

# Induction of Hypothermia After Intraoperative Hypoxic Brain Insult

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Hypoxic brain injury can be a devastating complication of anesthesia. Fortunately, it has become increasingly rare. Here we report a case of suspected intraoperative hypoxic brain injury treated with moderate hypothermia. Anesthesiologists should be aware of the option of using this therapy and how to employ it in the setting of suspected hypoxic brain injury.

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**T**his report describes an intraoperative hypoxic brain insult and the subsequent intraoperative and postoperative management. The therapeutic use of hypothermia in this setting is described and discussed in detail.

## CASE REPORT

A 57-yr-old, 87-kg woman, with failed back surgery syndrome underwent epidural spinal cord stimulator implantation through a laminotomy. She was positioned prone and a spinal anesthetic was placed (1). Isobaric bupivacaine (15 mg) was administered incrementally to achieve a T4 sensory level. A remifentanyl IV infusion was used to provide sedation and supplemental analgesia. Stimulator placement was successful. At 2 h, the block regressed and she experienced pain during suture placement. The remifentanyl infusion rate was increased. The patient continued to have pain and a remifentanyl bolus (100 µg IV) was given with subsequent loss of consciousness and apnea. Efforts to establish ventilation were unsuccessful. The patient was emergently turned supine. Bag-mask ventilation and resuscitation ensued. At its nadir, the oxygen saturation was 30%, heart rate approximately 20 bpm, and systolic blood pressure 40 mm Hg. Cardiac arrest did not occur. Bradycardia and hypotension responded to oxygenation and atropine.

The period of hypoxemia was approximately 7 min. Arterial blood gas analysis was not performed during this period. Hypoxic injury was suspected when the patient did not awaken 20 min after the last remifentanyl dose. Naloxone (400 µg IV) was incrementally administered with no improvement. At this time, the patient exhibited decerebrate posturing to painful stimuli, further suggesting the presence of brain injury. Transfer to the neurointensive care unit was organized.

Neurointensivist consultation led to the decision to induce moderate hypothermia (target 32°C) in the operating room. The patient was sedated with a propofol infusion titrated to a bispectral index (BIS®; Aspect Medical Systems, Newton, MA) of 40–60. Neuromuscular blockade was maintained with cisatracurium to prevent shivering. Ice packs were placed around the head. Six liters of refrigerated normal saline were infused and the nasopharyngeal temperature decreased from 36°C to 33°C over 60 min. A femoral venous catheter with circumferential fluid reservoirs (ICY® catheter/Coolgard®, Alsius Corp., Irvine, CA) was placed for continuous cooling and maintenance of hypothermia (2).

The patient was transported to the neurointensive care unit and cooled to 32°C. This temperature was maintained for 20 h. Passive rewarming was allowed the next morning. Hyperglycemia requiring an IV insulin infusion was the only therapeutic complication. Arterial blood gases were managed by an  $\alpha$ -stat approach (no temperature correction). The patient was neurologically normal on awakening and went home the next day. On follow-up 2 wk later, she reported “fuzziness” of her memory for events over the preceding 2 mo. The memory complaints persisted at a 3-mo follow-up visit. The true necessity for hypothermic therapy and magnitude of effect will never be known.

## DISCUSSION

Our purpose here is to highlight a therapy unfamiliar to many anesthesiologists and to consider pertinent literature that may guide therapy in this setting. A critical error was made in the management of this patient. This has been peer reviewed extensively at our institution. Specifically, remifentanyl bolus dosing with a prone unsecured airway should be avoided. Nonetheless, events leading to hypoxic injury occur in all medical centers on rare occasions. For an anesthesiologist in this situation, knowledge of therapeutic options beyond cardiopulmonary resuscitation may provide benefit.

Although treatment of hypoxic brain injury with moderate hypothermia was investigated more than 45 years ago (3), prospective randomized trials only recently demonstrated efficacy in certain scenarios. To date, there is grade I evidence for benefit in comatose

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adults after out-of-hospital cardiac arrest (4,5) and for neonates with hypoxic-ischemic encephalopathy (6).

Our patient was quickly cooled to 33°C in the operating room with refrigerated saline from the malignant hyperthermia stock. She was then cooled to 32°C with a unique femoral catheter and cooling system (ICY® catheter/CoolGard®). This equipment is not available at most medical centers. However, cooling can be effectively achieved with IV infusions of chilled crystalloid (7). Surface cooling with air or liquid blankets can be effective for maintenance of hypothermia (8). Sedation and neuromuscular blockade are necessary to avoid recall and shivering. The risk of adverse events from hypothermia is very small (4,5,8,9). Coagulopathy, major infections, and other complications are expected with longer, more profound cooling (more than 24 hours, <32°C).

There is grade I evidence for the efficacy of hypothermia in improving neurologic outcomes in comatose survivors of out-of-hospital cardiac arrest (4,5) and neonatal hypoxia/ischemia (6). Clinical trials to test therapies for perioperative hypoxia are unlikely because of its rarity. The International Liaison Committee on Resuscitation has recommended that cooling to 32°C–34°C also be considered for in-hospital cardiac arrest (10). We believe that the same data should be extrapolated to the perioperative arena. We suggest that the immediate induction of moderate hypothermia for 12–24 hours be considered for comatose survivors of perioperative hypoxic brain insults.

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