

Chapter 43

Hypothermia During Laparoscopic Nephrectomy

Michael Bronson

The patient is a 38-year-old female (163 cm, 80 kg, body mass index [BMI] 30) presenting for laparoscopic nephrectomy for the purpose of donating a kidney to a relative. She had no significant past medical history and was taking no medications. Prior to induction, she received 800 mL of normal saline (NS) and 2 mg of midazolam while monitors were placed. Following preoxygenation, anesthesia was induced with fentanyl 500 mcg, lidocaine 100 mg, propofol 150 mg, and vecuronium 7 mg. Immediately after induction, the patient's blood pressure dropped from 130/80 to 85/45 mmHg which was treated with an intravenous fluid bolus of 500 ml NS and 200 mcg of phenylephrine. Intubation proceeded without incident, and the patient was placed in a lateral decubitus position.

Over the hour following induction, 4 L of NS and a total of 600 mcg of phenylephrine and 5 mg of ephedrine were given to maintain the blood pressure around 100/50 mmHg. The patient's temperature was also noted at this time to be 34.3 °C (**L-1, L-2, L-3**). Over the next hour, two more liters of NS were given, and the temperature gradually decreased to 33.6 °C (**L-1, L-2, L-3**). A forced-air warming blanket was placed on the patient, and the intravenous (IV) line was also connected to a fluid warmer at this time (**L-4**). At the end of the case the patient's temperature was 34.5 °C. The patient was transported to the postanesthetic care unit (PACU) intubated. Her temperature rose to 36.0 °C within 30 min and she was extubated uneventfully. Her postoperative course was uncomplicated.

M. Bronson, MD

Department of Anesthesiology, University of California, San Diego, San Diego, CA, USA
e-mail: michaelpbronson@gmail.com

Lessons Learned

L-1: In the absence of warming the patient, what is the expected change in temperature following induction of general anesthesia?

In a nonanesthetized patient, a temperature gradient exists between the core (37 °C) and periphery (31–35 °C) due to thermoregulatory vasoconstriction [1, 2]. The induction of anesthesia has a dramatic effect on this gradient as vasodilation from volatile and intravenous anesthetics leads to a rapid redistribution of blood from the core compartments (thorax/abdomen) to the periphery (arms/legs), resulting in a subsequent decrease in core body temperature (Fig. 43.1) [1, 2]. Therefore, the initial drop in temperature over the first hour following the induction of general anesthesia is a function of the redistribution of body heat rather than the physical process of heat loss.

Following the initial period of redistribution, the patient's core temperature still gradually decreases over the next 2–3 h. This decline occurs as heat loss to the environment exceeds internal heat production. Four processes that contribute to this heat loss are (in order of most important to least important):

1. Radiation: all objects above absolute zero radiate heat
2. Convection: transfer of heat from object to air/fluid
3. Conduction: transfer of heat between objects
4. Evaporation: heat loss from water vaporization

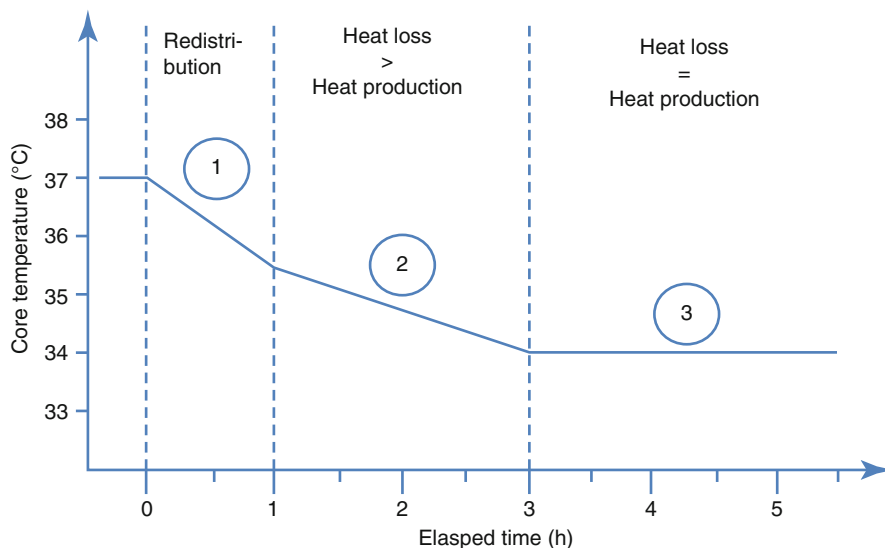


Fig. 43.1 Expected temperature changes following the induction of general anesthesia. Following induction, a temperature decrease of 0.5–1.5 °C occurs as blood is redistributed from the core components to the periphery from vasodilation (1). This is followed by a more gradual decline in temperature 1.0–1.5° over the next 2–3 h as heat loss remains greater than production (2). A state of equilibrium is achieved 3–4 h after the induction of anesthesia when heat loss becomes equal to heat production (3)

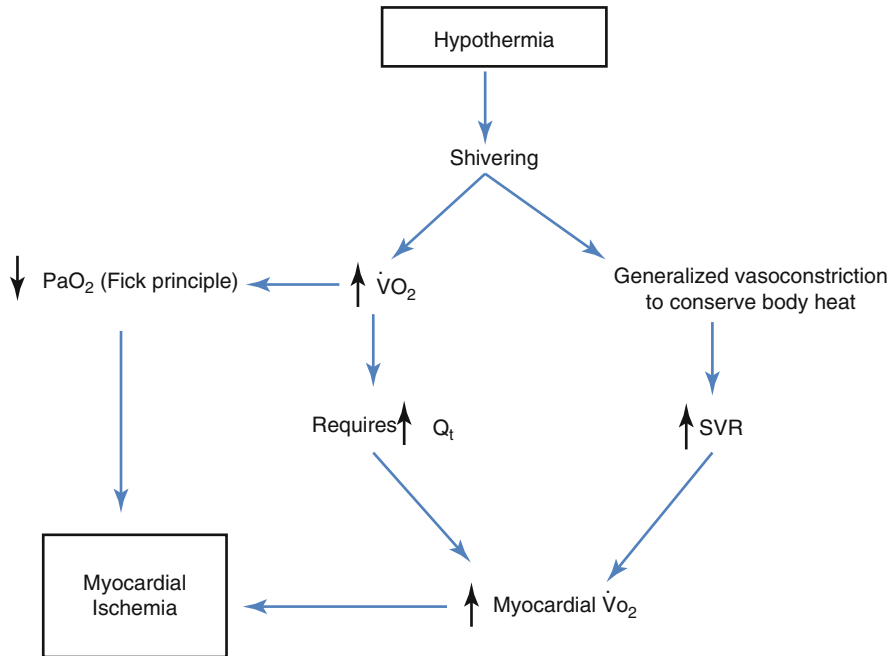


Fig. 43.2 Hypothermia can cause myocardial ischemia. Q_t , cardiac output, VO_2 oxygen consumption, SVR systemic vascular resistance

Approximately 3–4 h following induction of anesthesia, heat production becomes equal to heat loss so the patient's core body temperature will remain relatively constant. This state of equilibrium occurs as a function of cutaneous vasoconstriction.

L-2: What deleterious changes may occur if a patient becomes hypothermic?

Many adverse effects have been associated with hypothermia [1–3]:

1. Myocardial ischemia
2. Cardiac dysrhythmias
3. Wound infection/poor wound healing
4. Postoperative shivering
5. Peripheral vasoconstriction → increased systemic vascular resistance (SVR)
6. Coagulopathy
7. Increased blood loss and transfusion requirements
8. Decreased metabolism of drugs (muscle relaxants)
9. Increased PACU length of stay/decreased patient comfort

Myocardial ischemia is of particular concern (Fig. 43.2), especially in patients with known coronary artery disease (CAD) or risk factors for CAD. Postoperative shivering has been shown to increase oxygen consumption by up to 200 % resulting in a decreased PaO_2 and reducing overall oxygen supply to the myocardium [1–3].

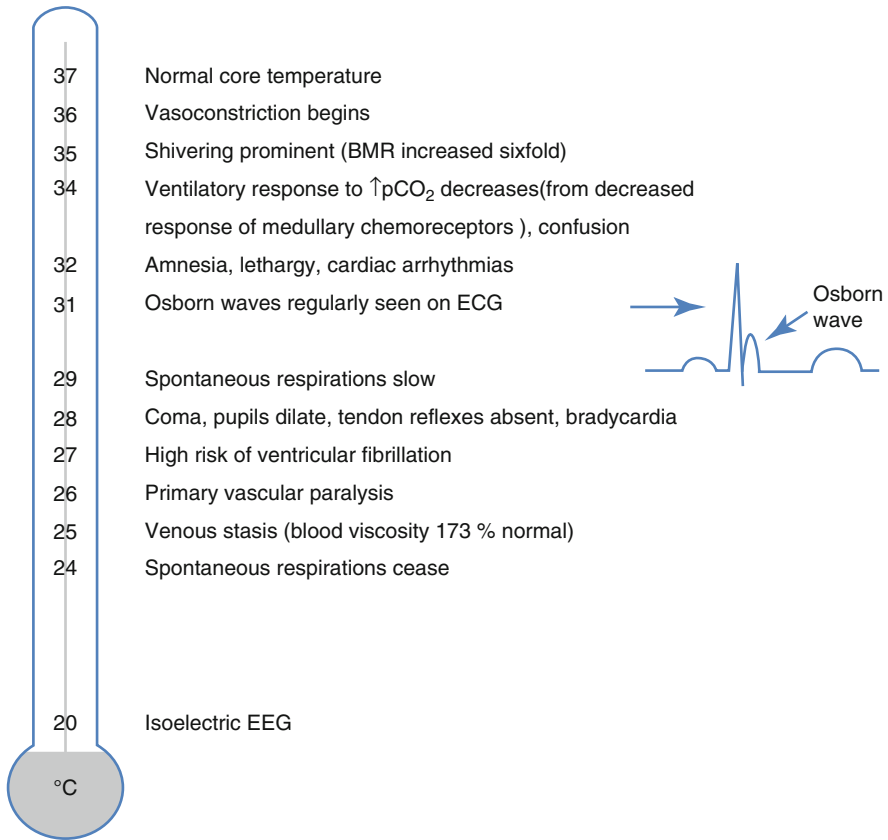


Fig. 43.3 Signs and symptoms of hypothermia (Adapted from [4])

This is coupled with generalized vasoconstriction that occurs as a physiologic response to conserve body heat. Consequently, an increased SVR places more after-load on the heart, which increases its demand. Therefore, both supply and demand of the myocardium become unfavorable during periods of hypothermia and as a result increase the risk of an acute coronary syndrome.

Many of the effects of hypothermia consistently occur at defined temperatures [4]. Figure 43.3 summarizes this correlation.

L-3: What is the effect on patient temperature following the administration of 1 L of IV fluid at room temperature (20 °C)?

It has been estimated that 1 L of crystalloid given at room temperature (20 °C) will result in a 0.25 °C decrease in the patient's core temperature [1]. As an example, in the case presented above, the patient received 6 L of crystalloid at room temperature before an IV warmer was connected; therefore, it is likely that the unwarmed IV fluids contributed to a 1.5 °C decrease in the patient's core temperature.

Table 43.1 Methods to warm a hypothermic patient (with reference to the patient)

1. Warm the OR to 23 °C (increases heat gain and decreases heat loss)
2. Place a forced-air warming blanket (increases heat gain and decreases heat loss)
3. Warm IV fluids (increases heat gain)
4. Humidify inspired gas (decreases heat loss)
5. Apply blankets/drapes to decrease surface area of exposed skin (decreases heat loss)

L-4: What are the methods to warm a hypothermic patient?

Methods aimed at warming a hypothermic patient are centered on two goals: (1) to transfer heat to the patient and (2) minimize heat loss from the patient (Table 43.1).

The most useful step in actively rewarming the patient is to increase the temperature of the operating room to donate radiant heat to the patient [1]. A forced-air warming blanket donates heat to the patient via convection currents. These methods of active rewarming also serve to minimize heat loss.

Intravenous administration of room temperature fluids contributes to ongoing heat loss (refer to L-3); therefore, connecting an IV warmer to the fluid line will help limit this. It is estimated that 10 % of heat loss occurs through respirations so applying an airway humidifier to the respiratory circuit should help decrease heat loss via this system [1]. Also, minimizing the exposure of the patient's skin to the environment is a key approach to maintaining body heat, so blankets and operating room drapes can be used to cover exposed areas.

References

1. Sessler DI. Temperature regulation and monitoring. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL, editors. *Miller's anesthesia*. 7th ed. Philadelphia: Churchill-Livingstone; 2010.
2. Sarti A, Recanati D, Furlan S. Thermal regulation and intraoperative hypothermia. *Minerva Anesthesiol*. 2005;71:379–83.
3. Putzu M, Casati A, Berti M, Pagliarini G, Fanelli G. Clinical complications, monitoring and management of perioperative mild hypothermia: anesthesiological features. *Acta Biomed*. 2007;78:163–9.
4. Benumof JL. *Anesthesia for pediatric thoracic surgery*. In: *Anesthesia for thoracic surgery*. 2nd ed. Philadelphia: W.B. Saunders Company; 1995.