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Recovery of consciousness after an 18-min global cerebral ischemia

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Introduction

The maximal tolerable time of circulatory arrest from which complete cerebral functional recovery could be expected under normal body temperature has been estimated to be 5–7 min. Recent findings [1] regarding postischemic biochemical events have indicated several promising drugs such as barbiturates [2–5], calcium channel blockers [6], excitatory amino acid antagonists [7], and free radical scavengers [8] which can be administered before or even after ischemic insult to avoid or ameliorate cerebral damage. However, as yet no practical means exists as effective and reliable as preischemic hypothermia [5, 9, 10] for minimizing the risk of cerebral damage in surgical procedures where temporary brain ischemia is unavoidable.

This report describes a patient who suffered an 18-min interruption of cerebral circulation during an emergency surgery for acute dissection of an ascending aortic aneurysm. The patient subsequently recovered full consciousness and was discharged with only slight paralysis of the right side 3 months after surgery.

Case report

A 77-year-old man (height 162 cm, weight 53 kg) who suddenly developed severe chest pain was sent to the intensive care unit (ICU) of our hospital with suspected acute dissection of an ascending aortic aneurysm.

He had been suffering from essential hypertension and taking nifedipine and furosemide orally for several years previously. On admission to the ICU, the patient was in apparent distress and his face was edematous. Consciousness was clear and no neurological disorders were noted. He presented orthopnea with a respiration rate of 22. Blood pressure was 150/104 mmHg and heart rate 110 bpm. After confirmation of the dissecting aortic aneurysm with intravenous angiography, an emergency surgery was determined to be necessary. Preoperative electrocardiogram and echocardiogram revealed the complication of myocardial infarction on the inferior wall. An aortocoronary bypass graft using the saphenous vein was also planned in addition to the replacement of the ascending aorta with an artificial vessel.

The patient was premedicated with intramuscular scopolamine 0.25 mg and morphine 3 mg. Anesthesia was induced with intravenous midazolam 5 mg and fentanyl 800 μg and the trachea was intubated after intravenous vecuronium 8 mg. Anesthesia was maintained with incremental doses of midazolam (total 10 mg) and fentanyl (total 4.8 mg) and inhalation of isoflurane <1.0% in oxygen. Systolic blood pressure was maintained at 100–120 mmHg by adjusting the intravenous infusion rates of nitroglycerin, diltiazem, and prostaglandin E_1, administration of which had been begun at the ICU. Blood pressure was monitored at three different sites: the right and left radial arteries at the wrist and the left dorsalis pedis artery. After sternotomy in the supine position, two venous cannulae were introduced through the right auricle, one into the superior vena cava and the other into the inferior vena cava, and an arterial cannula introduced through the right femoral artery into the aorta for the establishment of partial extracorporeal cardiopulmonary bypass (ECPB). The ECPB circuit was primed with lactated Ringer's solution (1000 ml), 20% mannitol (40 g), 25%

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albumin (25 g), 8.4% sodium bicarbonate (80 mEq), urinastatin (250 000 unit), poloxamer-188 (42 ml), and heparin (30 mg). Heparin 160 mg was intravenously administered immediately before the start of partial ECPB. Manual blind separation between the posterior wall of the aneurysm and the surrounding adhesive tissues was performed carefully with initiating partial ECPB. Betamethasone 80 mg was given from the arterial side after the initiation of ECPB.

The posterior wall of the aneurysm was torn in the surgical separating procedure, causing blood to well up in the operating field and flooding the thoracic cavity. The body temperature at that time was 33.3°C in the esophagus and 34.4°C in the rectum. Cooling proceeded as quickly as possible by using a heat exchanger integrated with the artificial lung. The radial artery pressures fell markedly and cardiac arrest followed 2 min after the rupture of the aneurysm. As the aneurysm adhering to the surrounding tissues extended distally along the aortic arch, we could not make space around the aortic arch for cross-clamping or taping.

We were obliged to interrupt the ECPB, because the oxygenated blood from the femoral cannula erupted through the widely ruptured aortic wall into the thoracic cavity. Continuation of ECPB did not contribute to increases in the radial and dorsalis pedis arterial pressures. Thiamylal sodium 125 mg was administered from the arterial side of the ECPB circuit for brain protection. The aneurysm along the ascending aorta, extending from near the Valsalva sinus to the branching site of the brachiocephalic artery, was resected and a 12 Fr balloon catheter with 50 ml liquid capacity was introduced through the true lumen at the distal cut end of the ascending aorta (Fig. 1). The ECPB was then restarted by holding the metal stylet of the balloon catheter against the blood stream. However, neither the right nor left radial pressures increased, indicating that the aorta was occluded with the inflated balloon at the branching site of the left subclavian artery or at a more distal site. Efforts to reposition the balloon at the proper site were time-consuming.

Selective perfusion to the brachiocephalic artery using oxygenated blood (300 ml/min) was additionally established in haste. The right radial arterial pressure rose to around 40 mmHg after the initiation of selective perfusion. Eighteen minutes had elapsed from cardiac arrest until the establishment of selective perfusion. The esophageal temperature immediately before the start of selective perfusion was 28.4°C and the rectal temperature was 31.6°C. Hematocrit was 22% and hemoglobin 7.7 g/dl, while the corresponding preoperative values were 38.6% and 13.4 g/dl, respectively. Cooling was continued down to an esophageal temperature of 16.4°C and a rectal temperature of 21.4°C. The pupil, which was 1.5 mm in diameter at the time of aneurysm

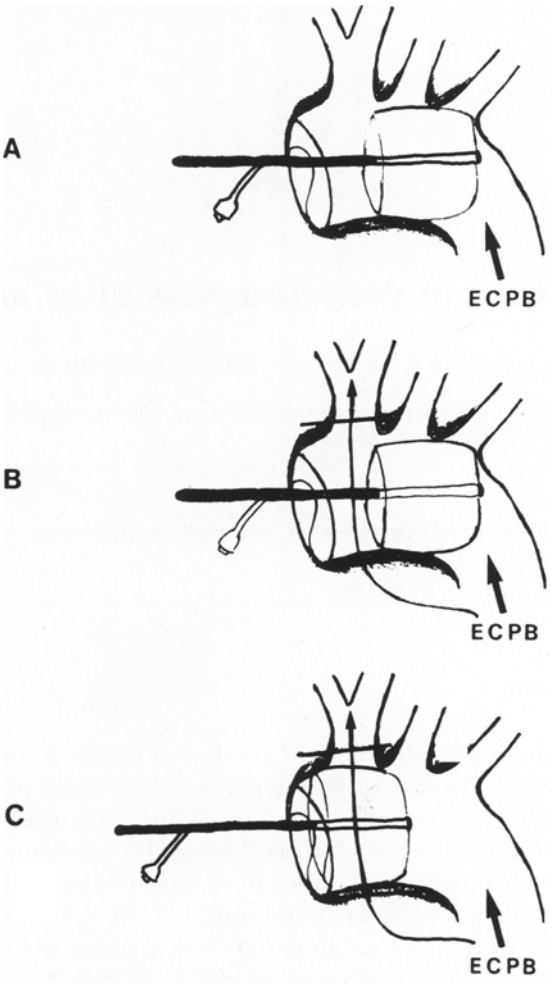

Fig. 1A–C. Schematic drawings of the process of reestablishing cerebral perfusion by using extracorporeal cardiopulmonary bypass (*ECPB*) and a ballon catheter. **A** 10 min, **B** 18 min, **C** 25 min after cardiac arrest

rupture, fully dilated to 7 mm at the initiation of selective perfusion. It showed a gradual return to the preischemic size. Pupil size diminished to 4 mm 2 h later and 2 mm 7 h later. Laterality of the pupil size was not evident during or after operation. Surgery was finished by replacing the ascending aorta and the proximal part of the aortic arch with an artificial vessel and by making anastomosis of the proximal end of the brachiocephalic artery to the graft.

Time spent on the surgical procedures was 11 h and 5 min. Conducting time of the extracorporeal bypass was 405 min and that of selective perfusion was 88 min. Weaning from ECPB was smooth and uneventful.

The patient was transported to the ICU without awakening from anesthesia under assisted ventilation. The level of consciousness tended to recover gradually and improved to 10 on the Japan coma scale [11] on the 10th postoperative day, though accompanied by right

incomplete hemiplegia. Magnetic resonance imaging revealed focal bleeding of the left cerebellum.

During subsequent hospitalized days the patient underwent rehabilitation, restoring consciousness to the preopertive level and was discharged with only slight paralysis of the right side 3 months after surgery.

Discussion

This patient almost fully recovered the cerebral function, though the arrest time of cerebral circulation apparently exceeded the conventional safety limit at normothermia. Such a good outcome seems to be attributable to the fact that the body temperature was in a mild hypothermia at the time of cardiac arrest and that the brain was forcefully perfused using the ECPB after the ischemic events. Cooling seems to be an established and the most reliable technique to protect the brain from ischemic insult. The cerebral metabolic rate of oxygen at 37°C body temperature decreases to 50% at 27°C and <8% at 17°C [9]. Todd et al. [5] demonstrated the protective effects of mild hypothermia (34°C) in cardiac patients. Tharion et al. [10] reported that 1 h of global complete ischemia of the brain was possible at 12°–15°C body temperature in pediatric cardiac patients.

Gilston [12] reported ECPB as a potential intervention for cerebral resuscitation. He reported two patients resuscitated using ECPB after 15 to 18-min accidental circulatory arrest under normal body temperature, who resumed consciousness with no neurological disorder. Rose et al. [13] also reported a similar case, recovering consciousness after prolonged circuratory arrest of more than 10 min under normothermia, though they stressed the beneficial effects of barbiturate therapy instead of ECPB. At present, barbiturate therapy after ischemic insult is still controversial [2–4].

In our institute, an extracorporeal lung and heart assist (ECLHA) system, which is compact, mobile, and long-term applicable, has been developed and applied on 26 moribund patients [14]. Of these, two patients who had not responded to conventional CPR and subsequently received ECPB via femoro-femoral V-A cannulae under closed chest cardiac massage, were especially impressive in their neurological recovery [15]. Safar [16] established the concept of emergency ECPB as a modern cardiopulmonary-cerebral resuscitation method through a series of animal experiments. Application of ECPB assures an instant and stable supply of oxygenated blood, not only to the brain, but to all postischemic vital organs. It also assists the pump function of the damaged heart, allowing the heart to take a rest for recovery. Additionally, mild hemodilution and heparinization, which are inevitable byproducts of ECPB, are beneficial for postischemic brain damage. Therapeutic hypothermia is easy to perform with a heat exchanger.

ECPB also enables controlled delivery of drugs, which benefit the brain but may depress the circulation.

Kirino [1] found that a deleterious pathophysiological process continues even after restoration of perfusion in the hippocampal neurons, which were thought to be highly vulnerable to ischemia and are responsible for memory function. His findings have raised the possibility that postischemic treatments may be applied before neuronal damage becomes irreversible. If postischemic injury progresses after reperfusion according to a series of biochemical cascades [1], the usefulness of hypothermia after exposure to ischemia must also be reevaluated. Leonov et al. [17] successfully performed ECPB resuscitation in dogs after 17 min of cadiac arrest and suggested the advantage of slight hypothermia during bypass on the basis of subsequent study of the behavior and the neurologic deficit score. Our studies, in which ECPB was initiated immediately after 15 min of cardiac arrest, also demonstrated that the neurological deficit score was less in dogs with slight hypothermia during the ECPB resuscitation compared with those without [18]. It seems reasonable to keep patients under mild hypothermia after ischemic insult for the period of delayed neuronal death in progress [1], although further investigations are needed to determine the the optimum degree and duration of hypothermia therapy.

An 77-year-old man whose cerebral circulation was intraoperatively interrupted for 18 min at an esophageal temperature of 33.3°C almost fully recovered cerebral function. We anesthesiologists should always be prepared for the worst scenario in a case like this and be trained to make resuscitative interventions for the brain instantly.

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