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Glycemic control in critically ill children: a review of current evidence and guidelines

Abstract

Glycemic control in critically ill children is a crucial aspect of intensive care management, as both hyperglycemia and hypoglycemia are associated with increased morbidity and mortality. This review discusses the pathophysiology of critical illness-induced dysglycemia, reviews pertinent literature on critically ill children, and shares insights from adult studies. Researchers have extensively studied tight glycemic control (TGC), yet its benefits in the burn and post-surgical subgroup in the pediatric critical care population remain uncertain due to heightened risks of hypoglycemia. The TGC-FAST trial has challenged established beliefs regarding glucose management, emphasizing the complex relationship between glycemic control, parenteral nutrition, and patient outcomes. Current guidelines from the Society of Critical Care Medicine (SCCM) recommend a judicious approach, focusing on moderate glycemic control (140-200 mg/dl) to prevent severe hyperglycemia without intensive lowering. Pediatric clinicians face challenges such as heterogeneity of disease population, immature blood-brain barrier development, and variations in institutional practice.

Keywords: Glycemic control, pediatric critical care, insulin use, stress hyperglycemia, tight glucose control.

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Introduction

A once-promising strategy to reduce ICU mortality has resulted in years of conflicting evidence and shifting guidelines. Researchers have linked both hyperglycemia and hypoglycemia to increased morbidity and mortality in critically ill children.[1,2] A single-center trial by Van den Berghe et al. in 2001 first sparked enthusiasm for tight glycemic control (TGC) after reporting a significant reduction in mortality with the implementation of intensive glucose management (80–110 mg/dL).[3] However, subsequent multicenter trials in adults failed to replicate these encouraging results. Rather, subsequent trials revealed a concerning increase in mortality due to severe hypoglycemia.[4] Twenty years later, Van den Berge et al. recently published the Tight Blood-Glucose Control without Early Parenteral Nutrition (TGC-FAST) trial, further challenging the benefits of TGC as the study identified no significant decrease in ICU stay or mortality. Their findings suggest that the initially perceived mortality difference may stem from the adverse effects of early total parenteral nutrition (TPN), rather than a true advantage of tight glycemic control.[5,6] Studies in pediatric critical care have shown mixed results. Researchers halted the HALF-PINT trial early after they observed an increase in hypoglycemia among participants in the lower-target group, raising concerns about overly aggressive glycemic management.[7]

Despite inconsistencies, interest in ICU glycemic control persists. Honarmand et al.,[8] published SCCM guidelines before the TGC-FAST trial, recommending a glucose target of 140–200 mg/dL and discouraging intensive glycemic control. Acknowledging that their review predated TGC-FAST, the guidelines suggest that this threshold and the more liberal range (180-215 mg/dl) from Gunst et al. may be safe. With evolving guidelines on early vs late parenteral nutrition and a growing understanding of stress hyperglycemia, this review examines key shifts in ICU glucose management over the past two decades, focusing on critically ill children. A comprehensive PubMed search was conducted to identify studies on glycemic control in critically ill children and adults, covering publications from 2000 to 2024. The search utilized Medical Subject Headings terms and keywords, including "pediatric critical care," "glycemic control," "tight glucose control," "stress hyperglycemia," "insulin therapy," "hypoglycemia," "hyperglycemia," "critical illness," "insulin-like growth factor binding protein 1," "continuous glucose monitoring," "parenteral nutrition," and "diabetes management in ICU." Priority was given to randomized controlled trials, observational studies, and systematic reviews evaluating glucose management strategies in critically ill pediatric patients, which were then compared with landmark adult trials to contextualize findings and guide clinical practice.

Epidemiology

Stress hyperglycemia (BG > 126 mg/dL) commonly occurs in the pediatric intensive care unit (PICU), affecting 78–86% of patients.[9,10] Blood glucose remains elevated throughout ICU admission, peaking at 19–23.5 hours (IQR: 5–236 hours), with non-survivors experiencing prolonged hyperglycemia.[10]

Pathophysiology

Before the Leuven trial,[3] clinicians primarily viewed moderate hyperglycemia in critically ill patients as an epiphenomenon or an adaptive response, requiring little to no intervention.[11] Studies linked hyperglycemia in non-diabetic patients to increased disease severity, yet management practices varied significantly.[1] This variability stemmed from concerns about therapy-induced hypoglycemia, a risk cited by 70% of pediatric intensivists.[12] Counter-regulatory stress hormones, including cortisol, glucagon, and catecholamines, drive hyperglycemia in this critically ill population alongside proinflammatory mediators such as IL-1, IL-6, and TNF-α. Additionally, administering exogenous corticosteroids, vasopressors, and dextrose-containing parenteral solutions further contributes to glucose dysregulation.[13] Increased hepatic gluconeogenesis—where glucagon plays a central role—and reduced insulin-mediated glucose uptake by skeletal muscles primarily drive stress hyperglycemia.[14,15]

Evidence for tight glucose control in the PICU

Researchers have pursued the elusive balance of maintaining euglycemia for over two decades. Results have varied due to heterogeneity in disease populations and hospital practices. In a study by Vlasselaers et al.,[16] TGC was linked to a significant reduction in mortality (3% compared to 6%, p=0.038) and shorter ICU stays (5.5 vs. 6.2 days, p=0.017). However, the study raised concerns for safety as there were higher rates of severe hypoglycemia (25% vs. 1%, p<0.0001) in critically ill children receiving intensive insulin therapy.

Following this, the SPECS trial in 2012[17] found no benefits in reducing mortality or infections among post-cardiac surgery patients but confirmed an increased risk of hypoglycemia. Similarly, Macrae et al.,[18] found no mortality benefits, and the HALF-PINT trial in 2017[7] reported no improvement in ICU-free days, leading to early termination of the study due to the higher occurrence of hypoglycemic events. Henceforth, current SCCM guidelines recommend a conservative glycemic target of 140-200 mg/dL in critically ill children.[8] The key findings of the trials are outlined in Table 1.

Table 1 Key clinical trials evaluating tight glycemic control outcomes in the pediatric ICU

Study & year	Design	Population	Intervention	Findings	Nutrition
Half-pint Trial (Heart and Lung Failure–Pediatric Insulin Titration) 2017	Multicenter RCT (35 centers)	Critically ill children aged 2 weeks -17 years receiving vasoactive support or IMV	TGC (80–110 mg/dL) vs. standard care (150-180 mg/dL)	Trial stopped early. No significant difference in ICU-free days (19.4 days [IQR 0 to 24.2] in TGC and 19.4 days [IQR, 6.7 to 23.9], respectively; p=0.58. increased hypoglycemia in the TGC group.	Over the first 8 study days, the GIR, the percentage of intake as enteral nutrition, and the level of total nutrition were similar in the two groups. While some patients did receive parenteral nutrition, it was not administered to all participants.
Macrae et al.[18]	Multicenter RCT- 13 centers	PICU patients ≤16 years who were expected to require IMV and vasoactive support for at least 12 hours	TGC (72-126 mg/dl) vs. standard care (BG < 216 mg/dl)	No significant difference in days alive and free from IMV at 30 days; increased hypoglycemia (7.3% vs. 1.5%, P<0.001) in the TGC group. In a secondary analysis, TGC was associated with a smaller proportion of patients receiving RRT and lower healthcare costs at 12 months (driven by the subgroup without cardiac surgery).	No comment was made on differences in nutrition intake between the two groups.
specs Trial (Safe Pediatric Euglycemia after Cardiac Surgery) 2012	Two-center RCT	Post-cardiac surgery children aged 0-36 months	TGC (80–110 mg/dL) vs. standard care (unit practices)	No significant difference in infection rates, mortality, LOS, and organ failure. Increased hypoglycemia in the TGC group.	Children in the TGC received a median of 41% (interquartile range, 19 to 65) of their total caloric intake enterally, and those in the standard-care group received 38% (interquartile range, 11 to 66) enterally, p=0.24).
Vlasselaers et al.[16]	Single-center RCT, evaluated the effect of targeting age-adjusted normoglycemia	Infants and children admitted to the PICU	TGC (Infants: 50.4–79.2 mg/dL, Children: 70.2–100.8 mg/dL) vs. standard care (< 214 mg/dl)	Reduced mortality (3% in TGC vs 6% in conventional, p=0.038) and length of stay; increased severe hypoglycemia in the TGC group.	Patients unable to receive enteral nutrition received intravenous glucose and Vamin-glucose, with fluid intake adjusted based on restriction needs. Enteral feeding was initiated as soon as possible—infants received breast milk or home formula, while older children were given a standard formula via a gastric tube in a 10-hour cycle.

Table 1 Findings of the main clinical trials that evaluate outcomes of tight blood glucose in the pediatric ICU. *IMV= Invasive Mechanical Ventilation, RCT= Randomized control trial, PICU= Pediatric intensive care unit, TGC= Tight glucose control, GIR= Glucose infusion rate, LOS= Length of stay, BG= Blood glucose.*

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Evidence for tight glucose control in specific subgroup populations

TGC and Neuro-cognitive development

Stress hyperglycemia compromises the integrity of the blood-brain barrier (BBB). Dysregulated glucose homeostasis interferes with brain development, impairing intelligence quotient, learning, memory, and executive function.[19] Extreme changes in glucose levels are especially concerning for children in the PICU, who are already at a higher risk for delirium and post-intensive care syndrome.

Sadhwani et al., followed up on the SPECS trial, examining the relationship between neurocognitive development and glycemic patterns in post-cardiac surgery patients. They found that, at one year of age, children who experienced moderate to severe hypoglycemia scored lower in cognitive, language, and motor skills on the Bayley-III test, even when accounting for other confounding factors.[20] By age three, although treatment groups showed no significant differences, those who had experienced hypoglycemia still demonstrated lower cognitive and motor function.[21] These findings are consistent with the Leuven trial's neurodevelopmental follow-up, which reported enhanced motor coordination and cognitive flexibility in children who received targeted glucose control.[22] While TGC does not consistently enhance long-term neurodevelopment, preventing hypoglycemia remains paramount for optimal cognitive and motor outcomes.

TGC following burn injury

A hallmark response to severe burn injury is the development of hyperglycemia, driven by stress-induced insulin resistance, increased gluconeogenesis, and impaired glucose uptake. Studies have linked uncontrolled hyperglycemia in pediatric burn patients to higher infection rates, delayed wound healing, increased catabolism, multiple organ failure, and a greater risk of mortality.[23,24]

Early research by Pham et al.,[23] demonstrated intensive insulin therapy to maintain normoglycemia (90–120 mg/dL) in severely burned children (defined as >30% of the total body surface area burned), reduced infection rates, and improved survival. Similarly, a cohort study from the University of Michigan found that TGC significantly lowered the incidence of pneumonia and urinary tract infections in pediatric burn patients.[25] The first RCT conducted in pediatric burn patients confirmed that intensive glycemic control significantly reduced infections, improved organ function, and alleviated burn-induced insulin resistance.[26] Insulin, an anabolic agent administered to attain tight glycemic control, has been demonstrated to improve bone mineral content and muscle strength in children with severe burn injuries.[27]

Despite these potential benefits, burn patients experience prolonged insulin resistance (persisting up to three years),[28] making them highly susceptible to glucose fluctuations. Furthermore, insulin administration has been shown to exacerbate hypermetabolism, as assessed by resting energy expenditure.[27] Thus, experts recommend careful management with current recommendations targeting glucose levels of 130–150 mg/dL.[26] Alternative pharmacologic approaches to attaining tight glycemic control in the burn population have been studied, including the biguanide metformin[29–32] and peroxisome proliferator-activated receptor (PPAR)-α agonist fenofibrate.[33] Further investigation to merit the safety and efficacy of these agents is warranted.

TGC in pre-existing diabetes

Critically ill children with pre-existing diabetes face a double-edged sword—strict glucose control increases hypoglycemia risk, while higher targets raise infection and metabolic instability. A key player in this metabolic dysregulation is Insulin-like Growth Factor Binding Protein 1 (IGFBP-1), a critical regulator of IGF bioavailability that is acutely modulated by insulin. In critical illness, hepatic insulin resistance and a heightened catabolic state drive elevated IGFBP-1 levels, exacerbating metabolic dysfunction.[34,35]

Intensive insulin therapy (IIT) in critically ill children has been shown to further increase IGFBP-1 levels, likely as a counterregulatory response to hypoglycemia and suppression of portal insulin.[36] Additionally, elevated IGFBP-1 is associated with increased catabolism, as reflected by a higher urea/creatinine ratio, suggesting that despite IIT, critical illness-induced catabolism and hepatic insulin resistance persist.[34] In adult ICU patients, the LUCID trial[37] showed that liberal glucose control (180–252 mg/dL) significantly reduced hypoglycemia (18% to 5%), yet researchers raised concerns about a potential increase in mortality. In contrast, the CONTROLING trial[38] individualized glucose targets based on HbA1c and found no mortality difference between liberal and conventional targets, reinforcing the need for personalized glucose management over a standardized approach. Despite this insight, large RCTs on glycemic control in children with diabetes are lacking, making it difficult to determine the most effective approach for this population.

TGC in post-operative cardiac patients

The Van den Berghe et al.,[3] transformed ICU glucose management, showing that intensive insulin therapy (80–110 mg/dL) reduced mortality in critically ill patients, particularly those with prolonged ICU stays (≥5 days). However, in their subgroup analysis, post-cardiac surgery patients with short ICU stays (≤3 days) saw no survival benefit. For more extended stays, TGC lowered sepsis, renal failure, and ventilation duration.

More recent studies have refined our understanding of TGC in post-cardiac surgery patients. A meta-analysis in diabetic cardiac surgery patients found strict glucose control (<140 mg/dL) reduced atrial fibrillation and sternal wound infections yet did not lower mortality or stroke rates and increased hypoglycemia risks.[39] In pediatrics, the SPECS trial confirmed that while TGC is feasible, it did not significantly impact infection rates or outcomes [17]. Reflecting this evidence, the Society of Thoracic Surgeons recommends maintaining blood glucose below 180 mg/dL for at least 24 hours postoperatively to balance benefits with hypoglycemia risks.[40] Thus, while TGC may reduce complications like atrial fibrillation, it lacks a mortality benefit and carries risks, leading to the widespread adoption of moderate glycemic control (140–180 mg/dL in adults, 140–200 mg/dL in children).[8]

Discussion

Previously regarded as a breakthrough in critical care, tight glycemic control now faces growing skepticism, mainly due to its consistent association with increased hypoglycemia. After decades of research, one may wonder—why glycemic control is still important for critically ill patients. The TGC-FAST trial in adults has reignited discussion, challenged age-old beliefs, and revealed the complex interplay between glycemic control, parenteral nutrition, and patient outcomes.[5] This adult trial demonstrated that strict glucose control did not improve ICU length of stay or mortality when early TPN was not employed. This finding is particularly relevant for neonates and patients undergoing cardiac surgery, who often depend on early parenteral nutrition for optimal recovery. Interestingly, findings from this trial also showed that TGC was associated with a reduced incidence of severe acute kidney injury (AKI) needing renal replacement therapy

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(RRT). Particularly relevant to the pediatric population in which those who exhibit hyperglycemia demonstrate a higher incidence of AKI and longer ICU stays.[41] Insulin therapy in the ICU places children at particular risk for the development of hypoglycemia which must be monitored diligently due to the catastrophic effects on neurocognitive outcomes. Although robust evidence supports euglycemia to improve outcomes in critically ill children, how glycemic control is safely attained remains yet to be determined.

Conclusion

Current evidence supports moderate glycemic control (140–200 mg/dL) in critically ill patients. Subgroup analyses reinforce the need for personalized glucose targets in pediatric burn patients, post-cardiac surgery patients, and children with pre-existing diabetes mellitus. Additionally, practitioners must be cognizant of the impact of hypoglycemia on long-term motor and cognitive development.

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Conflicts of interest

The author declares that there are no conflicts of interest.

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