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Does Close Temperature Regulation Affect Surgical Site Infection Rates?

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Keywords

• Intraoperative hypothermia • Surgical site infection • Surgical quality indicators

Key points

- Intraoperative temperature regulation is an increasingly important measure of surgical quality because of its association with surgical site infections.
- Several known biological effects support the observed clinical association between hypothermia and infection.
- Current clinical evidence challenges discrete thresholds for temperature control but supports rewarming and temperature monitoring.

INTRODUCTION

Close intraoperative temperature regulation has increasingly become part of the definition of high-quality surgical care over the last 2 decades. Seminal research at several centers showed that decreased body temperature during surgery is associated with increased total oxygen consumption [1], increased cardiac morbidity [2], increased coagulopathy [3], and increased surgical site infections (SSIs) [4,5]. As evidence for enhanced temperature control has accumulated, multiple health care quality efforts have applied these findings to regulatory interventions. Since 2006, maintenance of normothermia for patients has been part of the Surgical Care Improvement Project (SCIP) [6], a mandatory joint reporting scheme operated by the Joint Commission and the Centers for Medicare and Medicaid Services. The 2 measures, SCIP INF-10 and SCIP

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INF-7 (the latter measure is now retired and is used only for patients having colorectal surgery), require documentation of the use of a forced-air warming device in the operating room or monitoring of intraoperative temperature with minimum end-of-case temperature of 36.0°C for patients without documentation of a warming device. With the increased quality incentives under the US government's 2010 Patient Protection and Affordable Care Act, these quality metrics have become increasingly important to the ongoing operation and financial bottom line of US medical centers.

However, intraoperative temperature control requirements as found in national regulations and their application to surgical practice may not be aligned appropriately with current evidence. Overall, new evidence has mounted that increased compliance with surgical quality measures has not led to a reduction in SSIs [7]. Specific quality measures such as temperature control are now also being scrutinized more closely. The historical examination of temperature control began with observations of the association of SSIs in the absence of rewarming interventions. Although these findings provided early evidence of the importance of temperature, more recent evidence with improved methodologies has challenged this consensus. The 36.0°C target has been found to be less dichotomous than was previously thought.

This article assesses the conventional understanding of close intraoperative temperature control and specifically how the historical consensus may need to be revised in light of more recent findings. It begins by reviewing the physiologic basis from which clinical studies evolved and then shows how those principles were tested over the years through various clinical approaches.

PATHOPHYSIOLOGY PRINCIPLES

The importance of temperature for biochemical processes is well understood and the human body's physiologic mechanisms are optimized for a narrow temperature range around 37°C. Physiologic disturbances worsen as the body cools or warms significantly (±0.2°C) from this core mean temperature [8]. For hypothermic conditions, alterations of normal physiology include increased total oxygen consumption, induced coagulopathy, and immune system dysfunction.

Increased total oxygen consumption and decreased peripheral perfusion

In the intraoperative interval, the use of general anesthesia inhibits normal behavioral defenses to hypothermia, and thus requires complete autonomic regulation of core body temperature. Hypothalamic thermoregulation for hypothermia is a well-understood process of stepwise mechanisms that results from dispersed cold-temperature receptors triggering systemic vasoconstriction and shivering [8]. Vasoconstriction directly impairs oxygen delivery to tissue, and shivering has been shown to increase metabolic oxygen demands by 40% to 300% [1,9]. However, neither of these mechanisms is adequate to reestablish normothermia, even under normal physiologic circumstances. Thermoregulatory shivering in particular continues to increase metabolic demands but never achieves its goal of increasing core body temperature [10].