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THE REVIVAL

Promoting Academics to Improve Clinical Outcomes.

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EDITOR'S NOTE

Dr. Talha Meeran

MBBS, MD, FACC

Consultant - Cardiologist,
Dept of Advanced Cardiac Sciences and Cardiac Transplant
Sir HN Reliance Foundation Hospital, Mumbai

"The REVIVAL" is back after a small hiatus. Our team's focus remains on enhancing our understanding of topics related to advanced heart, lung failure and thoracic organ transplant.

The Jan 2026 edition of REVIVAL focuses on primary graft dysfunction post cardiac transplant. Primary graft dysfunction remains an enigma for cardiac transplant teams around the globe with a wide quoted prevalence of 10-30%.

This well written review was adapted from an outstanding SFHFT webinar delivered by Dr Chintan Sheth and his team from CIMS Hospital, Ahmedabad.

The review details the various approaches to managing PGD with particular focus on contemporary guidelines and recent literature updates.

Warm regards,
Dr. Talha Meeran

Please call or write to us:

Call: +91 91670 48815, +91 99994 45511

Email: talha.meeran@gmail.com, aditisinghvi4@gmail.com

Link for membership, http://www.sfhft.org/application.html

Special thanks to Dr. Chintan Sheth and Dr. Meet Kansagra for authoring this month's article.

Designed by Maithili Kulkarni

Society Reg.No.: EKM/TC/186/2013,

Society secretariat: Address 36/117, 2nd Floor, Lisie Hospital Road, Kottecanal Junction, Kochi 682018.
Ph.: 025750048,
Email: infoshft@gmail.com, www.sfhft.org

SUB EDITOR'S NOTE

Dr. Aditi Singhvi

FACC
Consultant - Heart Failure and Transplant Cardiology
Narayana Institute of Cardiac Sciences, Bangalore

It is a pleasure to present the January 2026 issue of "The REVIVAL", the academic newsletter of theSociety for Heart Failure and Transplantation. As the landscape of advanced heart failure and transplantation continues to evolve rapidly, our collective responsibility as clinicians is to remain informed, reflective, and forward-looking.

This issue focuses on Primary Graft Dysfunction after Heart Transplantation, a topic of immense clinical relevance and real-world impact. Despite advances in donor selection, preservation strategies, immunosuppression, and perioperative care, primary graft dysfunction remains one of the leading causes of early post-transplant morbidity and mortality. A clear understanding of its mechanisms, risk stratification, and evidence-based management is essential for improving outcomes.

We are grateful to Dr. Chintan Sheth and Dr. Meet Kansagra for their comprehensive and clinically oriented review, which synthesizes current evidence with practical insights from transplant critical care. Their contribution reflects the multidisciplinary collaboration that lies at the heart of successful transplant programs.

At SfHFT, our mission remains steadfast: to promote academic excellence, encourage collaboration across specialties, and translate knowledge into better patient outcomes. We hope this issue serves not only as an educational resource but also as a stimulus for discussion, innovation, and continued learning within the transplant community.

We thank our readers for their ongoing engagement and invite contributions, feedback, and ideas for future issues of ""The REVIVAL"

Warm regards,
Dr. Aditi Singhvi, FACC

PRIMARY GRAFT DYSFUNCTION AFTER HEART TRANSPLANT

AUTHOR

Dr Chintan Sheth

DNB, FICA, FTEAA

Cardiac Anesthesiologist

Co-Director - Heart Transplant Program, MARENGO CIMS Hospital, Ahmedabad

CO-AUTHOR

Dr Meet Kansagra

MBBS, DNB

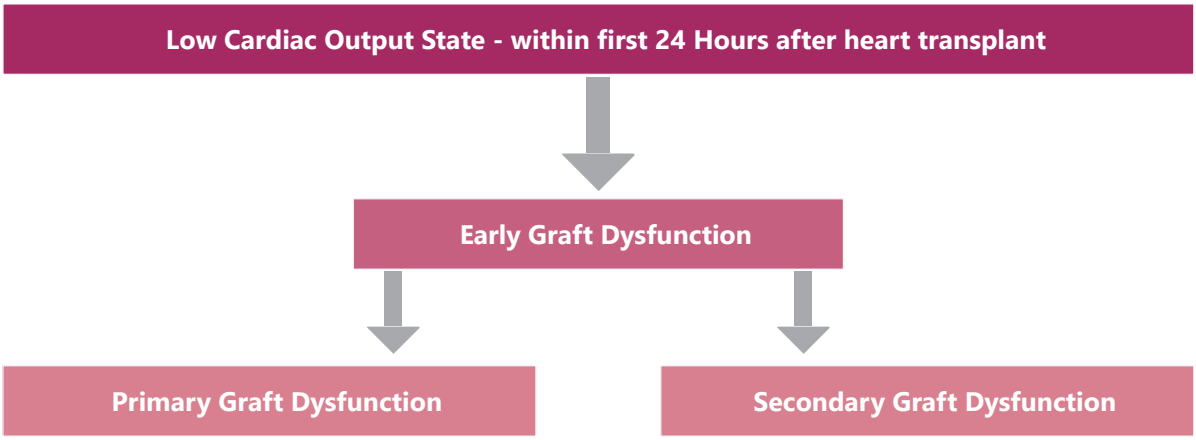
2nd yr Cardiac Anesthesia

MARENGO CIMS Hospital, Ahmedabad

Heart transplantation is the most effective treatment for end-stage irreversible heart failure. Globally, an annual average of 5000 heart transplantations are performed. Improvements in current treatment modalities and immunosuppression therapies have significantly improved postoperative survival. However, despite these advancements, transplant recipients remain vulnerable to early postoperative mortality attributable to early allograft failure. Primary graft failure is still the most common cause for 30 day mortality post heart transplantation.

Definition of Primary Graft Dysfunction

Any low cardiac output state in the first 24 hours after heart transplant is called early graft failure, which can be due to either primary or secondary graft dysfunction.



In 2014, Jon Kobashigawa[1] and colleagues proposed the ISHLT consensus definition of **Primary Graft Dysfunction (PGD).** According to this definition, the diagnosis of PGD requires **four essential criteria:**

1. **Ventricular dysfunction** – Left, right, or biventricular, insufficient to meet the recipient's circulatory needs
2. **Normal preload & afterload** – Suggests inotropic failure as the cause of low cardiac output
3. **Onset within 24 hours**
4. **Exclusion of secondary causes:**
    - Hyperacute rejection
    - High pulmonary vascular resistance leading to RV dysfunction
    - Anastomotic kinking or narrowing
    - Bleeding or tamponade

Grading of PGD[1] :

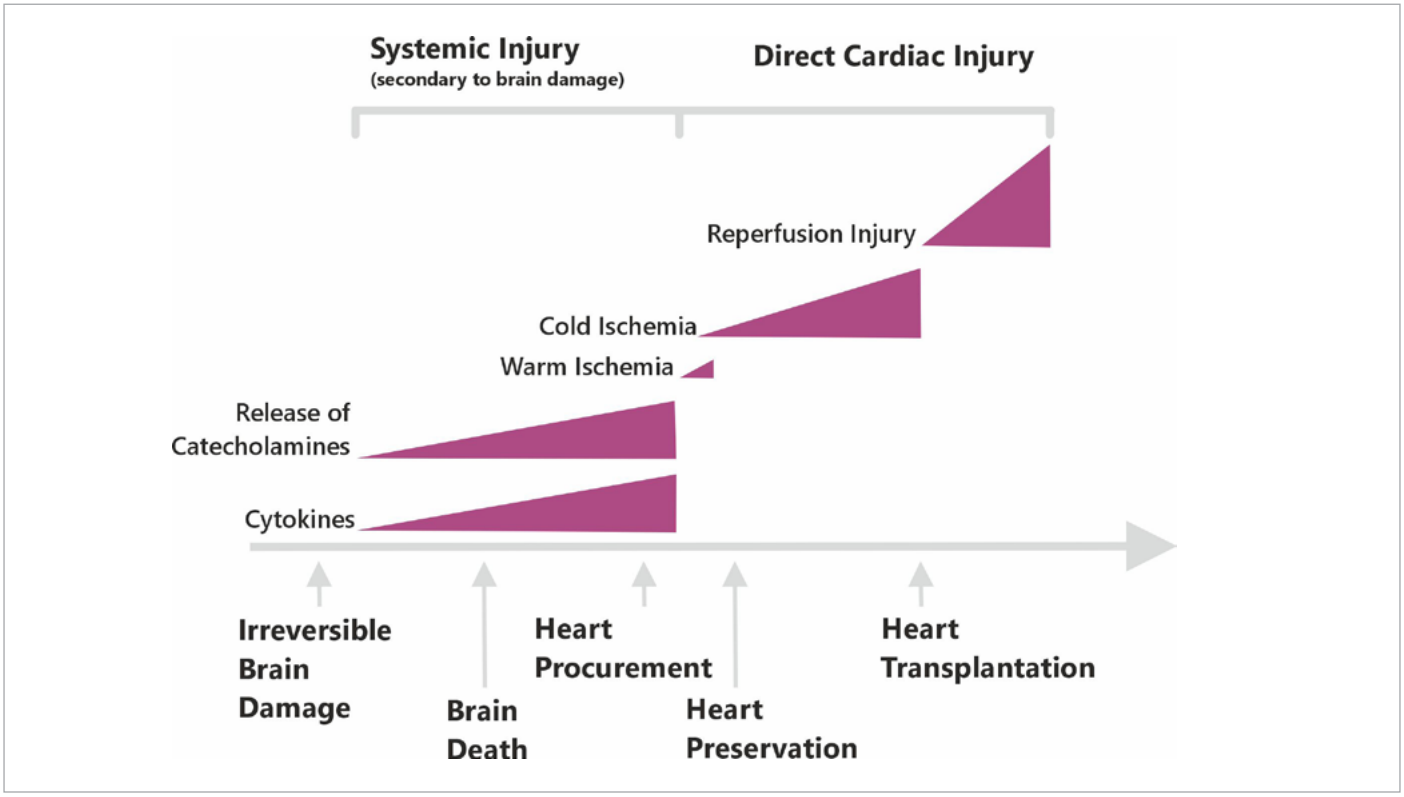
PGD LV	**Mild**	• LVEF ≤ 40% by echocardiography, or • Hemodynamics with RAP > 15 mm Hg, PCWP > 20 mm Hg, • CI < 2.0 L/min/m2(lasting more than 1 h) requiring low-dose inotropes
	Moderate **One criterion from 1 and one criterion from 2**	**1. Criteria** • LVEF ≤ 40%, or • Hemodynamic compromise with • RAP > 15 mm Hg, PCWP > 20 mm Hg, CI < 2.0 L/min/m2 • Hypotension with MAP < 70 mm Hg (> 1 h)
		2. Criteria • High-dose inotropes: Inotrope score > 10 or • Newly placed IABP (Regardless of inotropes)
	Severe	**Dependence on left or biventricular mechanical support including;** • ECMO, LVAD, BiVAD, or percutaneous LVAD
PGD RV	**Diagnosis requires either both 1 and 2, or 3 alone**	1. Hemodynamics withRAP > 15 mmHg, PCWP < 15 mmHg, CI < 2.0 L/min/m2
		2. TPG < 15 mmHg and/or sPAP < 50 mm Hg, or
		3. Need for RVAD

Inotrope score = dopamine (×1) + dobutamine (×1) + amrinone (×1) + milrinone (×15) + epinephrine (×100) + norepinephrine (×100) with each drug dosed in µg/kg/min.

Table 1: Grading of Primary Graft Dysfunction after Heart Transplant[1]

Pathophysiology of PGD

In the perioperative phase, the donor heart is susceptible to various forms of injuries resulting from brain death, cold ischemia during transport, warm ischemia during implant surgery, and reperfusion. Additionally, systemic factors within the recipient's body pose additional risks to the donor heart's function.[2]



Brain Death and Cardiac Effects[2]

Raised intracranial pressure with brainstem herniation causes **pontine ischemia,** triggering a **sympathetic storm** with massive catecholamine surge, increasing **LV afterload and myocardial stress.** This is followed by **spinal shock,** leading to **vasodilation, hypotension, and reduced coronary perfusion.** Both phases can damage the heart and require **prompt management.**

Hypothermic Ischemia in Donor Heart Preservation[2]

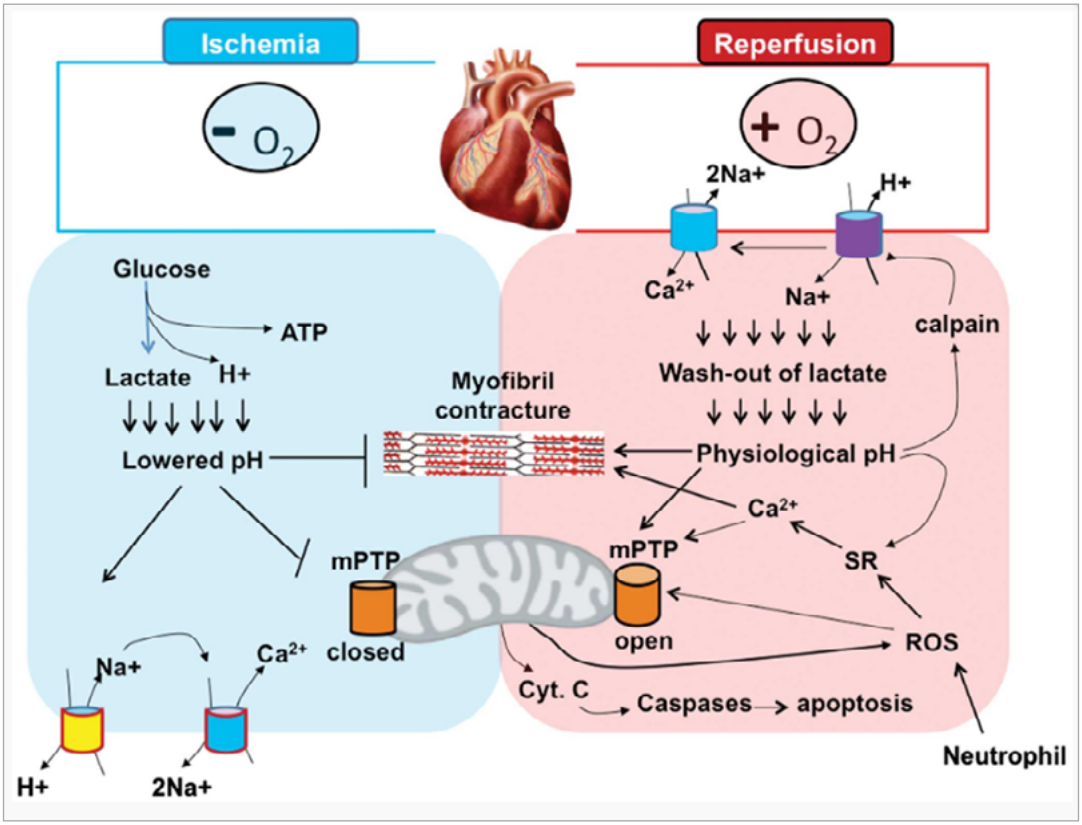
During donor heart procurement, cardiac arrest is induced with cold cardioplegia (~4 °C), and the heart is transported in hypothermic preservation solutions surrounded by ice. Hypothermia slows metabolism roughly 12-fold, but does not stop it, leading to progressive ischemic injury over time.

Warm Ischemia

Warm ischemia—from donor heart removal from cold storage to aortic unclamping—increases metabolism and free radical production, increasing the risk of primary graft dysfunction and early mortality.

Ischemia-Reperfusion Injury (IRI)

Reperfusion triggers calcium overload, oxidative stress, and mitochondrial pore activation, leading to apoptosis, necrosis, and **reperfusion-induced hypercontracture.** This can cause **contraction band necrosis, stiff ventricle, and hypercontracture-mediated sarcolemmal rupture (HMSR).**



Ischemia → ATP depletion → ion imbalance → cellular swelling → acidosis → primed ROS (Reactive Oxygen Species)

Reperfusion → Upon restoring blood flow to the graft → Sudden oxygen delivery → massive ROS generation (superoxide, hydrogen peroxide, hydroxyl radicals) **ROS burst + Ca^{2+} overload + inflammation → hypercontracture + necrosis → PGD**

Incidence and Prognostic Significance of PGD[3]

PGD is reported to occur in approximately 10–30% of heart transplant recipients and remains the leading cause of early mortality within the first 30 days following transplantation. The condition is associated with a short-term mortality rate ranging from 17% to 37%, while long-term mortality has been reported between 6.3% and 31.9%. Beyond its immediate impact, PGD has significant implications for long-term graft survival, as it predisposes recipients to an increased risk of acute rejection and the subsequent development of cardiac allograft vasculopathy (CAV).

Risk Factors for PGD After Heart Transplantation[1]

Category	Risk Factors	Mechanisms
Donor-related factors	Advanced donor age (>40–50 years)	Older myocardium more susceptible to ischemia–reperfusion injury.
	Female donor to male recipient (sex mismatch)	Associated with smaller LV mass and reduced coronary reserve.
	Donor–recipient size mismatch (undersized donor)	Disproportionate cardiac output requirement increases graft strain.
	Donor LV hypertrophy	Reflects chronic pressure load; reduced compliance and perfusion.
	Prolonged donor hypotension or high inotrope use	Leads to catecholamine-induced myocardial injury prior to procurement.
	Donor cardiac arrest / prolonged CPR	Associated with global ischemia and microvascular dysfunction.

	High donor serum sodium (>155 mmol/L)	Leads to osmotic stress and potential myocyte injury.
	Cause of donor death: trauma or stroke	Catecholamine surge and brain death physiology cause myocardial stunning.
	Donor left ventricular dysfunction on echo	Predictive of poor early graft performance.
	Use of marginal or extended-criteria donors	Includes donors with age > 55 years, LV dysfunction, or prolonged ischemia.
Recipient-related factors	Older recipient age	Reduced ability to tolerate ischemia/reperfusion insult.
	Female recipient sex	May influence immune and inflammatory responses.
	High pulmonary vascular resistance (>5 WU)	Increased RV afterload → RV-PGD risk.
	Pre-transplant mechanical circulatory support (VAD/ECMO)	Associated with systemic inflammation, coagulopathy, and sensitization.
	Renal dysfunction (elevated serum creatinine)	Indicates multisystem impairment; correlates with higher PGD risk.
	Diabetes mellitus, obesity	Promote endothelial dysfunction and microvascular injury.
	Prior cardiac surgery / re-sternotomy	Technical complexity, adhesions, and longer ischemic time.
	Recipient on inotropes or ventilatory support at transplant	Reflects critical illness and hemodynamic instability.
	Recipient hospitalized in ICU before transplant	Associated with systemic inflammation and poor reserve.
Operative / procedural factors	Prolonged total ischemic time (>4 h)	Major determinant of IRI (ischemia–reperfusion injury).
	Prolonged warm ischemic time (>60 min)	Especially detrimental for marginal donor hearts.
	Prolonged cardiopulmonary bypass duration	Leads to hemodilution, cytokine release, and myocardial edema.
	High transfusion requirements	Associated with inflammation and oxidative stress.
	Suboptimal preservation solution or technique	Inadequate myocardial protection exacerbates IRI.
	Use of non-optimal donor–recipient matching (ABO, size, ischemic risk)	Poor match may increase mechanical and metabolic stress.
	Excessive intraoperative catecholamine use	May exacerbate reperfusion injury and myocardial oxygen demand.
Post-operative factors	Reperfusion injury and oxidative stress	Central to PGD pathophysiology; manifests as ventricular dysfunction.
	Vasoplegia or systemic inflammatory response	Decreased coronary perfusion pressure and tissue edema.

Table 2: Risk Factors for Primary Graft Dysfunction (PGD) After Heart Transplantation[1]

RADIAL: A novel primary graft failure risk score in heart transplantation[4]

The RADIAL score incorporates six independent preoperative and donor-related risk factors, each assigned one point:

Right atrial pressure > 10 mmHg (recipient)

Age>60 years (recipient)

Diabetes

Inotrope dependence

Age> 30 years (Donor)

Length of ischaemic time>240 minutes

Radial Score	Incidence of PGD
Low risk (0-1)	10%
Intermediate risk (2)	20%
High risk >3	30%

Management of Mild PGD

Mild PGD is very common and often responds to optimization of preload, afterload, and inotropic support.

Inotropic support: Milrinone, Adrenaline, Dobutamine, or Levosimendan can be used to augment myocardial contractility and improve cardiac output.

Vasopressor therapy: Titrated according to systemic vascular resistance index (SVRI).

***Vasopressin is preferred over noradrenaline*, as vasopressin does not increase pulmonary vascular resistance (PVR), and many recipients have elevated PVR.**

Adjunct therapy:

Inhaled nitric oxide (iNO) may be administered to reduce PVR and improve right ventricular function.

Management of Moderate PGD

Moderate PGD requires escalation of pharmacologic support and insertion of IABP can be considered to restore adequate cardiac output and organ perfusion.

Intra-aortic balloon pump (IABP) is recommended when pharmacologic therapy is insufficient. IABP provides afterload reduction and coronary perfusion augmentation, typically resulting in a **5% to 30% increase in cardiac output (CO).**

Management of Severe PGD

Severe PGD, characterized by profound ventricular dysfunction unresponsive to pharmacologic support, necessitates mechanical circulatory support (MCS), including veno-arterial extracorporeal membrane oxygenation (VA-ECMO) or ventricular assist devices (VADs—left, right, or biventricular).

The choice of MCS ECMO versus VADS and timing of initiation remains a subject of ongoing debate.

VA ECMO versus VADs – VA ECMO preferred[5]

A study by Koji Takeda[5] et al showed that among 597 heart transplant recipients, 44 patients (7.4%) developed severe PGD. Of these, 17 patients received continuous-flow external VAD support, while 27 patients were managed with VA-ECMO within 24 hours post-transplant.

Temporary VAD implantation was associated with significantly longer cardiopulmonary bypass time compared with VA-ECMO (323 ± 86 min vs 216 ± 65 min, $p < 0.0001$), longer duration of mechanical support (14 ± 17 days vs 5.2 ± 3.9 days, $p = 0.011$), higher incidence of major bleeding requiring surgical reexploration (77% vs 30%, $p = 0.0047$), and increased postoperative renal failure requiring renal replacement therapy (53% vs 11%, $p = 0.0045$).

Overall hospital mortality was 27%, with mortality rates of 41% in the VAD group and 19% in the VA-ECMO group ($p = 0.16$). Successful weaning from support was achieved in 10 patients (59%) with VAD and 24 patients (89%) with VA-ECMO ($p = 0.03$). Three-year post-transplant survival was 41% for VAD and 66% for VA-ECMO ($p = 0.13$).

Conclusion:
In patients with severe PGD following heart transplantation, support with VA-ECMO appears to be associated with better short- and mid-term clinical outcomes compared with continuous-flow external VAD support.

Central vs Peripheral VA-ECMO

The choice between central and peripheral VA-ECMO depends on several factors, including the urgency of support, left ventricular (LV) contractility, the need for complete LV venting, institutional preference, and logistical availability.

In general, in emergency situations or when LV function is severely compromised (LVEF <10%), central VA-ECMO with surgical LV venting—either via a direct apical cannula or through the right superior pulmonary vein—is preferred. At our centre, we initially prefer **central VA-ECMO** with cannula tunnelling and chest closure, and if prolonged support is needed, we may transition to peripheral VA-ECMO once the patient is stabilized.

Below is a summary of the advantages and disadvantages of central and peripheral VA-ECMO:

Feature	Central ECMO	Peripheral ECMO
Advantages	- Faster installation in surgical settings - Antegrade aortic flow, preventing increase in LV afterload - Allows direct evaluation of cardiac function - Facilitates effective LV decompression - Hybrid approaches possible (e.g., tunneled cannula), allowing chest closure and patient mobilization	- Chest can remain closed - Reduced risk of infection and bleeding compared to central cannulation
Disadvantages	- Higher risk of infection and bleeding - Requires reoperation for decannulation - Difficult patient mobilization	- Retrograde aortic flow can increase LV afterload - Risk of limb ischemia (may require distal perfusion cannula) - Sometimes unable to provide adequate circulatory support - LV venting may require additional procedures (e.g., IABP, direct LV vent via minithoracotomy, percutaneous LA drainage, or Impella (ECPELLA) - Risk of Harlequin syndrome (north-south syndrome)

Early Versus Late VA-ECMO initiation: Early Preferred[6]

VA-ECMO is a key therapy for severe PGD after heart transplantation, but optimal timing remains uncertain. Scott C. DeRoo et al6 retrospectively analyzed 362 adult heart transplant recipients (2011–2017), of whom 38 (10.5%) developed severe PGD requiring VA-ECMO. Patients were divided into conservative ECMO (pre-2015, n = 18) and prompt ECMO (post-2015, n = 20) groups.

In the prompt (early) approach, VA-ECMO was initiated in the operating room for patients with mean arterial pressure <60 mmHg, central venous pressure >16 mmHg, or cardiac index <2.2 L/min/m^2 despite the use of more than two high-dose inotropes and two high-dose vasopressors. Median time to ECMO initiation was significantly shorter in the prompt group compared with the conservative group (1.95 vs 7.26 hours, P < .0001). In-hospital mortality decreased from 28% in the conservative cohort to 5% in the prompt cohort, while post-transplant survival at 1 year improved from 67% to 90%. Early and aggressive initiation of ECMO in patients with severe PGD results in excellent allograft recovery and suggests a potential reduction in mortality.

Isolated RV PGD after Heart Transplant

RV-PGD accounts for approximately 40–45% of all cases of PGD.

Differentiating RV-PGD from RV dysfunction secondary to elevatedPVR can often be challenging. In RV dysfunction due to high PVR, pulmonary artery pressures and PVR are initially elevated. The RV initially compensates by increasing

contractility, but progressive fatigue eventually leads to decreased RV contractility and stroke volume, which is then associated with low pulmonary artery pressure and elevated central venous pressure. In contrast, primary RV-PGD is characterized by persistently low pulmonary artery pressures from the onset.

Management of Isolated RV PGD

Target / Goal	Intervention / Strategy	Comments
Increase RV Contractility	Dobutamine, Adrenaline, Milrinone, Isoprenaline	Adrenaline is a good choice with severe RV systolic dysfunction and hypotension along with dobutamine
Reduce RV Afterload / Pulmonary Vasodilation	iNO, Milrinone, Sildenafil, Ambrisentan **RV-Friendly Ventilation strategies** Low PEEP, Low TV, Hyperoxia, Low-normal $PaCO_2$	iNO twhile patient is mechanically ventilated. Overlap iNO with IV sildenafil when planning extubation or after 72 hours of iNO, followed by oral sildenafil +/- ambrisentan
Optimize RV Preload	Target CVP 10–12 mmHg Early diuresis with loop diuretics and sequential nephron blockade Early Initiation of CRRT/SLED if needed	Avoid RV volume overload & over distension which decrease RV coronary perfusion pressure High CVP decreases tissue perfusion pressure as perfusion pressure = MAP - CVP
Maintain RV Coronary Perfusion	Maintain adequate mean pressure Vasopressin and /or norAdrenaline if vasodilated with low SVRI	Vasopressin is the preferred vasoconstrictor agent as it increases only SVR and has no effect on PVR, so it doesn't increase RV afterload, while noradrenaline increases both SVR and PVR, which increases RV afterload.
Maintain Sinus Rhythm	Cardioversion If required AV sequential pacing for atrial kick	
IABP Support	Early institution of IABP if indicated	Supports RV coronary perfusion In patients with RV dysfunction, elevated central venous pressure (CVP) reduces the effective perfusion pressure of vital organs such as the kidneys and splanchnic circulation, since perfusion pressure is determined by the gradient (MAP – CVP). The use of an IABP increases mean arterial pressure (MAP), thereby improving systemic and organ perfusion pressures. Enhanced renal perfusion promotes diuresis, which helps lower CVP, breaking the vicious cycle of venous congestion and impaired organ perfusion commonly seen in RV dysfunction.
MCS IABP Support with ECMO or RVAD [7]	For severe RV dysfunction not responding to above therapy VA ECMO RVAD with Centrimag, Impella RP, Protek Duo	**Centrimag (RA – PA)** Percutaneous Femoral Vein / direct RA cannulation for drainage and Pulmonary Artery graft and cannulation for returning. **Patient Unstable: Peripheral VA ECMO** with Femoral Vein & Artery cannulation **Patient relatively stable:** Shift to Cath Lab : Protek Duo /Impella RP

Plasmapheresis for treatment of PGD

Yosef Manla[8] and colleagues analyzed 42 patients with severe PGD on VA-ECMO, comparing outcomes between those treated with therapeutic plasma exchange (TPE) and those who were not. TPE was associated with higher 30-day (78.1% vs. 40%, p = 0.007) and 1-year survival (56.3% vs. 30%, p = 0.035), without significant differences in rejection, cardiac allograft vasculopathy, or major adverse events. TPE may be a promising strategy to improve survival in severe PGD post-transplant.

Key Takeaways:

Incidence of PGD is increasing as marginal donor heart usage and MCS usage as bridge to transplant are increasing.

Common risk factors for PGD:

- Recipient on MCS/ high inotropes pre transplant
- Recipients on amiodarone
- Undersized donor heart (PHM> -30%) , PHM D/R <0.8
- Long ischemia time > 4 hours
- Peri-operative excessive blood transfusions

Early IABP insertion when persistent low cardiac output with rising lactate even after optimization of inotropes / preload/ vasopressors is recommended.

VA-ECMO better over VADs for severe PGD

Early ECMO initiation better than late ECMO initiation

Central VA-ECMO preferred over peripheral VA-ECMO, especially when severe LV systolic dysfunction and patient is in OR.

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EDITORIAL TEAM

CARDIOLOGY

DR. TALHA MEERAN

DR. ADITI SINGHVI

PULMONOLOGY

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CARDIOTHORACIC SURGERY

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DR. DEBASIS DAS

ANESTHETIST/ INTENSIVE CARE

DR. PRADEEP KUMAR

DR. JACOB ABRAHAM

PRESIDENT

DR. JABIR ABDULLAKUTTY

Mob: +91 94470 11773

Email: drjabi@yahoo.co.in

VICE PRESIDENTS

DR. P CHANDRASEKHAR

Mob: +91 94430 47152

Email: chanpad@gmail.com

DR. JAYAGOPAL P B

Mob: +91 98470 23777

Email: jaigopallakshmi@gmail.com

SECRETARY

DR. MANOJ DURAIRAJ

Mob: +91 98223 22072

Email: manojdurairaj@hotmail.com

JOINT SECRETARY

DR. BASHA KHAN

Mob: +91 82968 25550

Email: bashajalal@gmail.com

DR. RAJESH MURALEEDHRAN

Mob: +91 94462 33343

Email: muralidharan.rajesh@gmail.com

TREASURER

DR JACOB ABRAHAM

Mob: +91 9847128123

Email: jacabraham1@gmail.com

IMMEDIATE PAST PRESIDENT:

DR. JULIUS PUNNEN

Mob: +91 99800 72785

Email: jpunnen@hotmail.com

PAST PRESIDENTS PERMANENT:

DR JOSE CHACKO PERIAPURAM

Mob: +91 9847043224

Email: joseperiapuram@hotmail.com

DR K VENUGOPAL

Mob: +91 9847005444

Email: venugopalknair@gmail.com

PAST PRESIDENTS

DR GEEVAR ZACHARIAH
(2013-2014 and 2014-2015)

Mob: +91 9846066816

Email: geevarzachariah@gmail.com

DR SHIV K NAIR (2015-2016)

Mob: +91 9846010220

Email: Shivnairmd@gmail.com

DR K VENUGOPAL (2016-2017)

Mob: +91 9847005444

Email: venugopalknair@gmail.com

DR JOSE CHACKO PERIAPURAM (2017-2018)

Mob: +91 9847043224

Email: joseperiapuram@hotmail.com

DR P P MOHANAN (2018-2019)

Mob: +91 9846076006

Email: drppmohanan@yahoo.com

EC MEMBERS

DR. NANDAKUMAR

Mob: +91 98430 15888

Email: drvnandakumar@gmail.com

DR. KUNAL SARKAR

Mob: +91 98300 80006

Email: kunal.cardiac@gmail.com

DR. GOVINI BALASUBRAMANI

Mob: +91 94449 32999

Email: dr.govini@gmail.com

DR. DHAVAL NAIK

Mob: +91 90991 11133

Email: dhavalnaik75@googlemail.com

DR. VIVEK PILLAI

Mob: +91 94470 07678

Email: drvivekpillai@yahoo.co.in

DR. PAUL RAMESH

Mob: +91 98400 70824

Email: paulramesh@gmail.com

DR. PRADEEP KUMAR

Mob: +91 98996 23669

Email: dr.pradeep.kumar.singh@gmail.com

DR. JO JOSEPH

Mob: +91 94008 74546

Email: jopoonjar@gmail.com

DR. TALHA MEERAN

Mob: +91 91670 48815

Email: talha.meeran@gmail.com

DR. ARUN GOPI

Mob: +91 81570 47371

Email: arungopi@gmail.com

DR. ADITI SINGHVI

Mob: +91 99994 45511

Email: aditisinghvi4@gmail.com

DR. DEBASIS DAS

Mob: +91 98318 52211

Email: dasdeba@yahoo.com