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PATHOPHYSIOLOGY, SURGICAL STRATEGIES, AND ADVANCED PERIOPERATIVE MANAGEMENT OF LATE DEVICE EXPLANTATION FOR LEFT BUNDLE BRANCH BLOCK AND LEFT VENTRICULAR DYSFUNCTION FOLLOWING TRANSCATHETER VENTRICULAR SEPTAL DEFECT CLOSURE.

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ABSTRACT Transcatheter device closure of ventricular septal defects (VSDs) has emerged as an alternative to surgical repair. However, its proximity to the cardiac conduction system can induce delayed electrical and mechanical complications. This article reviews the anatomical and mechanical pressure causations of late-onset Left Bundle Branch Block (LBBB) and subsequent Left Ventricular (LV) dysfunction following perimembranous VSD device deployment. We examine the biophysical pathways of dyssynchrony-induced cardiomyopathy, present advanced preoperative diagnostic workups, and outline surgical planning and intraoperative dissection protocols for device explantation. Furthermore, we address perioperative mechanical circulatory support (MCS) configurations, such as Extra-Corporeal Membrane Oxygenation (ECMO) and Ventricular Assist Devices (VADs). Finally, we analyze the therapeutic role of Cardiac Resynchronization Therapy (CRT) alongside future trends in biomechanical engineering and electrophysiology.

KEYWORDS :

1. INTRODUCTION

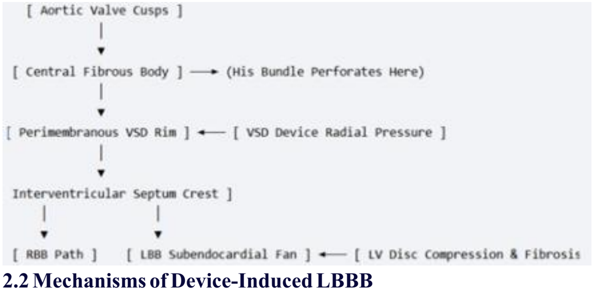
Transcatheter closure of perimembranous ventricular septal defects (pmVSD) provides a minimally invasive approach to congenital left-to-right shunts. However, the nitinol-mesh double-disc occluder sits close to vulnerable structures, specifically the bundle of His and the left bundle branch system. While complete atrioventricular block (CAVB) is a recognized acute risk, late-onset Left Bundle Branch Block (LBBB) leading to progressive Left Ventricular (LV) systolic dysfunction remains a rare but complex long-term challenge. This review evaluates the structural and pressure-driven mechanisms behind this pathology, establishes structured surgical remediation guidelines, and discusses advanced mechanical and electrophysiological support systems.

2. Anatomical and Pressure Causations of LBBB and LV Dysfunction

2.1 Conduction System Anatomy and Device Topography

The membranous septum is bordered by the right coronary cusp and non-coronary cusp of the aortic valve superiorly, and the septal leaflet of the tricuspid valve inferiorly. The bundle of His courses along the posterior-inferior margin of a perimembranous defect before perforating the central fibrous body.

Upon reaching the crest of the muscular interventricular septum, it branches into the right bundle branch (RBB) and the broad, fan-like left bundle branch (LBBB) system. The proximal main left bundle branch descends along the subendocardial surface of the left ventricular septum, rendering it highly susceptible to mechanical disturbances from the left ventricular retention disc of a VSD occluder.



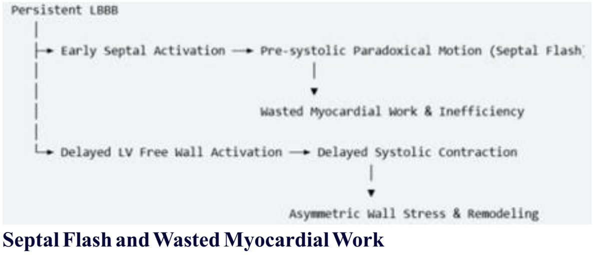
2.2 Mechanisms of Device-Induced LBBB

The transition from a normal conduction profile to a persistent LBBB involves three main mechanical processes:

- Direct Mechanical Compression: The radial expansion force of a nitinol device exerts continuous pressure against the margins of the VSD. If the defect extends into the muscular inlet or down the septum, this expansion directly compresses the subendocardial conduction tissue.
- Chronic Localized Inflammation and Fibrosis: The presence of a foreign nitinol-mesh matrix triggers a localized foreign-body response. Over several months, chronic inflammation gives way to dense collagen deposition and fibrosis, scarring the left bundle branch branches.
- Nitinol Shape-Memory "Spring" Kinetics: Nitinol devices undergo continuous micro-elastic expansion and contraction during each cardiac cycle. This cyclic shearing force along the septal endocardium causes micro-trauma, local ischemia, and conduction delays.

2.3 Biophysical Mechanisms of Progressive LV Dysfunction

LBBB alters the standard electrical activation sequence, decoupling normal interventricular and intraventricular mechanical performance through several distinct pathways:



Septal Flash and Wasted Myocardial Work

Electrical activation bypasses the left bundle, causing the interventricular septum to depolarize early via right-to-left trans-septal propagation. The septum contracts before the rest of the ventricle, during late diastole or pre-systole, resulting in an unproductive, paradoxical inward motion ("septal flash"). When the delayed LV free wall finally contracts during ejection, the septum is pushed back toward the right ventricle, wasting a portion of the regional myocardial energy.

Altered Wall Shear Stress and Pathological Remodeling

This mechanical asynchrony creates an uneven distribution of workload. The early-contracting septum faces low wall stress and undergoes disuse atrophy over time. Conversely, the late-activating LV free wall undergoes forceful contraction against an already-elevated wall tension, increasing local wall shear stress. This triggers eccentric hypertrophy, myofibrillar disarray, and localized chamber dilation, progressing toward heart failure.

Decreased Diastolic Filling Efficiency

Conduction delays prolong the overall duration of LV systole, truncating the subsequent diastolic filling period. Late free-wall contraction disrupts normal ventricular relaxation, which reduces the early diastolic filling velocity E, elevates end-diastolic filling pressures E/e', and decreases global cardiac output.

3. Preoperative Advanced Investigations

Evaluating a patient for device explantation requires detailed structural, functional, and metabolic imaging to quantify the degree of mechanical dyssynchrony and map device-to-tissue interfaces.

3.1 Advanced Echocardiography

Speckle-Tracking Echocardiography (STE): Quantifies the Left

Ventricular Global Longitudinal Strain (GLS). It monitors regional dyssynchrony by calculating the Standard Deviation of Time-to-Peak Longitudinal Strain across an 18-segment model T_SD.

- Real-Time 3D Echocardiography (3DE): Measures the Systolic Dyssynchrony Index (SDI), defined as the standard deviation of the time taken for individual ventricular segments to reach their minimum systolic volume. An SDI > 5.6 signifies severe mechanical dyssynchrony.

3.2 Cardiac Magnetic Resonance Imaging (CMR)

- Late Gadolinium Enhancement (LGE): Detects focal myocardial fibrosis or scarring surrounding the VSD device rims and within the LV free wall, which helps determine whether the myocardial injury is reversible.
- Myocardial Feature Tracking (CMR-FT): Provides high-resolution 3D strain mapping (radial, circumferential, and longitudinal) without needing specialized echo acoustic windows.
- Phase-Contrast CMR: Quantifies residual trans-device shunts Q_p/Q_s and checks for concurrent aortic valve insufficiency caused by upper retention disc protrusion.

3.3 Electrophysiology Study (EPS)

An electrophysiology study maps the His-ventricular HV interval and differentiates a proximal main stem LBBB block from distal arborization disease. It evaluates sinus node recovery times and AV node conduction margins to help guide post-surgical pacing plans.

4. Preoperative Planning and Patient Preparation

4.1 Surgical Risk Stratification and Anatomical Assessment

Preoperative planning relies on multi-planar CT or CMR imaging to analyze the device's

Risk Parameter	Key Anatomical & Hemodynamic Benchmarks	Clinical Implications
Aortic Valve Proximity	Distance from upper device rim to aortic valve annulus < 2 mm	Risk of aortic leaflet perforation during extraction; prepare for valve repair.
Tricuspid Valve Entanglement	Chordal tissue or septal leaflet tissue incorporated into the RV disc	High risk of tricuspid regurgitation; requires meticulous chordal separation.
Ventricle Size	End-Diastolic Volume Index (EDVI) > 100 ml/m^2	Indicates advanced eccentric remodeling; higher likelihood of requiring post-op inotropic support.
Systolic Function	Left Ventricular Ejection Fraction (LVEF) < 30%	High risk for post-bypass cardiotomy shock; requires active standby of mechanical circulatory support (MCS).

4.2 Medical and Preoperative Stabilization

- Neurohormonal Blockade Optimization: Optimize doses of beta-blockers, ACE inhibitors/ARNIs, and mineralocorticoid receptor antagonists (MRAs) up to 24 hours before surgery.
- Anticoagulation Reversal: Discontinue antiplatelet agents (e.g., Aspirin, Clopidogrel) 5–7 days preoperatively. Bridge patients on systemic anticoagulation using an intravenous unfractionated heparin infusion, to be stopped 4 hours before the skin incision.
- Inotropic Optimization: For patients in decompensated NYHA Class IV heart failure, initiate milrinone 0.375-0.75 mug/kg/min or levosimendan to improve forward flow and lower pulmonary vascular resistance without increasing myocardial oxygen demand.

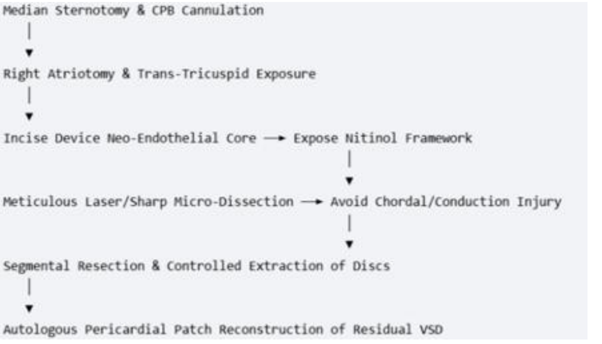
5. Surgical Directives: Indications, Dissection, and Explantation Techniques

5.1 Indications for Late Device Explantation

Surgical explantation is indicated when there is clear evidence of device-related electrical and mechanical decompensation:

1. New-onset or progressive LBBB accompanied by a decline in LVEF Delta LVEF > 10% or absolute LVEF < 45).
2. Persistent high-grade or complete AV block that does not resolve with corticosteroid therapy.
3. Severe, progressive aortic or tricuspid valve regurgitation caused by mechanical distortion from the device discs.
4. Device-associated refractory infective endocarditis or chronic mechanical hemolysis.

5.2 Method of Dissection and Explantation Protocols



Exposure and Cannulation

Perform a standard median sternotomy. Establish normothermic or mild hypothermic 34C cardiopulmonary bypass (CPB) via ascending aortic cannulation and bicaval snaring. Deliver cold blood cardioplegia directly into the aortic root to ensure complete electromechanical arrest and protect the hypertrophied myocardium.

Surgical Approach

Access the VSD device through a right atriotomy, exposing the defect across the tricuspid valve. If the VSD is positioned further down the muscular septum, a targeted right ventriculotomy oriented parallel to the sub-tricuspid architecture can be used instead to improve visualization.

Micro-Dissection of the Device Matrix

- Neo-Endothelial Incision: Device endothelialisation wraps the nitinol framework in a dense fibrous sheath. Carefully incise this neo-endocardium along the margins of the right ventricular disc using a No. 11 scalpel blade or fine micro-scissors.
- Disc Dissection: Use fine bipolar cutting diathermy or a low-power surgical laser to free the device mesh from the surrounding septal tissue. Do not pull or apply excessive traction on the device, as this can tear the conduction bundle or create large defects in the muscular septum.
- Separation from the Subvalvular Apparatus: Carefully dissect away any tricuspid valve chordae or septal leaflet tissue that has adhered to the device framework to preserve valve function.
- Segmental Extraction: If the device is firmly embedded, cut the central connecting pin using heavy wire-cutters. This allows the left and right ventricular discs to be dissected and extracted separately, minimizing translational traction forces on the conduction system.

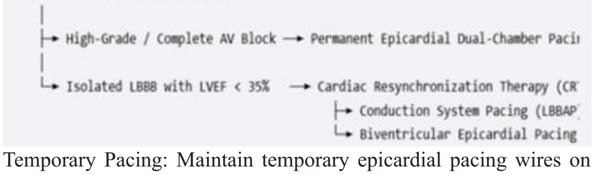
Defect Debridement and Patch Reconstruction

Once the device is removed, debride any calcified or necrotic tissue along the edges of the defect. Avoid aggressive scraping of the posterior-inferior margin where the conduction bundle runs. Reconstruct the residual ventricular septal defect using an autologous fresh pericardial patch fixed with interrupted or running polypropylene sutures. Ensure these sutures are placed well back from the vulnerable conduction margins.

6. Refractory Conduction Disease and Advanced Postoperative Support

6.1 Management of Persistent Post-Explantation LBBB

If LBBB or AV block persists after the device is removed and the tissue heals, downstream management focuses on preventing further ventricular remodelling:



Temporary Pacing: Maintain temporary epicardial pacing wires on both the right ventricle and the left ventricular free wall during the early post-operative period.

- Pharmacological Support: Continue guideline-directed medical therapy (GDMT), using low-dose neurohormonal inhibitors as tolerated by the patient's blood pressure.
- Permanent Pacing Indications: If high-grade atrioventricular block or symptomatic LBBB persists beyond 7–14 days' post-op, place a permanent pacing system.

6.2 Mechanical Circulatory Support (MCS) Back-up Strategies

Patients with severe preoperative LV dysfunction LVEF < 25-30% are at risk for post-cardiotomy cardiogenic shock, requiring planned mechanical support options.

Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO)

- Indications: Indicated for patients who cannot be successfully weaned from cardiopulmonary bypass despite high-dose inotropic support, or those who develop severe biventricular failure early after surgery.
- Cannulation Configuration: Central cannulation is typically preferred in pediatric patients. Insert a cannula into the ascending aorta for arterial delivery, and place single or dual venous cannulas in the right atrium or venae cavae. For older pediatric and adolescent patients, peripheral cannulation via the femoral artery and femoral vein can be used.
- Deployment Timing: Initiate support intraoperatively before secondary end-organ hypoperfusion or refractory metabolic acidosis develops.

Ventricular Assist Devices (VAD)

- Indications: Indicated for patients with isolated, severe left ventricular failure who have adequate right ventricular function, serving as a bridge to myocardial recovery or cardiac transplantation.
- Device Selection: For smaller pediatric patients, continuous or pulsatile paracorporeal pumps (e.g., Berlin Heart EXCOR) provide reliable support across a range of body surface areas. For larger adolescents BSA > 1.2-1.5m2, implantable continuous-flow pumps (e.g., HeartMate 3) can be positioned within the left ventricular apex.
- Deployment Management: Ensure the interventricular patch is completely sealed, as any residual VSD can cause a significant right-to-left or left-to-right shunt when the VAD is activated, leading to severe hypoxia or systemic volume overload.

7. Role of Cardiac Resynchronization Therapy (CRT)

7.1 Immediate Postoperative Period (0–30 Days)

In the early post-operative phase, temporary biventricular pacing can help manage acute dyssynchrony-induced low cardiac output syndrome:

- Pacing Configurations: Place temporary epicardial pacing leads on the right atrium, the right ventricular apex, and the lateral/posterior wall of the left ventricle.
- Hemodynamic Tuning: Optimize the interventricular delay VV = 10- 20 ms, activating the left ventricle first) to coordinate contraction, reduce septal flash, and maximize stroke volume, which can help facilitate weaning from inotropic support.

7.2 Late Postoperative Period (>30 Days)

If significant LBBB and ventricular dyssynchrony persist over the longer term, permanent resynchronization is key to preventing heart failure progression:

- Left Bundle Branch Area Pacing (LBBAP): An advanced pacing approach that uses a trans-septal lead deployed from the right ventricular side of the septum, screwing deeply into the tissue to directly engage the left bundle branch system distal to the site of device injury. This generates rapid, physiological left ventricular activation, minimizing the QRS duration without needing a coronary sinus lead.
- Pediatric Epicardial Resynchronization: In smaller children where transvenous access is restricted, place permanent epicardial leads directly onto the right atrium, the right ventricle, and the latest-activating region of the left ventricular free wall (typically identified via preoperative speckle-tracking imaging).

8. Future Innovations and Biophysical Frameworks

8.1 Advanced Physics of Conduction System Injury

Future diagnostic tools will use finite element modelling (FEM) to analyze the mechanical interaction between VSD devices and septal tissues. By incorporating patient-specific anatomical variations and nitinol radial stress calculations into these computational models, clinicians can predict localized tissue strain before a procedure.

$$\sigma_{\text{radial}} = \frac{E_{\text{nitinol}} \cdot \cdot \Delta D}{(1 - \nu^2) \cdot D_0}$$

Where:

- E_{nitinol} is the elastic modulus of Nitinol
- ΔD is the degree of device deformation or oversizing
- ν is Poisson's ratio
- D_0 is the unconstrained device diameter

8.2 Next-Generation Intelligent Biomaterials

To prevent chronic mechanical compression and subsequent tissue fibrosis, next-generation intracardiac devices are incorporating innovative material designs:

- Bioresorbable Polymeric Matrices: These devices provide temporary mechanical support to allow tissue healing and endothelization across the VSD before gradually breaking down into non-toxic metabolites, eliminating long-term foreign-body compression risks.
- Low-Profile Nitinol Geometries: Newer device designs feature thinner wire diameters and highly flexible, asymmetric retention discs that conform more naturally to the subaortic and subendocardial anatomy, reducing direct pressure on the conduction bundle.

8.3 Molecular Electrophysiological Therapies

Emerging genetic and cellular therapies aim to repair damaged conduction tissue without relying entirely on mechanical hardware:

- Targeted Gene Therapy: Delivering specific ion channel genes such as HCN4 or Nav1.5 via viral vectors to damaged septal areas can help restore normal pacemaking and conduction velocities within the left bundle branch system.
- 3D Bioprinted Conduction Patches: Bio-engineered patches embedded with specialized cardiac conduction cells can be surgically implanted along the septum during device explantation, helping to reconstruct damaged conduction pathways.

DISCUSSION

Nitinol VSD devices induce left bundle branch block through subendocardial compression, leading to ischemia and chronic scarring, which triggers dyssynchrony-driven ventricular remodelling and decreased ejection fraction. Management involves advanced imaging with speckle-tracking, 3D echo, and cardiac MRI for pre-operative planning, followed by surgical explantation via right atriotomy or ventriculotomy. Post-explantation care includes utilizing mechanical support and electrical pacing to manage severe dysfunction or persistent LBBB.

Late conduction abnormalities after perimembranous VSD device closure represent a complex interaction between device biomechanics, septal anatomy, electrophysiology, and ventricular mechanics. The proximity of the occluder to the His bundle and left bundle branch explains the vulnerability of the conduction system. Mechanical compression, chronic inflammatory fibrosis, and persistent nitinol radial forces may contribute to progressive conduction injury rather than an isolated acute electrical event [1-5].

The development of LBBB produces abnormal ventricular activation with delayed left ventricular free-wall contraction, septal flash, impaired myocardial work efficiency, and progressive adverse remodelling. Studies of LBBB-induced cardiomyopathy demonstrate that electrical dyssynchrony can become a primary driver of heart failure physiology and may improve after restoration of synchrony using CRT or conduction system pacing [6-8].

Advanced imaging has changed evaluation of these patients. Speckle-tracking echocardiography, global longitudinal strain assessment, mechanical dispersion analysis, three-dimensional echocardiography, and cardiac magnetic resonance tissue characterization allow identification of early ventricular deterioration before severe reduction in ejection fraction occurs [9-12].

Surgical explantation remains technically demanding because endothelialisation fixes the device to septal tissue and the conduction axis lies close to the inferior-posterior margins of the defect. Contemporary approaches emphasize controlled micro-dissection, avoidance of traction injury, preservation of valvular structures, and patch reconstruction with conduction tissue protection [1,13].

Future management will increasingly involve precision medicine. Artificial intelligence-based imaging algorithms may detect early dyssynchrony patterns. Digital twins created from CT/MRI datasets may simulate device stress distribution and predict conduction injury risk before implantation. Next-generation occluders incorporating bioresorbable polymers, flexible low-pressure frames, and tissue-compatible materials may reduce late complications [14-16].

Emerging electrophysiological technologies including left bundle branch area pacing, His bundle pacing, leadless pacing platforms, and hybrid CRT strategies provide new opportunities for physiological ventricular activation. Regenerative approaches including engineered conduction tissue scaffolds, stem-cell-derived cardiomyocyte integration, and bioelectronic interfaces represent future possibilities for repairing conduction injury rather than simply compensating for it [17-19].

Future clinical pathways should include international registries, standardized criteria for explantation, AI-assisted surveillance, computational modelling before device selection, and integration of surgical, interventional, electrophysiological, and heart failure expertise.

Summary

Detailed Mechanism Summaries

1. Mechanical Breakdown of Conduction Tissue

The continuous radial force and cyclic shape-memory expansion of nitinol VSD devices cause localized subendocardial compression. This reduces capillary perfusion to the left bundle branch system, leading to focal ischemia, inflammation, and eventual collagen deposition and scarring. These structural updates permanently disrupt normal electrical propagation through the left bundle branch.

2. Dyssynchrony-Driven Ventricular Remodelling

LBBB prompts early, paradoxical contraction of the interventricular septum (septal flash), which reduces regional work efficiency. The late-activating left ventricular free wall experiences increased mechanical stress and workload, triggering asymmetric wall stress, eccentric ventricular remodelling, chamber dilation, and a progressive decline in ejection fraction.

3. Advanced Diagnostic Protocols

Detailed preoperative imaging is essential to accurately evaluate ventricular function and plan the surgical approach. Speckle-tracking echocardiography measures global longitudinal strain, while 3D echocardiography tracks the systolic dyssynchrony index. Cardiac MRI with late gadolinium enhancement identifies myocardial scarring, and electrophysiology studies map specific conduction delays to help optimize pacing strategies.

4. Intraoperative Explantation and Defect Repair

The embedded device is accessed via a right atriotomy or right ventriculotomy. Surgical micro-scissors or low-power lasers are used to carefully dissect the neo-endothelial tissue and free the device discs while preserving nearby tricuspid valve chordae. Once the device is extracted, the residual ventricular septal defect is closed using an autologous fresh pericardial patch.

5. Advanced Support and Conduction Recovery

For patients with severe preoperative ventricular dysfunction, veno-arterial ECMO or ventricular assist devices should be ready for immediate intraoperative deployment if needed. If electrical conduction delays or LBBB persist after the device is removed, epicardial biventricular pacing or left bundle branch area pacing can be established to restore mechanical synchrony and support long-term ventricular recovery.

Expanded Future Technology Roadmap

1. Artificial Intelligence and Machine Learning:

AI algorithms for automated ECG interpretation, strain analysis, prediction of LBBB progression, and personalized risk modelling.

2. Cardiac Digital Twin Technology:

Patient-specific computational models integrating CT, MRI, electrophysiology and finite-element biomechanics to predict device-related tissue stress.

3. Next-generation VSD Closure Devices:

- Bioabsorbable occluders
- Polymer-based scaffolds
- Low radial force nitinol designs
- Shape-adaptive asymmetric discs
- Drug-eluting anti-inflammatory device coatings

4. Advanced Electrophysiology:

- Left bundle branch area pacing
- His bundle pacing
- Leadless physiological pacing
- Wireless cardiac stimulation systems
- Adaptive closed-loop pacing algorithms

5. Regenerative Medicine:

- 3D bioprinted conduction patches
- Engineered Purkinje-like cellular networks
- Biomaterial-guided myocardial regeneration
- Gene-based modulation of conduction proteins including Nav1.5 and HCN channels

6. Surgical Innovation:

- Robotic and minimally invasive explantation
- Image-guided device removal
- Augmented reality surgical planning
- Intraoperative electrophysiological mapping

7. Mechanical Circulatory Support:

Future systems will include smaller pediatric VAD platforms, wearable artificial hearts, smarter ECMO systems, and AI-guided haemodynamic control.

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