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From Hypothermia Prevention to Hyperthermia Crisis: Iatrogenic Temperature Disturbance in Paediatric Anaesthesia

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KEYWORDS

Hypothermia Prevention, Hyperthermia Crisis

ABSTRACT:

Background: Maintenance of normothermia is a cornerstone of perioperative care in children, and active warming methods are routinely employed to prevent hypothermia. However, inadvertent hyperthermia can occur and may lead to serious complications.

Case Presentation: We report the case of a 10-month-old infant (8 kg, ASA I) scheduled for cleft palate repair. Anaesthesia was induced with sevoflurane, fentanyl, and atracurium, and maintained with sevoflurane in oxygen/nitrous oxide. Intraoperatively, active warming with a forced-air device was employed. After two hours, the child developed progressive tachycardia (HR 160–200/min) with warm peripheries. Rectal temperature measured 40 °C. Shortly thereafter, ventricular tachycardia was noted. Sevoflurane was discontinued, active cooling was initiated, and intravenous lignocaine, calcium gluconate, and furosemide were administered. The arrhythmia reverted to sinus rhythm, and temperature normalized with supportive measures. The child was extubated uneventfully and had a smooth postoperative recovery.

Conclusion: This case highlights the potential hazards of perioperative warming devices in infants, where high surface-area-to-weight ratio predisposes to rapid heat gain. Routine temperature monitoring is essential in paediatric surgeries, even for procedures of short expected duration, to detect and prevent iatrogenic hyperthermia.

INTRODUCTION

Perioperative hypothermia is a frequent concern in paediatric anaesthesia, associated with adverse effects on haemostasis, drug metabolism, wound healing, and recovery[1,2]. Active warming methods are widely used to mitigate this risk. However, hyperthermia is less commonly anticipated and may result in severe complications such as arrhythmias, coagulopathy, or neurological injury[3,4]].

We report a case of iatrogenic hyperthermia leading to ventricular tachycardia in an infant undergoing cleft palate repair, underscoring the importance of vigilant intraoperative temperature monitoring when using active warming techniques.

CASE PRESENTATION

A 10-month-old infant (weight 8 kg) with no comorbidities was scheduled for elective cleft palate repair under general anaesthesia. Pre-anaesthetic evaluation was unremarkable, with Hb 11 g/dL, TLC 6,000/mm³, BT 3 min, and CT 5 min. The child was ASA I and received oral midazolam (0.5 mg/kg) as premedication.

Standard monitors were applied (ECG, SpO_2, NIBP, $EtCO_2$). Inhalational induction was performed with sevoflurane (8% in oxygen), and intravenous access was secured. Anaesthesia was maintained with O_2/N_2O (33%:67%) and sevoflurane (1 MAC) using a Jackson Rees modification of the Ayres T-piece. Fentanyl (2 µg/kg) and atracurium (0.5 mg/kg) facilitated tracheal intubation with an uncuffed 4.0 RAE tube. Rectal paracetamol suppositories (250 mg) were placed for analgesia.

For the first two hours, vitals were stable (HR 125–135/min, BP 84/55–93/60 mmHg, SpO_2 99%). Subsequently, HR increased to 160/min, BP 108/60 mmHg, with $EtCO_2$ 35–38 mmHg. Fentanyl (1 µg/kg) and a fluid bolus (80 mL Ringer lactate) were administered. Despite this, HR rose steadily to 180/min, with warm peripheries. Rectal probe revealed a core temperature of 40 °C. Forced-air warming was discontinued. Cooling measures included tepid sponging, removal of drapes, and use of fans.

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The child developed ventricular tachycardia with palpable peripheral pulses (HR 200/min). Sevoflurane was discontinued, and the patient ventilated with 100% O_2. IV lignocaine (10 mg) was administered, resulting in reversion to sinus rhythm (HR 170/min). Arterial blood gas revealed: pH 7.43, $PaCO_2$ 41 mmHg, PaO_2 98 mmHg, Na^+ 130 mmol/L, K^+ 5.6 mmol/L, Ca^{2+} 1.13 mmol/L, HCO_3^- 19 mmol/L. Calcium gluconate (400 mg) and furosemide (4 mg) were administered.

Rectal temperature decreased gradually, reaching 37 °C over 90 minutes, accompanied by stabilization of HR (140–150/min). Total intraoperative blood loss was 30–40 mL; fluids administered were 390 mL, with urine output 40 mL. The child was extubated uneventfully and monitored in PACU for four hours, remaining afebrile and haemodynamically stable. The postoperative course was uneventful, and discharge occurred on postoperative day 2.

DISCUSSION

Active forced-air warming is widely endorsed to prevent perioperative hypothermia in children[1,2,5]. However, it carries the under-recognized risk of iatrogenic hyperthermia. A pooled analysis of paediatric patients ≤ 6 years demonstrated that 16.5% developed hyperthermia (> 38 °C) during intraoperative warming, with risk factors including younger age, high surface-area-to-weight ratio, prolonged warming, and higher baseline temperatures [6].

In our case, forced-air warming at 40 °C combined with disposable drapes, focused surgical lighting, and minimal evaporative losses from palate surgery contributed to progressive hyperthermia. The absence of early temperature monitoring delayed detection, and tachycardia was the first clinical clue. The episode culminated in ventricular tachycardia, likely triggered by sympathetic overactivity and hyperkalaemia. Features of malignant hyperthermia were absent (normal $EtCO_2$, lack of acidosis, rapid improvement with cooling) [7].

Hyperthermia has also been reported in paediatric patients during MRI, where radiofrequency energy absorption can overwhelm heat dissipation under anaesthesia [8,9]. Conversely, large series suggest that hypothermia remains more common in MRI settings [10]. These reports highlight the spectrum of perioperative temperature perturbations and the critical role of monitoring.

According to Bräuer *et al.*, paediatric perioperative hyperthermia is defined as core body temperature > 38 °C in children < 5 years [11]. Iatrogenic causes include excessive or prolonged use of active warming devices. As hyperthermia progresses insidiously, continuous core temperature monitoring with narrow alarm limits is recommended [11,12].

This case underscores two critical lessons:

1. **Routine temperature monitoring** must be instituted in all paediatric surgeries employing active warming, regardless of expected duration.
2. **Iatrogenic hyperthermia** should be managed by stopping heat sources and initiating cooling, rather than with antipyretics.

CONCLUSION

This case illustrates that while prevention of hypothermia remains crucial in paediatric anaesthesia, vigilance is also required to avoid inadvertent hyperthermia. Routine intraoperative temperature monitoring is essential when active warming devices are used in infants, to prevent potentially life-threatening complications such as arrhythmias.

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