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Perioperative Blood Transfusion in Pediatric Cardiac Surgery: A Literature Review

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***Abstract*—*Children undergoing cardiac surgery often require blood product support because of their unique physiologic, yet transfusion itself carries measurable risk. This literature review emphasizes evidence on transfusion indications, component selection, patient blood-management strategies, and future directions specific to the perioperative care of congenital heart disease in pediatric. Restrictive red-cell thresholds ≤7 g dL^{-1} appear safe for most biventricular repairs, whereas single-ventricle palliation may merit higher triggers (≈9 g dL^{-1}). Viscoelastic testing, near-infrared spectroscopy, and miniaturized cardiopulmonary bypass (CPB) circuits enable precision transfusion and reduced donor exposure. Adoption of multimodal conservation programs correlates with lower morbidity, shorter ventilation, and cost savings.*

***Keywords*—** *Pediatric Cardiac Surgery, Pediatric Cardiac Anesthesia, Blood Transfusion, Congenital Heart Disease*

I. Introduction

Pediatric cardiac surgery happened for >35,000 cases annually worldwide, with transfusion rates ranging from 40 % to 90 %, inversely related to weight and circuit priming volume. Infants have a circulating blood volume (CBV) ≈80–90 mL kg^{-1}, such that a 10 kg child experiences ~50 % hemodilution when connected to a standard CPB system primed with 300 mL. These anatomical and technical factors contribute to the substantial transfusion demand and call for customized blood management strategies.

II. Physiologic Considerations in Cardiac Surgery

Physiological Considerations in Congenital Heart Disease

1) Oxygen-delivery balance

Cardiac defects that create right-to-left shunting stimulate erythrocytosis and elevate the baseline hematocrit, complicating hemoglobin-based transfusion triggers. Anemia makes oxygen delivery (DO_2) vulnerable in newborns because they lack developed myocardial reserve and compliance.

2) Coagulation immaturity and CPB effects

Thrombocytopenia, qualitative platelet dysfunction, hypofibrinogenemia, and dilutional coagulopathy are all caused by CPB; additionally, neonates have reduced plasma vitamin K-dependent factor levels, which increases their risk of bleeding.

3) Pulmonary Hypertension

Pulmonary hypertension (PH) in children—whether idiopathic or associated with congenital heart disease—exacerbates perioperative risk. These patients are prone to right ventricular (RV) dysfunction, reduced preload tolerance, and catastrophic hypertensive crises. To avoid rises in pulmonary vascular resistance, it is essential to maintain sufficient oxygen delivery. In this context, restrictive transfusion strategies (Hb <7 g/dL) may be insufficient; many experts advocate a transfusion threshold of 9–10 g/dL to sustain oxygen delivery and optimize RV performance. PH also correlates with increased bleeding tendency and platelet dysfunction, necessitating careful hemostatic monitoring. Prophylactic pulmonary vasodilators (inhaled nitric oxide or sildenafil) and close intraoperative TEE assessment are often used adjunctively.

Indications and Thresholds for Blood Components

1) Red Blood Cells (RBCs)

Restrictive transfusion (Hb <7 g dL^{-1}) is not inferior to liberal strategy for stable biventricular repairs; single-ventricle palliation or uncorrected cyanosis warrants 9 g dL^{-1}. CPB prime targets a hematocrit ≥25 % to maintain DO_2 > 350 mL min^{-1} m^{-2}.

2) Platelets

Evidence is limited; institutional practice often transfuses when platelet count <100×10^9 L^{-1} during active bleeding or <50×10^9 L^{-1} without bleeding but profound hypothermia. Viscoelastic parameters (e.g., TEG MA <45 mm) better correlate with microvascular bleeding.

3) Plasma and Coagulation Factor Therapies

Fresh-frozen plasma (10–15 mL kg^{-1}) corrects prolonged prothrombin time >1.5× baseline; fibrinogen concentrate (30–60 mg kg^{-1}) achieves target 1.5–2 g L^{-1}. Prothrombin complex concentrates and recombinant factor VIIa (72–90 µg kg^{-1}) serve as rescue agents but carry thrombotic risk.

4) Cryoprecipitate

Recommended when fibrinogen <1.0 g L^{-1} or ROTEM FIBTEM A10 <10 mm, at 1 unit 10 kg^{-1} (≈5 mL kg^{-1}).

Monitoring and Triggers Beyond Hemoglobin

Near-infrared spectroscopy (NIRS) detects cerebral/somatic rSO_2 decline and may prompt transfusion when saturation <50 % or a 20 % drop from baseline. Retrospective cohorts show rSO_2 rises ≈10 % post-RBC transfusion even at Hb >7 g dL^{-1}, supporting physiology-based thresholds.

Viscoelastic tests (TEG/ROTEM) guide goal-directed therapy:
* R time >12 min → plasma;
* MA <45 mm → platelets;
* FIBTEM A10 <10 mm → fibrinogen.

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Risks and Complications of Transfusion

Table 1 summarizes acute and delayed hazards. TRALI and TACO remain uncommon (<1 % incidence) but increase ICU stay. All cellular products for neonates should be leukoreduced, irradiated when risk for TA-GVHD exists (e.g., DiGeorge anomaly), and preferably CMV-negative. Notably, TXA reduces postoperative blood loss by ~2 mL kg^{-1} but may raise postoperative seizure risk (1.6 % vs 0.2 %).

TABLE 1. Selected transfusion complications

Acute hemolytic reaction, Febrile non-hemolytic reaction, Allergic reaction, TRALI, TACO, Metabolic alkalosis, Hyperkalemia, Hypocalcemia, Sepsis, TA-GVHD, Alloimmunization

Blood Conservation and Patient Blood-Management (PBM) Strategies

1) Preoperative

Treat iron-deficiency and test coagulation; consider erythropoietin (600 IU kg^{-1} weekly) two weeks pre-op for non-urgent cases.

2) Intraoperative

* Retrograde autologous priming and reduced circuit volumes (≤150 mL).

* Acute normovolemic hemodilution (10–15 mL kg^{-1}).

* Cell salvage with continuous centrifugation devices processing ≥30 mL.

* Ultrafiltration (Z-BUF, modified ultrafiltration) to concentrate hemoglobin and coagulation factors.

* Antifibrinolytics: TXA 30 mg kg^{-1} loading + 10 mg kg^{-1} h^{-1} infusion; ε-aminocaproic acid alternative.

3) Postoperative

Algorithmic transfusion using Hb, lactate, rSO_2, and viscoelastic assays; minimize phlebotomy volume and flush losses. Comprehensive PBM reduces allogeneic exposure by 30–50 %.

Massive Transfusion and Hemostatic Resuscitation

Massive transfusion is weight-based: >40 mL kg^{-1} RBCs within 24 h or >20 mL kg^{-1} within 3 h. Suggested ratio 1:1:1 (RBC:FFP: platelet) initiated early, adjusted by viscoelastic feedback. Cryoprecipitate or fibrinogen concentrate administered when FIBTEM A10 <10 mm.

Special Scenarios

1) Neonates and Low-birth-Weight Infants

Micro-volume transfusion sets, washed irradiated RBCs, and priming blood <7 days old to mitigate hyperkalemia.

2) Single-Ventricle Palliation

Higher Hb threshold (≥9 g dL^{-1}) maintains systemic oxygen delivery in parallel circulation

3) Pulmonary Hypertension

Patients with PH have reduced cardiopulmonary reserve and elevated right-sided pressures, leading to increased transfusion needs. Maintaining a hemoglobin >9–10 g dL^{-1} and optimizing pulmonary vasodilation are key to prevent RV failure. Continuous hemodynamic monitoring and goal-directed transfusion based on tissue oxygenation and lactate clearance may help avoid crises.

4) Extracorporeal Membrane Oxygenation (ECMO) and Ventricular Assist Devices

Maintain Hb 10–12 g dL^{-1}; monitor plasma-free hemoglobin and antithrombin activity.

4) Genetic and Immunologic Considerations Patients with DiGeorge anomaly or post-transplant immunosuppression require irradiated products to prevent TA-GVHD.

Emerging Technologies and Future Directions

Ongoing trials evaluate hemoglobin-based oxygen carriers, pathogen-reduced platelets, low-dose TXA for seizure mitigation, and real-time, artificial-intelligence predictive models for transfusion need. Precision PBM algorithms integrating NIRS, lactate, and machine-learning risk scores show early promise in reducing unnecessary transfusion without compromising outcomes.

III. Conclusion

Transfusion in pediatric cardiac surgery is transitioning from empiric, volume-based practice to individualized, physiology-guided therapy. Adherence to restrictive hemoglobin thresholds, deployment of viscoelastic monitoring, and institution-wide PBM pathways safely decrease allogeneic exposure and associated morbidity. Future research should refine platelet and fibrinogen triggers, clarify long-term neurodevelopmental impact of hemostatic drugs, and leverage predictive analytics to deliver the right component, at the right dose, at the right time.

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