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Intraoperative hemodynamic management for minimally invasive direct coronary artery bypass: two case reports

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Introduction

The in-hospital mortality rate for conventional coronary artery bypass graft (CABG) surgery remains approximately 3% [1,2], despite advances in surgical and anesthetic techniques as well as in extracorporeal cardiopulmonary devices. The high mortality may be explained by an increase in the population of aged patients, the increased number of candidates for CABG [3], and the increased incidence of emergency CABG in the last decade [4]. The mortality of CABG is still high in high-risk patients with severe atherosclerosis, cardiac failure, renal failure, or cerebral infarction [5,6]. High-risk patients are more likely to experience complications of cardiopulmonary bypass (CPB) as a result of embolic phenomena associated with aortic cannulation and cross-clamping [7,8]. Therefore, the technique of coronary revascularization on a beating heart without CPB was developed [9] to prevent complications caused by CPB in CABG patients. Recently mini-thoracotomy or mini-sternotomy has been adopted as an alternative surgical approach because it is less invasive than median sternotomy [10,11]. This procedure is called minimally invasive direct coronary artery bypass (MIDCAB). This report describes the hemodynamic management of two patients undergoing MIDCAB procedures.

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Case 1

A 70-year-old man (53 kg, 152 cm) with hypertension and unstable angina was scheduled for single coronary bypass of the left internal mammary artery (LIMA) to the left anterior descending coronary artery (LAD) due to 90% stenosis of the left main coronary artery. The MIDCAB procedure was chosen to avoid CPB because of the patient's severe aortic calcification, cerebral infarction, and renal insufficiency. He had been orally medicated with amyl nitrite (40 mg·day^{-1}), diltiazem hydrochloride (90 mg·day^{-1}), nicorandil (15 mg·day^{-1}), and metoprolol tartrate (60 mg·day^{-1}).

The patient was premedicated with oral flunitrazepam (1 mg) 1 h before arrival at the operating room. Anesthesia was induced with intravenous fentanyl (300 μg) and midazolam (10 mg). The patient was intubated with a double-lumen endotracheal tube (DLT) after intravenous vecuronium (6 mg) for one-lung ventilation (OLV) during the preparation of LIMA. A probe for regional cerebral hemoglobin saturation (rSO_2) was placed on the right forehead. The radial artery was cannulated for blood pressure monitoring and blood gas analysis. A pulmonary artery (PA) catheter (A-V Paceport Thermodilution Catheter, Baxter Healthcare, USA) and a double-lumen central venous catheter (Safe Guide, Argyle, Japan) were placed through the right internal jugular vein. Atrial pacing began at 72 beats·min^{-1} using a pacing probe (Baxter Healthcare) introduced through the PA catheter, because severe sinus bradycardia (35 beats·min^{-1}) appeared soon after induction of anesthesia. Sterilized external paddles were made ready for emergency cardioversion. Conventional electrocardiograms (II and V_5) and transesophageal echocardiography (TEE) monitoring were provided for the detection of myocardial ischemia during LAD occlusion. An additional 300 μg of fentanyl was administered before skin incision. Subsequent anesthesia was maintained with intrave-

nous infusion of propofol (5 mg·kg^{-1}·h^{-1}), supplemented with inhalation of sevoflurane (1%–2% end-tidal) as needed.

A mini-thoracotomy with an incision of about 10 cm was performed in the left fourth intercostal space with the patient in the supine position with a slight lateral tilt of the left hemithorax. The LIMA was exposed and prepared for a length of 5 cm from the inner surface of the chest wall under OLV. OLV was terminated when the heart was suspended in the pericardial cradle through the 4th intercostal space.

Heparin (30 mg) was injected intravenously to adjust the active coagulation time to around 200 s in order to avoid thrombosis formation during coronary occlusion. An intravenous infusion of nitroglycerin (0.5 μg·kg^{-1}·min^{-1}) was started before the temporary occlusion of the LAD for surgical anastomosis. The pacing rate was reduced to 40 beats·min^{-1} to facilitate surgical procedures for the coronary anastomosis. After the LAD had been clamped titration of blood pressure with phenylephrine and several bolus injections of calcium gluconate (total dose, 425 mg) were used to keep the systolic blood pressure above 80 mmHg. The cardiac output decreased from 3.6 to 2.0 l·min^{-1}, and the pulmonary capillary wedged pressure (PCWP) increased from 8 to 14 mmHg (Fig. 1). Although the wall motion was slightly diminished in the LAD area supplied (monitored by transesophageal echocardiography), no further deterioration of cardiac contraction was observed during 15 min of coronary occlusion. The rectal temperature was 36°C during this period. Lidocaine (1 mg·kg^{-1}) was administered intravenously before coronary reperfusion. Several ventricular premature contractions were observed, but fatal arrhythmia did not occur after coronary reperfusion. Soon after the pacing rate had returned to the initial 72 beats·min^{-1}, the cardiac output and PCWP returned to the preocclusion values (Fig. 1). An intravenous infusion of diltiazem (0.5 μg·kg^{-1}·min^{-1}) was started to prevent spasm of the grafted LIMA. A single chest tube was left in the thoracic cavity, and an intercostal nerve block was performed with 0.5% bupivacaine before closing the chest. The double-lumen endotracheal tube was replaced with a standard single tracheal tube at the end of surgery, and the patient was transferred to ICU. The patient was extubated after 4 h without any postoperative complication.

Case 2

An 80-year old man (59 kg, 160 cm) with a 5-year history of hemodialysis for renal insufficiency was the second candidate for MIDCAB. He had had an acute myocardial infarction of the inferior wall 4 weeks previously. Coronary angiography (CAG) revealed coronary stenosis of both the right coronary artery (RCA) and the left main trunk (LMT). Percutaneous coronary angioplasty (PTCA) was performed and was successful in releasing the RCA stenosis. However, the patient needed circulatory assistance with an intraaortic balloon pump (IABP) to compensate for impending myocardial ischemia and severe left cardiac dysfunction. Emergency CABG was planned to graft the LIMA to the LAD. Because of single-vessel grafting, renal insuf-

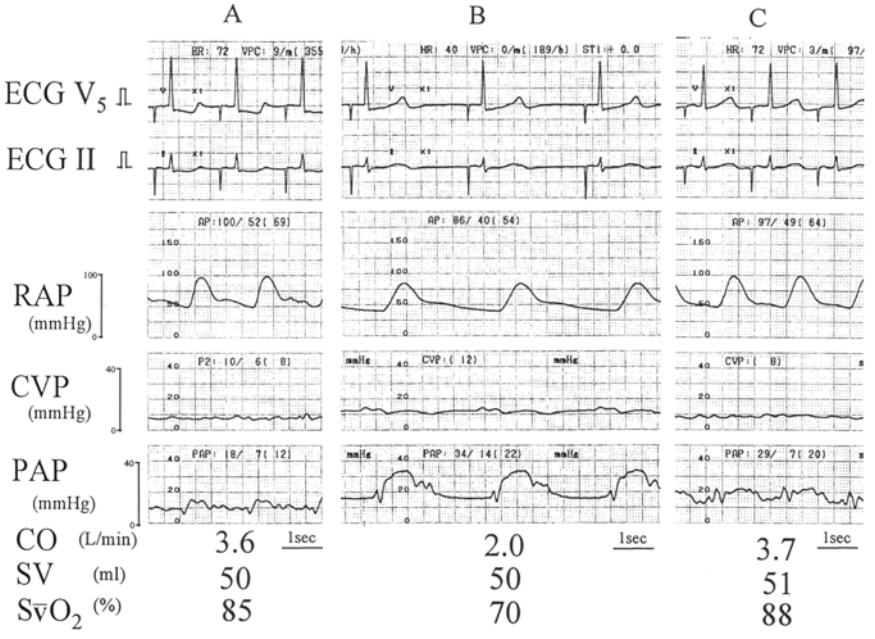

Fig. 1. Atrial pacing rate was reduced from 72 to 40 beats·min^{-1} for coronary anastomosis in case 1. Radial arterial pressure and cardiac output decreased following deterioration of heart rate. **A** Before coronary occlusion, **B** during anastomosis, **C** after coronary reperfusion. *RAP*, Radial arterial pressure; *CVP*, central venous pressure; *PAP*, pulmonary arterial pressure; *CO*, cardiac output; *SV*, stroke volume; $S\bar{v}O_2$, hemoglobin saturation of mixed venous blood

ficiency, and poor cardiac function, the MIDCAB procedure was done.

This patient had been treated with an intravenous infusion of nitroglycerin ($0.5\,\mu g \cdot kg^{-1} \cdot min^{-1}$), nicorandil ($0.5\,\mu g \cdot kg^{-1} \cdot min^{-1}$), and dopamine ($5\,\mu g \cdot kg^{-1} \cdot min^{-1}$) preoperatively. The induction of anesthetia was similar to that described in the first case. The PA catheter was also introduced for A-V pacing after induction of anesthesia, although the heart rate was as high as 108 beats·min^{-1}. Infusion of dopamine was terminated and nicorandil was replaced by diltiazem ($2\,\mu g \cdot kg^{-1} \cdot min^{-1}$) to slow the heart rate. The assist ratio of the IABP was increased from 2:1 to 1:1 in compensation for the presumed deterioration of cardiac performance due to the termination of dopamine infusion. Anesthetic and surgical procedures were similar to those in case 1, except that the prepared LIMA was too short for the direct anastomosis to the LAD so that the LIMA was extended by interposing a 5-cm-long segment of artery harvested from an inferior epigastric artery. The heart rate gradually decreased from 108 to 80 beats·min^{-1} after surgical preparation, but the cardiac movement was still too rapid to allow the coronary anastomosis to be performed. A bolus dose of 10 mg adenosine triphosphate disodium (ATP) (Adetphos-L, Kowa, Japan) was injected through the central venous line to slow the heart rate. This produced 20–40 s of complete A-V block within 10 s after the ATP bolus injection. A-V sequential pacing was conducted to adjust the heart rate to 40 beats·min^{-1} during complete A-V block (Fig. 2). Ten ATP bolus injections (total, 100 mg) were needed to cover the clamping period of 15 min for the anastomosis of the LIMA to the LAD. Titration of blood pressure with phenylephrine and several bolus injections of calcium gluconate (425 mg) were used to keep the systolic blood pressure above 80 mmHg. No further signs of myocardial ischemia were observed during coronary occlusion, though the precordal ECG-ST segment had already been depressed 2 mm before coronary occlusion. Lidocaine ($1\,mg \cdot kg^{-1}$) was administered just before coronary reperfusion to avoid fatal arrhythmia. No alterations of the ECG or of hemodynamics were noted after coronary reperfusion. The dose of diltiazem was reduced to $0.5\,\mu g \cdot kg^{-1} \cdot min^{-1}$, and dopamine was restarted at $5\,\mu g \cdot kg^{-1} \cdot min^{-1}$. The patient was transferred to CCU after the replacement of the DLT with a single-lumen endotracheal tube. Hemodialysis was performed because of the patient's chronic renal insufficiency. He was extubated on the first postoperative day.

Blood transfusion or additional administration of narcotics was not necessary in either of the cases. Myocardial infarction never developed in the postoperative course and the patency of the coronary bypass was confirmed by postoperative CAG 2 weeks after operation in both cases.

Discussion

Since a minimally invasive approach to CABG was reported by Robinson et al. in 1995 [12], several hundred cases of CABG with the MIDCAB procedure have been reported from around the world [11]. This less invasive CABG has been recognized to be beneficial, especially for high-risk patients.

At the same time, anesthesiologists have been faced with tough problems, such as hemodynamic management during the MIDCAB procedure [13,14]. During

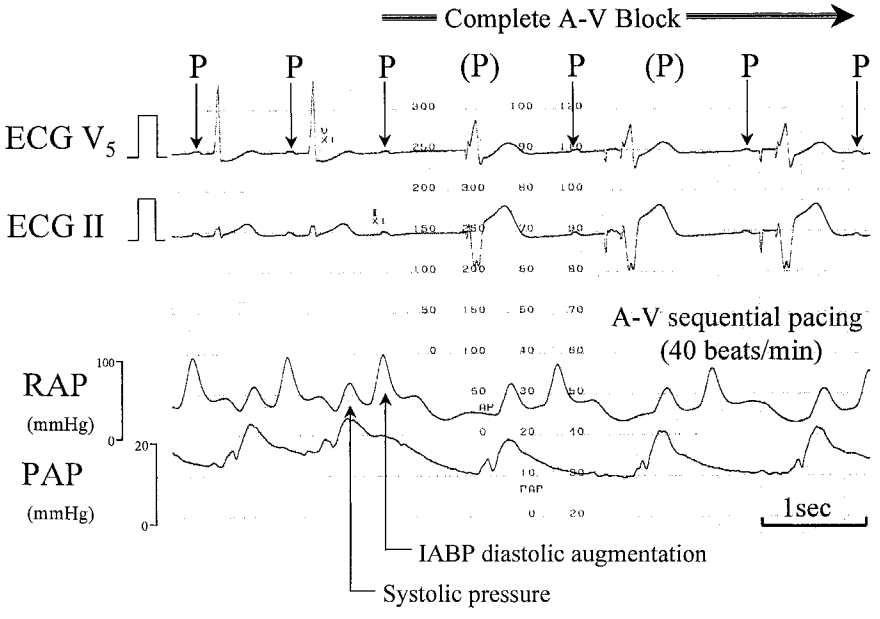

Fig. 2. A-V sequential pacing was conducted at 40 beats·min^{-1} during complete A-V block produced by bolus ATP (10 mg) injection in case 2. *P*, p wave of ECG; *RAP*, radial arterial pressure; *PAP*, pulmonary arterial pressure; *IABP*, intraaortic balloon pump

coronary anastomosis through a narrow surgical field on a beating heart, cardiac surgeons request that the surgical field be kept as still as possible. In the present cases, we coped with movements of the lung by making a pericardial cradle. For beating movements of the heart, we intended to reduce the heart rate by using a pacemaker or ATP bolus injection.

In the first case, the patient's proper rate decreased below 40 beats·min^{-1} after induction of anesthesia, probably because of his preoperative medications, including a beta-blocking agent and a calcium antagonist. Atrial pacing successfully increased his heart rate instantly. Although drip infusion of an ultra-short-acting beta-blocking agent is generally recommended to slow the heart rate during coronary anastomosis [13,14], we could ameliorate periodic movements of the surgical field by changing the rate of atrial pacing from 72 to 40 beats·min^{-1} because of his severe bradycardia (30–35 beats·min^{-1}). Bradycardia led to hypotension, with low diastolic pressure due to the long diastole. The combination of a vasopressor and an inotropic agent such as calcium was useful to keep the systolic blood pressure above 80 mmHg without affecting the heart rate in our cases. The stroke volume was well maintained, but the cardiac output, which is dependent on the heart rate, was decreased remarkably. Although the cardiac output decreased to half of the preocclusion value during the period of slow heart rate, the saturation of mixed venous hemoglobin was preserved at around 70%, and the value of rSO_2 did not indicate any significant change. In this patient a heart rate of 40 beats·min^{-1} seemed to be sufficient for the surgical procedure and also for his oxygen demand during the short period under anesthesia. However, because ischemic heart problems are often accompanied by carotid or cerebral artery disease [15], we must pay attention to the oxygen balance of the brain in the low cardiac output state. To detect brain ischemia during the period of induced slow heart rate, rSO_2 [16] is considered a valuable monitor in these patients.

When MIDCAB is chosen in an emergency case with heart failure due to unstable angina, more concerns and cautions are required in the management of the heart rate, cardiac function, and oxygen supply. In general, the heart rate is high during inotropic treatment of patients with poor cardiac function. It is difficult to decrease the heart rate without further deterioration of cardiac function, which often leads to a severe low cardiac state, including severe hypotension, myocardial ischemia, and fatal arrhythmia. In the second patient, who was on dopamine infusion and had an IABP the heart rate was actually more than 100 beats·min^{-1} at the beginning of anesthesia. Termination of dopamine infusion and an increase in the ratio of the IABP did not reduce the heart rate sufficiently for coronary suturing. Therefore, we were obliged to use ATP repeatedly, which slowed the conduction time through the A-V node [17,18]. A bolus injection of 10 mg ATP produced complete A-V block lasting 28 ± 5.0 s (n = 10). During complete A-V block induced by ATP, A-V sequential pacing was extremely useful to secure a heart rate of 40 beats·min^{-1} and to avoid severe cardiac failure.

To reduce the heart rate, we used ATP instead of an ultra-short-acting beta-blocker such as esmolol, which is not available at present in Japan. When a bolus injection of ATP is administered to reduce the heart rate, careful observation of the subsequent hemodynamic changes is required, because it may lead to severe hypotension due to systemic vasodilation and A-V uncoupling. Cholinesterase inhibitors may also reduce the heart rate temporarily. These agents are believed to produce sinus bradycardia by acting on the atrial muscarinic receptors while keeping blood pressure high [19].

Myocardial protection during coronary occlusion should be discussed carefully, because myocardial blood flow occurs only through collateral vessels until the coronary anastomosis has been accomplished. A combination of high coronary perfusion pressure and infusion of nitroglycerin during coronary occlusion is expected to increase the collateral blood flow, which minimizes myocardial ischemia. ATP, which is quickly metabolized to adenosine, also acts as a cardioprotective agent. Adenosine is a powerful coronary dilator and also has a cardioprotective effect through the coupling system of the adenosine (A_1) receptor and the ATP-sensitive K^+ channel during myocardial ischemia [20]. A recent study showed that isoflurane had a similar cardioprotective effect through the ATP-sensitive K^+ channel during myocardial ischemia [21]. Therefore, isoflurane seems to be a suitable anesthetic for the MIDCAB procedure if the heart rate is kept slow. The cardioprotective effect of propofol has not been verified during myocardial ischemia, though propofol can reduce myocardial oxygen consumption [22].

The surgical indication of MIDCAB should be decided carefully [13,14], because intraoperative hemodynamic management is accompanied by high risk during coronary anastomosis. Percutaneous transluminal coronary angioplasty (PTCA) for the LMT stenosis is thought to be one of the contraindications, because of its high mortality [23]. On the other hand PTCA after CABG to the LAD is considered an effective and safe treatment for patients with LMT stenosis [24]. Therefore PTCA after MIDCAB might be a new tactic for the treatment of patients with LMT stenosis. If hemodynamic risks during the MIDCAB procedure are overcome completely, this combination of less invasive and more economical MIDCAB with PTCA will be a promising treatment when compared with conventional CABG [13,14].

In conclusion, we report two successful cases of MIDCAB, one elective and the other emergency. We think further clinical experiences and efforts regarding intraoperative hemodynamic management will lead to safe MIDCAB.

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