wave-Doppler tracing of the left ventricular outflow tract jet obtained during one-lung ventilation. The peak systolic velocity was determined to be 4.41 m/s, indicating a maximum systolic gradient of 77.8 mmHg

rate, 70 beats·min^{-1}, blood pressure, 130/72 mmHg; right atrial pressure, 2 mmHg; PCWP, 18 mmHg; and CCO, 5.2 l·min^{-1} . Under 2 l·min^{-1} oxygen inhalation, the arterial blood gas analysis showed a pH of 7.411, a $PaCO_2$ of 42.3 mmHg, and a PaO_2 of 141.3 mmHg. On the following day, the patient was transferred to the surgical ward without any hemodynamic deterioration.

Discussion

HOCM is characterized by asymmetrical increase in ventricular mass, involving the interventricular septum or the anterolateral free wall of the LV, and is accompanied by a decrease in LV diastolic compliance and obstruction in dynamic LV outflow.

The preoperative LV-Ao PG value obtained from EE was 178 mmHg before the second anesthesia, but the value obtained from cardiac catheterization was 100 mmHg and greatly lower than the EE-derived value. In calculating LV-Ao PG by the continuous wave-Doppler (CW-D) method, a high-speed blood flow waveform showing a single peak is detected at the LVOT from mid systole to late systole. From this wave, the pressure amplitude can be calculated using the simplified Bernoulli's formula ($\Delta P = 4V^2$) [4]. In EE, the probe is placed on the body surface near the apex, and projection from this position toward the LV apex parallel to the ejected blood flow is required. In TEE, it is necessary to project the beam parallel to the ejected blood flow in the deep gastric view. To do so, the probe has to be bent greatly forward. Angle correction is conducted when the beam is not parallel to the ejected blood flow; however, correction for over 30° results in great error. The PG value obtained from the CW-D method is the greatest pressure amplitude. Because the location of blood flow cannot be identified from the beam, it is possible that a different outflow from MR or the LV apical flow is detected [5]. Therefore, the PG calculated from cardiac echography may underestimate or overestimate the true value depending on the situation.

By cardiac catheterization, one end of the catheter is advanced up to the LV apex, and pressure is measured directly while the catheter is slowly retracted up to the aorta. During the initial advancement to the LV apex, if the tip is trapped among the LV trabeculae carneae, then it may be mistakenly registered as a high-pressure area. Furthermore, in patients with obstruction in the LVOT, the internal pressure may differ greatly depending on the location of the catheter tip in the LV [6]. Cardiac catheterization records the peak-to-peak gradient between LV pressure and aortic pressure. This arithmetic difference is a nonphysiologic measurement, because the two peak pressures are not synchronized. The catheterization-derived peak-to-peak gradient may be 30% to 40% lower than the maximal instantaneous gradient obtained by the CW-D method [5]. In the present patient, although physicians diagnosed this highly elevated LV-Ao PG of 178 mmHg to be derived from a sigmoid hypertrophic septum, the PG value calculated from EE may be overestimated.

Several factors that increase the degree of obstruction and LV-Ao PG may induce harmful cardiac deterioration. The LV outflow obstruction is exacerbated when the outflow tract is narrowed by contraction of the surrounding hypertrophied muscle and by abnormal anterior movement of the thickened anterior mitral leaflet. LV-Ao PG is augmented by contractile increase (catecholamines, arrhythmia, tachycardia, ketamine), the preload decrease (spinal or epidural anesthesia, hypovolemia), and the afterload decrease (vasodilators, spinal or epidural anesthesia, inhaled anesthetics, opioids) [7]. Therefore, it is essential to prevent excessive reduction of preload and afterload, contractile force, and tachycardia during anesthesia [8]. However, when the present patient was prepared for anesthesia the first time, he had not received any medical treatment for HOCM because his CI and EF remained normal despite the high LV-Ao PG. Although fentanyl and propofol have been recommended for anesthetic considerations in patients with poor cardiac reserve because of their slight cardiodepressive effects [9,10], hypotension and persistent bradycardia occurred in our patient when he was induced with even small doses of these two anesthetics. This harmful event suggested that an untreated HOCM patient with high LV-Ao PG secondary to asymmetric septal hypertrophy is extremely sensitive to the administration of these mildly cardiodepressive anesthetics. We avoided the use of propofol to prevent harmful hemodynamic deterioration. Instead, we induced anesthesia with low-dose midazolam and continuous infusion of fentanyl, and maintained it with 2%–4% sevoflurane to prevent excessive preload and afterload reduction, while carefully observing the hemodynamic changes with CCO and EE or TEE monitoring. Epidural administration of a large bolus dose of local anesthetics may produce marked preload and afterload reduction [11]. Therefore, after surgery was started, we administered epidurally intermittent small bolus doses of 3 ml of 2% mepivacaine, 2 mg of morphine, and continuous infusion of morphine and mepivacaine for postoperative pain relief. No marked hemodynamic changes were observed during and after surgery.

DDD pacing has been introduced as an adjunct treatment in severely symptomatic HOCM patients with marked obstruction of LV outflow refractory to drug therapy. However, the exact mechanisms by which re-

duced LV-Ao PG contributes to the beneficial hemodynamic effect are unclear. Several reports have suggested that pacing may influence myocardial perfusion and asynchronous ventricular septal activation, produce paradoxical septal motion or a negative inotropic effect, decrease mitral valve systolic anterior motion, or increase end-systolic volume [12–14]. However, Maron et al. recently demonstrated in a randomized, double-blind, crossover study that DDD pacing could not be regarded as a primary treatment option for severely symptomatic, drug-refractory patients with HOCM but could be a therapeutic option for some elderly patients [15]. Because medical treatment with atenolol and disopyramide did not sufficiently decrease the LV-Ao PG in the present patient, we started DDD pacing before surgery and carefully determined the optimum AV interval setting to minimize LV outflow obstruction, resulting in the marked reduction of LV-Ao PG to 6 mmHg. This HOCM patient with sufficiently reduced LV-Ao PG tolerated anesthesia well. DDD pacing is a useful and reliable therapeutic means to reduce LV-Ao PG, resulting in a stable circulatory state during the perioperative period.

CCO allows continuous monitoring of intracardiac pressure, CO, and mixed venous blood oxygen saturation. Among these parameters, PCWP is an indirect indicator to estimate the LV end-diastolic volume. However, in patients with HCM, the LV diastolic pressure-volume relations were shifted upward [16], reflecting impaired LV compliance. This finding suggests that LV end-diastolic pressure was disproportionately increased [17]. PCWP may be elevated, particularly if there is MR, but it may not accurately reflect LV end-diastolic volume. TEE measures the left ventricular end-diastolic volume directly and also permits observation of local wall movement. Therefore, TEE not only monitors the preload, but it also monitors cardiac contractility, myocardial ischemia, stenotic change of LOVT, and the degree of MR [18]. However, the TEE monitor can only be used during anesthesia. Patients with HOCM are at risk of exacerbation of LVOT narrowing during surgery because of various factors such as dilatation of peripheral blood vessels, reduced preload and afterload caused by low arterial perfusion, reduced cardiac contractility, and light anesthesia [8]. By monitoring hemodynamics using CCO and TEE simultaneously in these patients, the changes of cardiac function and the causes can be diagnosed promptly from pressure and morphological data, and treatment can also be evaluated under more of a real-time situation. Thus, COO and TEE that mutually supplement each other are important monitors of cardiac function during anesthesia in patients with HOCM.

We placed both a CCO catheter and a TEE probe for intraoperative continuous cardiac monitoring in our present patient. Specifically, when mild hypotension occurred during surgery, we immediately adjusted the infusion rates to restore the preload to a suitable level, meanwhile checking the LV end-diastolic volume, the LV-Ao PG, and the magnitude of the narrowing of the LVOT. TEE monitoring is a useful means for early detection of a preload reduction and consequent narrowing of the LVOT, permitting early treatment for hypotension.

In summary, in our patient with HOCM, DDD pacing effectively improved his deteriorated cardiac function and maintained a stable circulatory state. This strategy is effective for maintaining suitable circulation during anesthesia.

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References

1. Wynne J, Braunwald E (1997) The cardiomyopathies and myocardities. In: Braunwald E (ed) Heart disease: a textbook of cardiovascular medicine, 5th edn. WB Saunders, Philadelphia, pp 1404–1457
2. Kappenberger L, Linde C, Daubert C, Mckenna W, Meisel E, Sadoul N, Chojnowska L, Guize L, Gras D, Jeanrenaud X, Ryden L, the PIC Study Group (1997) Pacing in hypertrophic obstructive cardiomyopathy. A randomized crossover study. Eur Heart J 18:1249–1256
3. Slade AKB, Sadoul N, Shapiro L, Chojnowska L, Simon JP, Saumarez RC, Dodinot B, Camm AJ, McKenna WJ, Aliot E (1996) DDD pacing in hypertrophic cardiomyopathy: a multicentre clinical experience. Heart 75:44–49
4. Sasson Z, Yock PG, Hatle LK, Alderman EL, Popp RL (1988) Doppler echocardiographic determination of the pressure gradient in hypertrophic cardiomyopathy. J Am Coll Cardiol 11:752–756
5. Nishimura RA, Tajik AJ (1994) Quantitative hemodynamics by Doppler echocardiography: a noninvasive alternative to cardiac catheterization. Prog Cardiovasc Dis 36:309–342
6. Grossman W (1996) Profiles in dilated (congestive) and hypertrophic cardiomyopathy. In: Baim DS, Grossman W (eds) Cardiac cathertherization, angiography, and intervention, 5th edn. Wiliams and Wilkins, Baltimore, pp 787–800
7. Shanewise JS, Hug CC (2000) Anesthesia for adult cardiac surgery. In: Millar RD (ed) Anesthesia, 5th edn. Churchill Livingstone, Philadelphia, pp 1753–1804
8. Thompson RC, Liberthson RR, Lowenstein E (1985) Perioperative anesthetic risk of noncardiac surgery in hypertrophic obstructive cardiomyopathy. JAMA 254:2419–2421
9. Hui TW, Short TG, Hong W, Suen T, Gin T, Plummer J (1995) Additive interactions between propofol and ketamine when used for anesthesia induction in female patients. Anesthesiology 82: 641–648
10. Sorbara, C, Pittarello D, Rizzoli G, Pasini L, Armellin G, Bonato R, Giron GP (1995) Propofol-fentanyl versus isoflurane-fentanyl anesthesia for coronary artery bypass grafting: effect on myocardial contractility and peripheral hemodynamics. J Cardiothorac Vasc Anesth 9:18–23
11. Kwok MH, Warwick DNK, Michael CMP (1997) Combined spinal and epidural anesthesia in a parturient with idiopathic hypertrophic subaortic stenosis. Anesthesiology 87:168–169

12. Jeanrenaud X, Goy JJ, Kappenberger L (1992) Effects of dual-chamber pacing in hypertrophic obstructive cardiomyopathy. Lancet 339:1318–1323
13. Posma JL, Blanksma PK, Van Der Wall EE, Vaalburg W, Crijns HJGM, Lie KI (1996) Effects of permanent dual chamber pacing on myocardial perfusion in symptomatic hypertrophic cardiomyopathy. Heart 76:358–362
14. Pak PH, Maughan WL, Baughman KL, Kieval RS, Kass DA (1998) Mechanism of acute mechanical benefit from VDD pacing in hypertrophied heart: similarity of responses in hypertrophic cardiomyopathy and hypertensive heart disease. Circulation 98: 242–248
15. Maron BJ, Nishimura RA, McKenna WJ, Rakowski H, Josephson ME, Kieval RS, for the M-PATHY Study Investigators (1999) Assessment of permanent dual-chamber pacing as a treatment for drug-refractory symptomatic patients with obstructive hypertrophic cardiomyopathy. A randomized, double-blind, crossover study (M-PATHY). Circulation 99:2927–2933
16. Yamakado T, Okano H, Higashiyama S, Hamada M, Nakano T, Takezawa H (1990) Effects of nifedipine on left ventricular diastolic function in patients with asymptomatic or minimally symptomatic hypertrophic cardiomyopathy. Circulation 81:593–601
17. Louie EK, Edwards LC (1994) Hypertrophic cardiomyopathy. Prog Cardiovasc Dis 36:275–308
18. Grigg LE, Wigle ED, Williams WG, Daniel LB, Rakowski H (1992) Transesophageal Doppler echocardiography in obstructive hypertrophic cardiomyopathy: clarification of pathophysiology and importance in intraoperative decision making. J Am Coll Cardiol 20:42–52