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REVIEW

Cardiopulmonary Bypass, Fluid Dynamics, and Fontan Palliation: Hemodynamic Interactions and Implications for Catheter-Based Interventions

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ABSTRACT

Fontan palliation creates a circulation in which systemic venous return reaches the pulmonary arteries without a subpulmonary ventricle. Thus, its performance is dependent on low pulmonary vascular resistance, unobstructed cavopulmonary pathways, appropriate ventricular filling pressure, and efficient flow distribution. Cardiopulmonary bypass (CPB) allows Fontan completion and associated cardiac procedures but may compromise this circulation through hemodilution, non-pulsatile flow, inflammation, capillary leakage, and microembolization. This review aimed to integrate Fontan hydrodynamics with CPB-related physiological changes and assess their implications for invasive hemodynamic assessment and catheter-based interventions. PubMed/MEDLINE and publisher-hosted records were searched from database inception to July 2026. Clinical, experimental, and modeling studies addressing Fontan hemodynamics, CPB, microemboli, computational fluid dynamics (CFD), four-dimensional flow magnetic resonance imaging (4D-flow MRI), catheterization, stenting, collateral embolization, and fenestration were assessed. Current evidence supports physiology-guided perfusion, controlled venous drainage, meticulous air management, and adequate oxygen delivery. When non-invasive assessments cannot determine pressure, resistance, collateral burden, or lesion significance, catheterization is still used. Stenting of the Fontan pathway or pulmonary artery obstruction, collateral embolization, and fenestration creation or closure may correct selected physiological abnormalities, but intervention should still be guided by mechanism rather than anatomy alone. CFD and 4D-flow MRI may improve patient selection and procedural planning, although prospective validation remains limited. The preservation of Fontan circulation requires a multidisciplinary strategy integrating surgery, perfusion, imaging, and congenital interventional cardiology. This integration may help distinguish reversible anatomical lesions from broader physiological failure and support more individualized catheter-based decision-making across the Fontan pathway.

Keywords: Cardiopulmonary bypass, catheter intervention, computational fluid dynamics, Fontan circulation, microembolism, single ventricle

INTRODUCTION

The Fontan procedure is the final palliative stage for congenital heart disease with a functionally single ventricle. Evolution from the atriopulmonary connection to the lateral tunnel and extracardiac total cavopulmonary connections have reduced early complications without restoring normal physiology. Systemic and pulmonary blood flows are arranged in series, and systemic venous return must cross the pulmonary vascular bed without a subpulmonary ventricle.[1,2] Improved survival has created a growing population of children and adults with Fontan circulation, shifting clinical priorities from operative survival to the preservation of long-term circulatory efficiency.[3,4]

Cardiopulmonary bypass (CPB) remains necessary in many Fontan procedures for intracardiac repair, pulmonary artery reconstruction, atrioventricular valve surgery, rhythm procedures, or de-airing. However, CPB alters rheology, vascular tone, endothelial function, and embolic exposure. In Fontan physiology, even modest increases in pulmonary resistance or ventricular filling pressure can reduce preload and cardiac output. These interactions are useful not only to surgeons and perfusionists but also to interventional cardiologists assessing postoperative or late Fontan failure, in which anatomical obstruction must be distinguished from broader physiological limitations.

This narrative review integrates CPB management, Fontan pathway performance, invasive hemodynamics, and catheter-based treatment, emphasizing how potentially correctable lesions can be separated from ventricular, pulmonary vascular, lymphatic, or systemic causes of failure. A targeted PubMed/MEDLINE search and publisher-record review was conducted from database inception to July 2026 using terms related to Fontan hemodynamics, total cavopulmonary connection, CPB, microemboli, vacuum-assisted venous drainage, computational fluid dynamics (CFD), four-dimensional flow magnetic resonance imaging (4D-flow MRI), catheterization, stenting, collateral

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embolization, fenestration, and cavopulmonary support. Moreover, scientific statements, systematic reviews, clinical cohorts, patient-specific modeling studies, and experimental CPB studies have been prioritized. Since evidence is heterogeneous and largely observational, mechanistic plausibility is distinguished from demonstrated clinical benefit.

Hydrodynamic Basis of the Fontan Circulation

Fontan flow can be conceptualized as the pressure difference between the systemic venous and pulmonary venous or atrial compartments divided by the combined resistance of the cavopulmonary pathway and pulmonary vasculature. This resistance involves the venae cavae, conduit or lateral tunnel, anastomoses, pulmonary artery branches, microcirculation, and pulmonary veins. The most restrictive segment functions as a bottleneck, and the correction of one lesion may expose another.[5] Ventricular systolic function remains important, but forward output cannot exceed the preload delivered through the pulmonary circuit. Diastolic dysfunction, atrioventricular valve regurgitation, arrhythmia, or increased atrial pressure can narrow the effective transpulmonary gradient and mimic or magnify pathway obstruction.

Respiration and intrathoracic pressure are integral to Fontan flow. Spontaneous inspiration, skeletal muscle pumping, and low atrial pressure support venous return, whereas positive-pressure ventilation, increased mean airway pressure, atelectasis, hypoxia, hypercapnia, acidosis, pain, and hypothermia may increase pulmonary vascular resistance or reduce the effective driving gradient. Thus, simply increasing the systemic arterial pressure or administering fluid may fail when pulmonary transit is the dominant limitation.

Geometry also determines energy efficiency. The collision of superior and inferior vena caval streams, sharp turns, focal narrowing, and conduit-pulmonary artery mismatch dissipate mechanical energy. Patient-specific CFD shows a substantial variation in power loss and hepatic flow distribution even among anatomically acceptable connections.[6] Y-graft and offset configurations may improve selected flow characteristics, but performance changes with variations in the caval flow ratio, respiration, exercise, growth, and pulmonary artery size. Thus, no geometry is universally superior.[7,8] Low-velocity recirculation may promote thrombosis, whereas excessive local wall shear may affect endothelial biology. Model outputs depend on segmentation, boundary conditions, wall assumptions, and viscosity models and thus require careful interpretation.[9]

CPB-Related Disturbances and Interventional Implications

CPB exposes blood to artificial surfaces, blood-air interfaces, surgical trauma, ischemia-reperfusion, and temperature shifts, activating the inflammatory, coagulation, and endothelial pathways. Vasoplegia, capillary leakage, and organ edema may follow.[10,11] Pulmonary interstitial edema is especially important after Fontan completion because a small increase in pulmonary vascular resistance can elevate systemic venous pressure while reducing ventricular preload. Associations between longer CPB duration and adverse outcomes should be carefully interpreted because bypass time often reflects anatomical complexity and additional procedures rather than an isolated toxic effect.

In children, circuit prime may represent a large fraction of the circulating volume, making hemodilution clinically relevant. Thus, pump flow, hemoglobin, arterial oxygen content, temperature, and metabolic demand should be managed as an integrated oxygen delivery strategy rather than by fixed flow or pressure targets alone. Low oxygen delivery may facilitate lactate accumulation and organ injuries, whereas excessive crystalloid and uncontrolled volume replacement can worsen tissue edema and venous congestion. Serial lactate, venous oxygen saturation, cerebral oximetry, acid-base status, and urine output are best interpreted as trends, particularly during rewarming when oxygen consumption increases.

Table 1. Mechanism-based assessment of common Fontan problems

Problem	Hydrodynamic consequence	Clinical or interventional implication
Fontan pathway or branch pulmonary artery obstruction	Increased resistance and energy loss; reduced preload	In low-flow states, a pullback gradient ≥1 mmHg or approximately 25% luminal narrowing may be clinically relevant; absence of a gradient does not exclude obstruction. Consider stenting when anatomy and hemodynamics are concordant.
Elevated pulmonary vascular resistance	TPG increases and a larger proportion of the transpulmonary pressure gradient is dissipated across the pulmonary vascular bed	If TPG exceeds approximately 6-8 mmHg, assess ventilation, gas exchange, acidosis, filling pressure, and reversible vasoconstriction together; interpret values >8 mmHg in the context of increased risk.
Elevated ventricular filling pressure	The effective pressure gradient for pulmonary flow is reduced	Assess diastolic function, atrioventricular valve regurgitation, rhythm, and systemic ventricular function
Venovenous collateral	Provides decompression at the cost of systemic desaturation	Do not close on the basis of saturation alone; determine whether the collateral is compensatory
Fenestration	Increases preload and cardiac output at the cost of desaturation	Perform test occlusion before closure; creation or enlargement may serve as bridge therapy in selected Fontan failure states
CPB-related edema or microembolism	May increase pulmonary resistance and organ injury	Optimize oxygen delivery, venous drainage, filtration, de-airing, and post-CPB lung recruitment

TPG: Transpulmonary pressure gradient, CPB: Cardiopulmonary bypass

Gaseous microembolization can arise from venous-line air entrainment, cardiotomy suction, low reservoir level, temperature changes, or vacuum-assisted venous drainage. The circuit components differ in gas removal performance, but these technical differences do not establish catheter intervention indications.[12-16] Their interventional relevance lies in the postoperative phenotype: pulmonary edema, impaired gas exchange, transient pulmonary vasoconstriction, or microembolic injuries may transiently increase Fontan pressure, reduce output, or worsen desaturation. Before these findings are attributed to fixed pathway obstruction, clinicians should reassess ventilation, gas exchange, volume status, rhythm, ventricular systolic and diastolic function, atrioventricular valve competence, and residual surgical lesions.

Transition From CPB to Fontan Physiology

Separation from CPB is the transition from pump-supported circulation to a preload-limited Fontan circuit. The lungs should be recruited without having excessive airway pressure, correcting acid-base disturbances, normalizing temperature, and optimizing rhythm. Transesophageal or epicardial echocardiography should assess ventricular filling, systolic function, atrioventricular valve competence, pathway patency, and residual air. An acceptable systemic arterial pressure does not exclude low Fontan flow. Moreover, venous pressure, lactate, central or mixed venous oxygen saturation, cerebral oximetry when available, urine output, and echocardiographic filling should be interpreted collectively.

Volume management requires discipline. Preload dependence does not justify unrestricted fluid administration. Excess volume increases venous pressure, promotes pleural and peritoneal effusions, worsens hepatic congestion, and impairs lymphatic drainage. When low output coexists with high venous pressure, additional fluid should not precede the evaluation for pulmonary vasoconstriction, pathway obstruction, ventricular diastolic dysfunction, atrioventricular valve regurgitation, tamponade, or the loss of atrioventricular synchrony.

Fenestration provides a controlled right-to-left shunt that can preserve cardiac output when Fontan pressure increases at the cost of arterial desaturation and paradoxical embolic risk. Lumped parameter modeling supports benefit in selected high-resistance conditions, but decisions remain patient-specific.[17] Off-pump extracardiac Fontan completion has been associated with less inflammatory exposure and favorable early recovery in selected series, although observational design and selection bias compromise inference.[18,19] When pulmonary artery reconstruction, valve repair, rhythm surgery, or intracardiac intervention is required, CPB may be considered a safer strategy.

Early postoperative venous hypertension can induce hepatic congestion, bowel wall edema, increased lymphatic production, protein-losing enteropathy, and plastic bronchitis. These complications rarely reflect a single stenosis; pathway resistance, pulmonary vascular disease, ventricular filling pressure, and lymphatic anatomy tend to interact. After excluding a correctable mechanical lesion, management should integrate ventilation, rhythm, inotropy, pulmonary vasodilation, and lymphatic assessment rather than escalating fluid or circulatory support alone.

Invasive Assessment and Catheter-Based Interventions

Catheterization complements echocardiography, computed tomography, and MRI when a definitive pressure profile, pulmonary vascular load, collateral burden, or lesion-specific gradient is required. Measurements should be interpreted in the context of anesthesia, ventilation, inspired oxygen, hydration, hemoglobin, and collateral flow. Fontan/systemic venous pressure, atrial or pulmonary capillary wedge pressure, transpulmonary pressure gradient (TPG), cardiac index, oxygen saturations, and anatomy should be assessed together rather than by a single threshold. In asymptomatic adults, resting Fontan pressure is generally below 15 mmHg and ventricular filling pressure below 10 mmHg. Thus, a Fontan pressure of ≥15 mmHg is best considered as a warning sign of abnormal hemodynamic burden rather than as an isolated intervention indication.[20-22]

A TPG of approximately 6-8 mmHg or higher requires attention to pulmonary vascular load, and values of >8 mmHg have been associated with cavopulmonary palliation failure in some cohorts.[23] However, a high Fontan pressure with a low TPG may instead reflect increased atrial or ventricular filling pressure. An accurate leveling and zeroing of pressure transducers, anatomically appropriate saturation sampling, and the documentation of respiratory conditions are essential because small absolute errors can materially affect interpretation in this low-pressure circuit.

Low flow across a Fontan pathway or pulmonary artery stenosis may produce little pressure drop despite important anatomical obstruction. Contemporary practice has used a catheter pullback gradient of ≥1 mmHg or approximately 25% luminal narrowing as evidence supporting significance, but the absence of a measurable gradient does not exclude clinically important obstruction.[20] Accordingly, even 1-2 mmHg gradients should be interpreted based on vessel caliber, flow, collateral burden, Fontan pressure, symptoms, and exercise capacity. Sedation and positive-pressure ventilation may further suppress flow and make resting gradients appear deceptively small.

Aortopulmonary collaterals (APCs) create pulmonary recirculation by delivering systemic arterial blood to the pulmonary vascular bed outside the cavopulmonary pathway. When the APC burden is substantial, the pulmonary venous return and total ventricular output can exceed the effective caval flow. Thus, conventional oximetric or Fick calculations may misrepresent systemic and pulmonary flows, Qp/Qs, and derived pulmonary vascular resistance. Interpreting total aortic or ventricular output as effective systemic flow can overstate tissue perfusion since recirculated collateral flow is included. Combined invasive pressures and phase-contrast CMR assessments of aortic, caval, pulmonary arterial, and pulmonary venous flows can better characterize this physiology.[24] Assumed oxygen consumption, differential caval saturations, fenestrations, and venovenous collaterals are additional sources of Fick error and should be documented.

Catheter-based therapy should target a physiological mechanism, not an angiographic appearance alone. Stenting of a significant conduit, lateral tunnel, caval pathway, or branch pulmonary artery obstruction can reduce resistance and improve flow.[25-27] Systemic-to-pulmonary collaterals may also increase ventricular volume load and perioperative

bleeding; selected embolization can be useful, whereas indiscriminate closure may remove compensatory pulmonary blood flow. Since venovenous collaterals may worsen cyanosis but decompress a high-pressure Fontan circuit, closure decisions require comprehensive hemodynamic assessments rather than oxygen saturation alone.[25,28]

Fenestration management illustrates a mechanism-based intervention. In a stable patient with acceptable Fontan pressure and preserved output, transcatheter closure may improve saturation; when reserve is uncertain, temporary test occlusion is appropriate. Conversely, creation or enlargement may provide rescue or bridge therapy in severe venous hypertension, low output, protein-losing enteropathy, or plastic bronchitis, although symptomatic response and long-term patency are variable. Contemporary adult Fontan practice also includes pathway stenting, collateral embolization, lymphatic interventions, and selected transcatheter AV procedures.[28,29] Moreover, these therapies are not substitutes for the treatment of pulmonary vascular disease, ventricular dysfunction, or advanced Fontan-associated liver disease.

Percutaneous cavopulmonary support devices designed to add energy to venous flow remain largely preclinical and face challenges of thrombosis, hemolysis, anatomical variability, and chronic implantation.[30] Timing also matters: the relief of obstruction after advanced organ congestion, severe liver disease, or marked ventricular dysfunction may yield limited clinical recovery, whereas earlier treatment of a clearly hemodynamically important lesion may prevent progressive energy loss and venous load. Since prospective thresholds for prophylactic intervention remain unavailable, decisions should integrate serial imaging, exercise capacity, organ findings, invasive pressures, and lifetime reintervention risk (Table 1).

CFD, 4D-Flow MRI, and Procedural Planning

CFD converts patient-specific anatomy into estimates of velocity, pressure drop, power loss, flow separation, wall shear stress, and hepatic flow distribution. 4D-flow MRI provides time-resolved three-dimensional velocity data that can define or validate model boundary conditions; the techniques are complementary rather than interchangeable.[31] Virtual stenting or geometric modification can determine whether relieving narrowing is likely to reduce power loss and how flow may redistribute, which is particularly useful when angiographic appearance and measured gradients disagree.

Modeling can also reveal lesions that appear minor at rest but become important as flow increases with exercise. A fixed-diameter extracardiac conduit may become relatively undersized as a child grows, and the respiratory phase, caval flow ratio, and hepatic flow distribution can influence the predicted performance. Clinical translation remains limited by imaging quality, boundary condition assumptions, computational time, and the lack of standardized outcome thresholds. A predicted reduction in power loss does not guarantee improved exercise capacity, venous pressure, or survival. Thus, CFD should support, not replace, invasive assessment and clinical judgment. Prospective work should also determine whether model-guided and digital twin approaches improve patient-centered outcomes.[32,33]

Practical Clinical Framework

Before Fontan completion, multidisciplinary assessment should define the pulmonary artery anatomy, collateral burden, pulmonary venous return, ventricular systolic and diastolic function, atrioventricular valve regurgitation, rhythm, pulmonary pressure and resistance, end-diastolic pressure, oxygen saturation, hemoglobin, and renal and hepatic status. Moreover, the need for preoperative catheter intervention, surgical reconstruction, fenestration, and CPB should be agreed upon before surgery. During CPB, the circuit should be appropriately sized, oxygen delivery monitored, venous drainage optimized before increasing vacuum assistance, and responsibility for air management clearly assigned.

After Fontan completion, spontaneous breathing can support venous return, but early extubation is unhelpful if it produces atelectasis, hypercapnia, or acidosis. Persistent high venous pressure or low output should prompt early imaging and, when uncertainty persists, catheterization for a correctable obstruction or unfavorable pressure relationship. Thromboprophylaxis is required because prosthetic material, low-velocity flow, endothelial activation, arrhythmia, and altered coagulation create a thrombogenic environment. Network meta-analyses support aspirin, warfarin, and direct oral anticoagulants over no prophylaxis, although pediatric comparative evidence remains limited.[34,35] Furthermore, therapy should be individualized according to thrombosis history, arrhythmia, fenestration, ventricular function, bleeding risk, organ function, and planned interventions.

Study Limitations

Evidence linking CPB variables, Fontan flow, and catheter intervention outcomes is largely derived from single-center cohorts, in vitro circuit studies, and computational models. Differences in anatomy, previous palliation, age, conduit type, fenestration, collateral burden, and center practice create substantial heterogeneity, while CPB duration and intervention requirements are strongly confounded by disease and procedural complexity. A standardized reporting of oxygen delivery, venous drainage pressure, microembolic burden, invasive hemodynamics, ventilation, transfusion exposure, prime volume, temperature strategy, residual lesions, and intervention timing is limited. Thus, these constraints reduce comparability and require caution when generalizing findings across patients and centers.

CONCLUSION

Fontan circulation operates within a narrow hemodynamic range in which pulmonary resistance, pathway geometry, ventricular filling pressure, and respiratory mechanics jointly determine the preload and cardiac output. CPB remains necessary in many patients, but its effects on inflammation, edema, oxygen delivery, and embolic exposure should be considered. Although catheter-based interventions can relieve important obstructions, manage collaterals, and modify fenestration physiology, technical success is meaningful only when the treated lesion materially contributes to circulatory failure. The most coherent strategy combines physiology-guided perfusion, high-quality imaging, invasive hemodynamics, and patient-specific flow analysis.

This may reduce anatomically successful but physiologically ineffective procedures in clinical practice.

Future research should integrate multicenter registries with prospective physiological data to refine perfusion targets, validate microembolic measures against neurological and pulmonary outcomes, and define the hemodynamic criteria for pathway stenting and fenestration management. A prospective evaluation of CFD, 4D-flow MRI, and digital-twin modeling should also determine whether these tools improve decisions and patient-centered outcomes. Furthermore, research linking the operating room, catheter laboratory, and long-term follow-up data may best identify the modifiable physiological and mechanical determinants of Fontan performance.

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