

Ventricular Assist Devices

Mechanical support, hemodynamics and clinicopathologic complications

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

- Explain why mechanical circulatory support is used in selected patients with severe cardiac failure.
- Trace the blood-flow circuit and predict the physiologic effects of LV unloading.
- Recognize the mechanisms of device-related thrombosis, bleeding, infection and organ injury.
- Integrate gross/device findings with clinical presentation and device function.

Clinical problem: Inadequate forward cardiac output

Myocardial injury or dysfunction

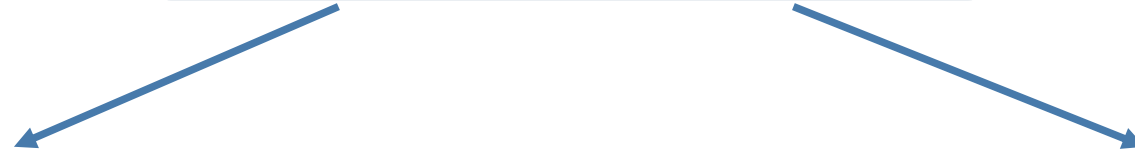
Ventricular pumping fails

Reduced effective forward flow

Organ perfusion becomes inadequate

Elevated filling pressure

Pulmonary or systemic congestion



Robbins: Causes that may require VAD support

Potentially reversible dysfunction

- Transient postoperative myocardial stunning
- Acute severe dysfunction following myocardial infarction

Progressive disease

- Advanced dilated or other cardiomyopathy
- End-stage ventricular failure despite treatment

Why medical therapy may not be enough

- Advanced heart failure can persist despite guideline-directed medications and device-based therapies.
- Continuous inotropic dependence or recurrent low-output admissions may indicate exhausted reserve.
- An assist pump provides forward flow without replacing the whole heart.
- The treatment is reserved for carefully selected patients—not routine heart failure care.

Low output and congestion are distinct targets

Low forward output

- Fatigue, hypoperfusion and end-organ dysfunction
- VAD flow can augment systemic delivery

High filling pressure

- Pulmonary venous congestion and dyspnea
- Left ventricular unloading may reduce left-sided pressure

The conceptual task of a VAD

Blood enters the pump

Inflow from a ventricular chamber



External mechanical Work

Rotor creates forward pressure / flow



Blood exits to artery

Flow returns to systemic or pulmonary circuit

VADs supplement rather than replace cardiac anatomy

- The native ventricle, valves and vessels remain present in conventional VAD support.
- The implanted circuit diverts some blood through a mechanical pump.
- Native ejection may continue, depending on residual ventricular function and settings.
- Device performance depends on interactions with both ventricles and vascular resistance.

What transplantation shortage changes

**Advanced refractory
Heart failure**

Need for sustained circulation



**Donor heart
Unavailable now**

Transplant access is limited



Durable VAD support

Survival and conditioning during
wait

Bridge to transplantation

- Durable support sustains circulation while an eligible patient awaits a donor heart.
- Improved mobility and end-organ function may make transplantation more feasible.
- VAD-related complications can still alter candidacy during the waiting period.
- The intent is to transition from support to transplant when an organ becomes available.

Bridge to myocardial recovery

- Temporary support can provide time for reversible dysfunction to improve.
- Recovery is plausible in selected postoperative or acute-injury settings.
- Durable support can occasionally enable reassessment for sufficient native recovery.
- Explantation requires documented physiologic reserve—not symptoms alone.

Long-term support without planned transplant

- Destination therapy is an established pathway for selected transplant-ineligible patients.
- Increasing use outside the transplant-candidate population.
- Long-term benefit must be weighed against bleeding, stroke, infection and device dependence.
- Transplant ineligibility requires individualized assessment, not a single historical label.

Bridge to decision: When prognosis is uncertain

Shock or rapidly Changing condition

Immediate hemodynamic
instability



Temporary Stabilization

Acute mechanical support when
appropriate



Repeated evaluation

Recovery, durable VAD,
transplant or other care

Distinct indications, shared mechanical principle

Strategy	Main therapeutic goal	Reassessment point
Bridge to transplant	Maintain support until a donor is available	Transplant eligibility
Bridge to recovery	Permit native function to recover	Demonstrable ventricular reserve
Destination therapy	Long-term circulation without planned transplant	Ongoing benefits and complications
Bridge to decision	Stabilize while next step is clarified	Reversibility and candidacy

Temporary and durable support are not interchangeable

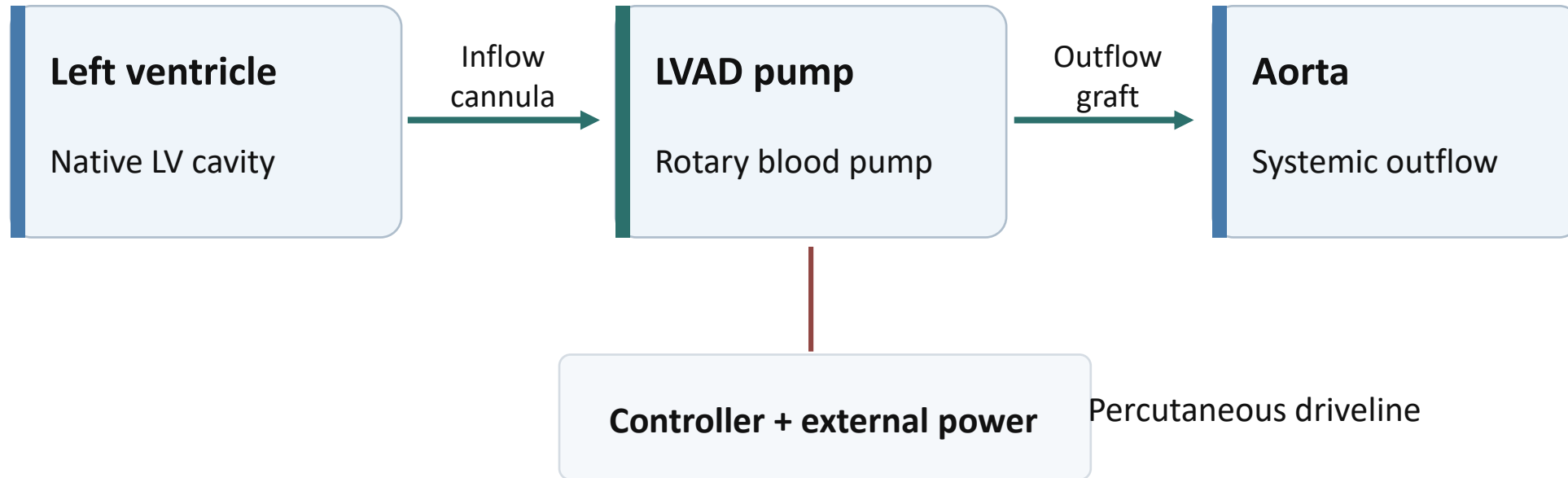
Temporary mechanical support

- Designed for acute instability or short stabilization
- Duration and invasiveness vary by platform
- May bridge to recovery or durable therapy

Durable VAD

- Intended for sustained support in selected patients
- Requires long-term monitoring and external support systems
- Device-related complications accrue over time

A left-sided VAD supports systemic output



Schematic: Conventional LV-to-aorta bypass. Not a surgical diagram.

Right- and biventricular support

RVAD

- Drains systemic venous / right-heart blood
- Returns flow into pulmonary artery
- Selected when RV output is inadequate

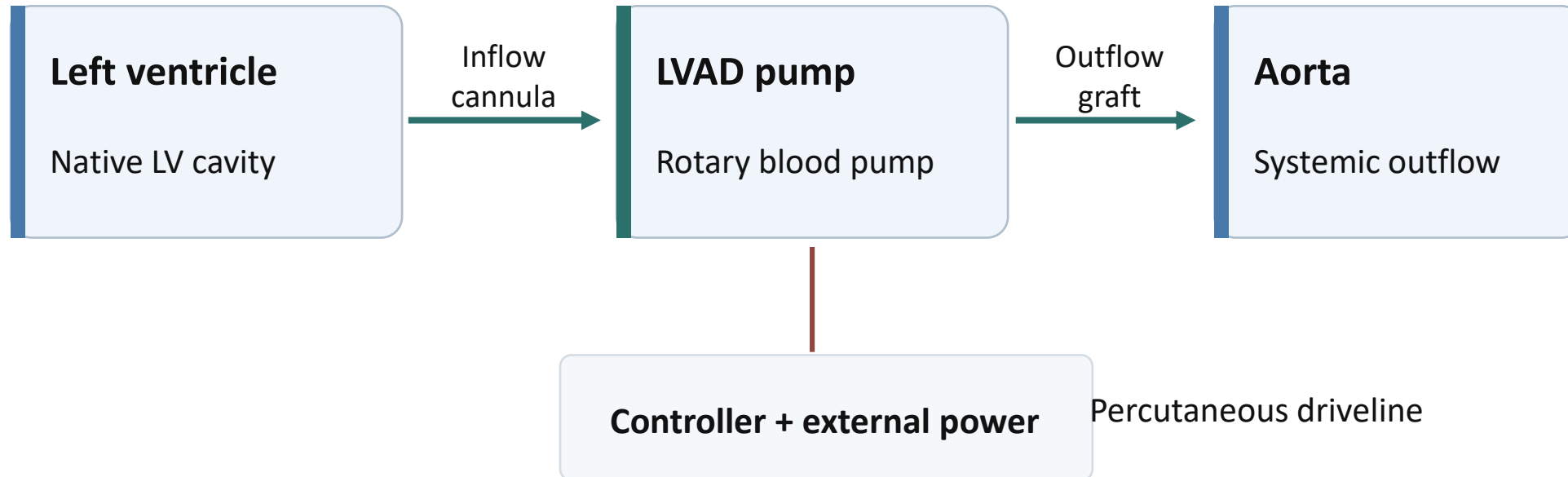
Biventricular support

- Supports both circulations when both ventricles fail
- Requires matched systemic and pulmonary flows
- Adds device and management complexity

The LVAD circuit: Four crucial components

Component	Function	Failure mode to recognize
Inflow cannula	Directs LV blood into pump	Obstruction or malposition
Pump	Adds energy and drives forward flow	Thrombosis or mechanical malfunction
Outflow graft	Delivers blood toward aorta	Kinking or obstruction
Driveline / controller / power	Supplies and regulates pump operation	Infection, cable or power problems

Flow path is parallel to native LV ejection



Native ventricular ejection may also pass through the aortic valve (not shown in this simplified circuit).

Rotary continuous-flow pump physiology

Blood contacts Engineered surfaces

Rotary element generates
pressure



Continuous pump output

Less pulsatile arterial waveform



Blood–material Interaction

Thrombotic and bleeding
tradeoffs

Continuous flow does not mean zero cardiac pulsatility

- Native ventricular contraction can contribute a varying pulse waveform.
- Pulse amplitude depends on residual native ejection and degree of pump support.
- Absent or weak peripheral pulses do not by themselves indicate circulatory arrest.
- Clinical assessment uses the patient, device parameters and appropriate pressure measurement.

LV unloading reduces myocardial wall stress

**Pump withdraws LV
Blood**

Lower ventricular volume



**LV filling pressure
May fall**

Less pulmonary venous
congestion



**Wall stress may
Decrease**

Altered cardiac loading
conditions

Preload is a limiting condition for pump output

Insufficient LV Filling

Hypovolemia or reduced RV
output



Low inflow Availability

Unstable LVAD flow



Excessive unloading

Risk of suction event

Afterload also matters to LVAD performance

Higher systemic Vascular resistance

Larger pressure gradient to aorta



Rotary pump flow may Decline

At unchanged pump setting



Hemodynamic Optimization

Requires individualized
assessment

Interventricular coupling is central

Potential benefit

- Lower LV filling pressure can reduce pulmonary congestion
- Systemic perfusion may improve

Potential hazard

- Greater systemic flow increases RV venous return
- Leftward septal displacement can worsen RV mechanics

Why right-ventricular failure can appear after LVAD

LVAD augments systemic Flow

More venous return reaches RV



RV must deliver Pulmonary flow

Limited contractility or elevated PVR



RV decompensation

Low LV filling and systemic congestion

RV dysfunction versus pump malfunction

Finding	Consider RV failure	Consider inflow / device issue
Systemic venous congestion	Often present	Not necessarily
LV filling	May be inadequate	May be inadequate
Device alarms	Can occur indirectly	May suggest direct malfunction
Best interpretation	Echo + hemodynamics	Device log + imaging + labs

Aortic valve opening may diminish

Increased LVAD Unloading

Lower LV systolic pressure



Aortic valve may open Less often

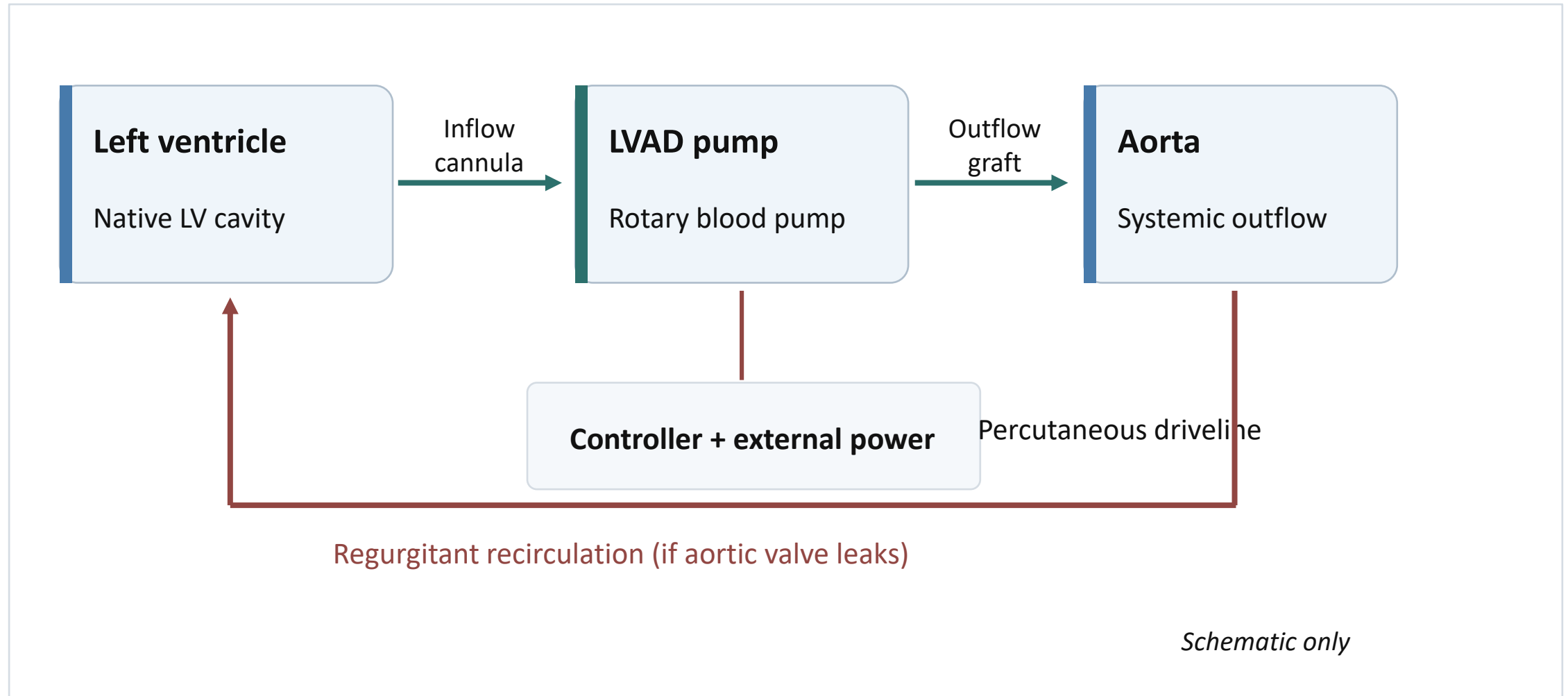
Dependent on native
contractility



Valve and root Dynamics change

Potential contribution to late
complications

Aortic regurgitation can create recirculation



Who deserves evaluation for durable VAD?

- Selected patients with advanced refractory heart failure despite appropriate treatment.
- Patients dependent on intravenous inotropes or short-term mechanical support may warrant review.
- Functional limitation, repeated admissions and end-organ injury add important context.
- Indication requires multidisciplinary assessment rather than one laboratory cutoff.

Anatomic and clinical feasibility must align

Cardiac and vascular factors

- Size and geometry of ventricular cavity
- Right ventricular performance and valve lesions
- Pulmonary vascular resistance

Patient and systems factors

- Neurologic, renal, hepatic and infectious comorbidity
- Ability to manage external power and driveline
- Patient goals, caregiver resources and follow-up

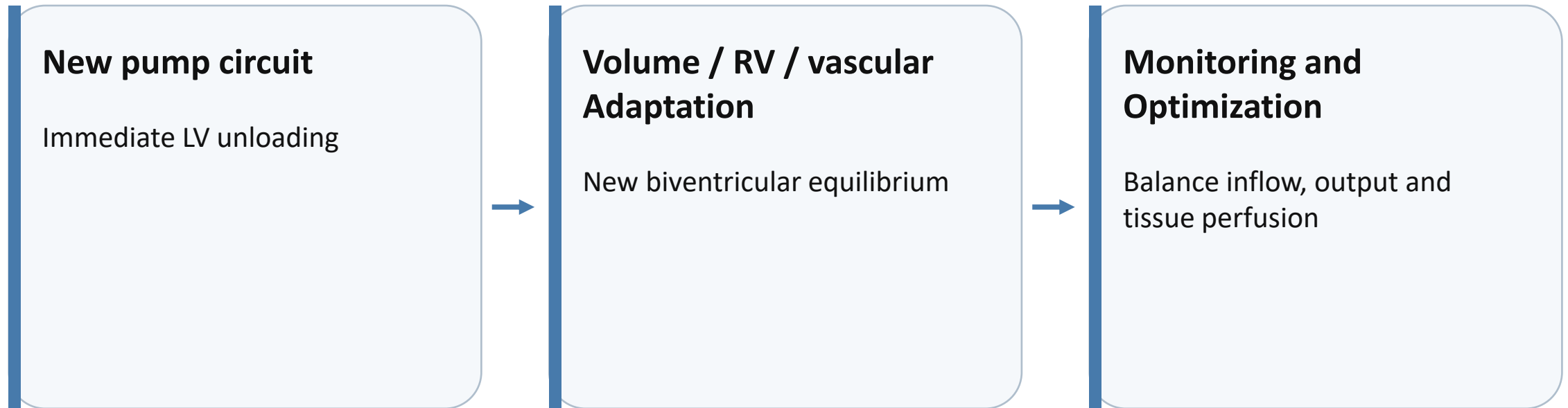
Contraindications are context-dependent

- Irreversible severe end-organ disease may limit meaningful benefit.
- Active uncontrolled infection and inability to tolerate requisite therapy raise major concerns.
- Uncorrectable anatomy or profound right-heart dysfunction may change device strategy.
- Eligibility decisions depend on device, timing, goals and expert review.

What to assess before implantation

Domain	Main question	Typical assessment
Ventricular function	Can the RV support pulmonary flow?	Echocardiography / hemodynamics
Peripheral organs	Is injury reversible?	Renal, hepatic and perfusion assessment
Hemostasis / infection	Can device therapy be sustained?	Bleeding and infectious evaluation
Patient factors	Is ongoing device care feasible?	Shared decision-making and support evaluation

Early postoperative physiology can change rapidly



Living with a durable implanted system

Implanted components

- Pump, inflow and outflow connections
- Percutaneous driveline in many systems

Daily external dependence

- Controller and reliable power supply
- Aseptic exit-site care and alarm response
- Regular specialist surveillance

The central hemocompatibility paradox

Thrombosis risk

- Foreign surface activates coagulation and platelets
- Flow stasis or disturbed flow favors thrombus
- Embolism and pump dysfunction may result

Bleeding risk

- Antithrombotic treatment reduces thrombotic risk
- Rotary shear changes von Willebrand function
- Fragile mucosal vessels may bleed

Why artificial blood-contacting surfaces thrombose

Protein adsorption

Plasma proteins coat foreign surface



Platelet adhesion / activation

Cellular thrombotic response



Thrombus formation

Local occlusion or embolization



Coagulation cascade

Thrombin and fibrin generation

Mechanical shear can damage erythrocytes

High shear / abnormal Flow

Mechanical red-cell stress



Intravascular Hemolysis

Plasma free Hb and LDH rise



Possible clinical Signals

Dark urine, anemia or organ injury

Acquired von Willebrand syndrome

**High shear in
Continuous-flow
Circuit**

Von Willebrand factor unfolds /
is altered



**Loss of large
Functional multimers**

Impaired primary hemostasis



**Increased mucosal
Bleeding tendency**

Especially with other bleeding
risks

Why gastrointestinal bleeding is multifactorial

Circulating factors

- Required antithrombotic therapy in many devices
- Acquired von Willebrand abnormalities
- Other patient-specific coagulation risks

Vascular factors

- Gastrointestinal angiodysplasia or vascular lesions
- Changes in pulsatility and local perfusion may contribute
- Mechanisms of lesion development remain incompletely settled

Hemorrhage versus thrombus: Diagnostic tension

Clinical problem	Priority question	Important tradeoff
GI hemorrhage	Where is the bleeding source?	Interrupting therapy may increase thrombosis
Suspected pump thrombus	Is flow impaired and blood being hemolyzed?	Therapy may increase bleeding
Ischemic stroke	What mechanism and brain territory?	Bleeding risk affects management
Intracranial hemorrhage	What is the extent of bleeding?	Device still requires safe circulation

Pump thrombosis: A mechanically amplified problem

Disturbed flow / Prothrombotic state

Thrombus develops in pump circuit



Flow obstruction or High shear

Worsened mechanical efficiency



Hemolysis and low-flow Signals

Systemic hypoperfusion or embolization

Stroke can be ischemic or hemorrhagic

Ischemic mechanisms

- Thromboembolism from pump, heart or vessel
- Underlying vascular disease also contributes
- May cause sudden focal neurologic deficits

Hemorrhagic mechanisms

- Antithrombotic exposure
- Hemostatic abnormalities and other vascular risks
- Requires urgent neurologic evaluation

Suction events reflect excessive local unloading

**Low LV preload or high
Relative support**

LV cavity becomes small



**Inflow cannula
Intermittently
Contacts wall**

Reduced inflow / abnormal flow



**Hemodynamic
Instability**

Assess patient, RV and device
together

Mechanical obstruction: Locate the problem

Site	Illustrative problem	Effect
Inflow	Cannula malposition or thrombus	Poor ventricular drainage
Pump	Internal thrombosis or malfunction	Unreliable forward energy transfer
Outflow	Graft kink, twist or thrombus	Resistance to aortic delivery
Power / control	Disconnected or failing components	Interrupted or abnormal pump function

Aortic insufficiency can reduce effective output

Pump delivers blood to Aorta

Aortic pressure maintained



Regurgitant leak into LV

Part of blood returns to chamber



Blood recirculates Through pump

Effective systemic flow may fall

Right-heart failure remains a major limitation

- A left-sided pump cannot compensate for completely inadequate RV output.
- Right atrial and systemic venous pressures may rise despite apparently functioning LVAD.
- Hepatic and renal congestion can persist or worsen.
- RV mechanics, pulmonary circulation and pump flow must be assessed together.

The driveline crosses an infection-prone barrier

External-to-internal cable

Percutaneous skin crossing



Barrier disruption

Potential microbial entry



Systemic complication

Bloodstream or deep device infection



Local exit-site infection

May track deeper along hardware

Different infectious syndromes must be separated

Syndrome	Anatomic emphasis	Clinical implication
Exit-site infection	Skin / superficial tract	Local inflammation, possible drainage
Deep driveline infection	Internal tract or contiguous tissues	Higher burden of source control
Pump / pocket infection	Implanted device surroundings	Biofilm and difficult eradication
Bloodstream infection	Intravascular dissemination	Potential systemic organ injury

Foreign bodies alter infection biology

Implanted surface + Microbial access

Device colonization can occur



Biofilm-associated Persistence

Organisms protected within
matrix



Hard-to-eradicate Focus

Treatment may be prolonged or
complex

Mechanical and power failures are time-critical

Potential causes

- Cable or connector failure
- Controller or power problems
- Mechanical failure or obstruction

Clinical consequences

- Declining forward flow
- Hemodynamic compromise
- Urgent device-team evaluation

Evaluate the patient—not a number alone

- Integrate perfusion, mentation, congestion, bleeding and infection signs.
- Review pump speed, estimated flow, power use and alarm history.
- Interpret echocardiographic anatomy and ventricular interactions.
- Compare serial trends; Isolated device readouts may mislead.

Differential of unexpectedly low VAD flow

Physiologic category	Examples	Initial interpretive clue
Low preload	Volume depletion, RV failure	Small or underfilled LV
High afterload	Elevated systemic resistance	Reduced pump flow at given speed
Flow pathway obstruction	Inflow or outflow problem	Anatomic or device abnormality
Electrical / device malfunction	Power or controller issue	Alarm and device-log context

A practical workup for suspected pump thrombosis

Clinical / device concern

Low flow, power change or hemolysis



Laboratory and device evaluation

LDH, anemia / hemolysis, controller logs



Cardiac and vascular imaging

Echo and targeted conduit assessment



Expert integrated diagnosis

Differentiate thrombus from other causes

Echocardiography links anatomy to device function

Heart assessment

- LV size and septal position
- RV size and systolic performance
- Aortic valve opening and regurgitation

Mechanical-support questions

- Does LV unload appropriately?
- Could inflow be impeded?
- Does physiology explain low flow?

Pathologic examination of an explanted VAD

Maintain orientation And chain of Documentation

Identify inflow, pump, outflow
and driveline



Inspect for occlusion And surface deposits

Photograph and localize
abnormalities



Correlate with Microbiology and Histology

Interpret with clinical and device
data

Thrombus pathology: Location matters

What may be present

- Adherent thrombus on blood-contacting components
- Inflow or outflow luminal material
- Native ventricular thrombus

What must be established

- Precise site and degree of obstruction
- Relationship to local flow geometry
- Potential embolic or hemolytic consequences

Tissue response under sustained unloading

- Ventricular loading conditions change substantially during LVAD support.
- Myocardial size and geometry may remodel; Improvement does not ensure recovery.
- Underlying cardiomyopathic injury may remain despite better perfusion.
- Morphology alone cannot establish that device explantation is safe.

Assessment of genuine myocardial recovery

Clinical stability on support

Symptoms and organ function improve



Serial structured testing

Imaging and hemodynamic reassessment



Explant only if recovery is Sufficient

Durable native circulation expected



Trial of reduced support when Suitable

Native reserve assessed by expert team

The key device–pathology correlation

Observed problem	Potential mechanism	Evidence to seek
Dark urine / rising LDH	Shear-related hemolysis	Pump / circuit and laboratory correlation
Melena / anemia	Angiodysplasia + hemostatic defects	GI source and therapy review
Fever / driveline drainage	Device-associated infection	Exit tract, cultures, imaging
Systemic venous congestion	RV failure	Right-heart echo and hemodynamics

Case: Stunned myocardium after surgery

Illustrative teaching case

A postoperative patient has profound but potentially reversible ventricular dysfunction and cannot maintain adequate perfusion without advanced support.

Discussion questions

- What makes this a recovery-oriented scenario?
- Why might temporary support precede a durable device?
- Which organ-function trends would matter?

Case: Cardiomyopathy awaiting a donor

Illustrative teaching case

A transplant-eligible patient with refractory cardiomyopathy is deteriorating while waiting for a donor heart.

Discussion questions

- What is the main intent of durable support?
- How might end-organ function and physical condition change?
- Which device complications must be monitored?

Case: End-stage failure without transplant plan

Illustrative teaching case

A patient with refractory LV failure is judged unsuitable for transplantation, but durable support might align with the patient's goals.

Discussion questions

- What is the intended therapeutic pathway?
- Why is transplant ineligibility not equivalent to VAD ineligibility?
- What must informed consent explicitly include?

Case: Gastrointestinal bleeding on LVAD

Illustrative teaching case

A recipient of a continuous-flow LVAD develops recurrent melena and anemia despite no obvious device malfunction.

Discussion questions

- Which nonmechanical hemostatic change can contribute?
- What vascular lesion type is often implicated?
- Why is the treatment balance difficult?

Case: Hemolysis and altered device power

Illustrative teaching case

A long-term LVAD recipient develops dark urine, biochemical hemolysis and an unexpected change in pump parameters.

Discussion questions

- What potentially serious device complication must be considered?
- What alternative explanations should be assessed?
- Why are clinical, laboratory and device records all needed?

Clinical synthesis: Benefits and burdens

Physiologic benefit	Reason it matters	Associated vulnerability
Improved systemic flow	Tissue perfusion / functional status	Dependence on electrical pump
LV unloading	Less filling pressure / congestion	RV–LV interaction and suction
Long-term support	Transplant bridge or destination	Cumulative infectious / blood risks
Blood pumping across device surfaces	Sustained circulatory support	Thrombus, hemolysis and bleeding

Five clinicopathologic principles

- VADs restore forward flow; They do not erase underlying myocardial disease.
- The intended trajectory—recovery, transplant or long-term support—determines the care pathway.
- LVAD function depends on preload, afterload and adequate RV output.
- Blood–device interaction creates competing risks of clotting and hemorrhage.
- Infection and mechanical complications require anatomical as well as clinical evaluation.

Selected primary and specialist references

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Elsevier; 2026.
- ISHLT Guidelines for Mechanical Circulatory Support: A 10-Year Update. 2023.
- ISHLT/HFSA Guideline on Acute Mechanical Circulatory Support. 2023.
- ISHLT Hemocompatibility-Related Adverse Events Consensus. 2024.
- ISHLT Infection Definitions for Durable and Acute MCS Devices. 2024.
- AHA/ACC/HFSA Guideline for the Management of Heart Failure. 2022.