

Quick Reference: Hemodynamic Equations

Pressures mmHg · flows L/min · Hb g/dL · saturations as decimals inside formulas · contents mL O₂/dL · "I" = indexed to BSA · dyn = dyn·s·cm⁻⁵

Flow		
CO	HR × SV / 1000	4-8 L/min
CI	CO / BSA	2.5-4.0 L/min/m ²
SV	CO × 1000 / HR	60-100 mL
SVI	SV / BSA	33-47 mL/m ²
BSA	√(ht cm × wt kg / 3600)	m ²
MAP	(SBP + 2·DBP) / 3	70-105 mmHg
mPAP	(PASP + 2·PADP) / 3	≤20 mmHg
Fick		
Fick CO	VO ₂ / [(CaO ₂ - CvO ₂) × 10]	L/min
Fick CO (Hb)	VO ₂ / [13.4 × Hb × (SaO ₂ - SvO ₂)]	sats as decimals
VO ₂ Dehmer	125 × BSA	mL/min
VO ₂ weight	3 × wt (kg)	mL/min
VO ₂ I LaFarge ♂	138.1 - 11.49·ln(age) + 0.378·HR	mL/min/m ²
VO ₂ I LaFarge ♀	138.1 - 17.04·ln(age) + 0.378·HR	mL/min/m ²
Resistance		
SVR	80 × (MAP - CVP) / CO	800-1200 dyn
SVRI	80 × (MAP - CVP) / CI	1970-2390
PVR	80 × (mPAP - PAWP) / CO	<160-250 dyn
PVRI	80 × (mPAP - PAWP) / CI	255-285
Wood units	Drop the × 80	PVR <2 WU
TPG	mPAP - PAWP	≤12 mmHg
DPG	PADP - PAWP	<7 mmHg
Work & power		
CPO	MAP × CO / 451	~1 W; <0.6 bad
CPI	CPO / BSA	0.5-0.7 W/m ²
LVSWI	SVI × (MAP - PAWP) × 0.0136	50-62 g·m/m ²
RVSWI	SVI × (mPAP - CVP) × 0.0136	5-10 g·m/m ²
Right ventricle		
PAPi	(PASP - PADP) / RAP	>0.9 (AMI); >1.85 (LVAD)
RA:PCWP	RAP / PAWP	<0.63
RVSP	4 × (TR V _{max}) ² + RAP	<35-40 mmHg
TAPSE	M-mode annular excursion	≥17 mm
CO ₂ markers		
CO ₂ gap	PvCO ₂ - PaCO ₂	≤6 mmHg
Gap ratio	CO ₂ gap / (CaO ₂ - CvO ₂)	<1.4
Bypass & perfusion		
EBV	wt × 70 (♂) or 65 (♀) mL/kg	mL
Hct on CPB	EBV × Hct / (EBV + prime + CP + fluid)	≥21-24%
ABL	EBV × (Hct _i - Hct _t) / Hct _{avg}	mL
Pump flow idx	Pump flow / BSA	2.2-2.4 L/min/m ²
DO ₂ I on CPB	Flow index × CaO ₂ × 10	≥272-280
Heparin	300-400 U/kg	ACT >480 s
Protamine	1-1.3 mg per 100 U heparin	mg
Transplant sizing		
LV mass	a × ht ^{0.54} × wt ^{0.61} (a 8.25♂/6.82♀)	g
RV mass	a × age ^{-0.32} × ht ^{1.135} × wt ^{0.315} (a 11.25♂/10.59♀)	g
PHM mismatch	(donor - recip PHM) / recip × 100	worse than -30% risky
pTLC	♂ 7.99·ht - 7.08; ♀ 6.60·ht - 5.79	D/R ~0.8-1.2

Oxygen transport		
CaO ₂	1.34 × Hb × SaO ₂ + 0.003 × PaO ₂	17-20 mL/dL
CvO ₂	1.34 × Hb × SvO ₂ + 0.003 × PvO ₂	12-15 mL/dL
DO ₂	CO × CaO ₂ × 10	900-1100 mL/min
DO ₂ I	CI × CaO ₂ × 10	500-600
VO ₂	CO × (CaO ₂ - CvO ₂) × 10	200-250 mL/min
VO ₂ I	CI × (CaO ₂ - CvO ₂) × 10	110-160
O ₂ ER	VO ₂ /DO ₂ ≈ (SaO ₂ - SvO ₂)/SaO ₂	0.22-0.30
SvO ₂	SaO ₂ - VO ₂ / (CO × Hb × 13.4)	65-75%
ScvO ₂	Measured (SVC)	>70%
Shunt & gas exchange		
Qp:Qs	(SaO ₂ - SmvO ₂) / (SpvO ₂ - SpaO ₂)	1.0; >1.5 significant
Flamm MV	(3 × SVC sat + IVC sat) / 4	%
PAO ₂	FiO ₂ × (P _{atm} - 47) - PaCO ₂ / 0.8	mmHg
A-a	PAO ₂ - PaO ₂	≈ age/4 + 4 (RA)
P/F	PaO ₂ / FiO ₂	>300
CcO ₂	1.34 × Hb + 0.003 × PAO ₂	mL/dL
Qs/Qt	(CcO ₂ - CaO ₂) / (CcO ₂ - CvO ₂)	<5-10%
Valves & coronary		
Bernoulli	ΔP = 4v ²	mmHg
Gorlin	CO(mL/min) / (HR × SEP × 44.3 × √MG)	>1.0 cm ²
Hakki	CO(L/min) / √peak-to-peak	>1.0 cm ²
Continuity	π(LVOTd/2) ² × VTI _{LVOT} / VTI _{AV}	AVAi >0.6
CPP	DBP - LVEDP (or PAWP)	>50-60 mmHg
Fluids & drugs		
PPV	(PP _{max} - PP _{min}) / PP _{mean} × 100	>12-13% responsive
SVV	(SV _{max} - SV _{min}) / SV _{mean} × 100	>10-12% responsive
PLR	Δ SV/CO/VTI with leg raise	≥10% responsive
VIS	dopa + dobu + 100(epi + NE) + 10(mil) + 10,000(vaso U/kg/min)	>20-30 high risk
Forrester	CI 2.2 × PAWP 18 grid	I-IV
Thoracic & ventilator		
ppoFEV ₁	FEV ₁ % × (1 - segments / 19)	>60% low risk
ppoDLCO	DLCO% × (1 - segments / 19)	>60% low risk
VO ₂ max	CPET	>20 safe; <10 prohibitive
Driving P	Pplat - PEEP	<15 cmH ₂ O
C _{stat}	V _T / (Pplat - PEEP)	50-80 mL/cmH ₂ O
V _D /V _T	(PaCO ₂ - PēCO ₂) / PaCO ₂	0.2-0.3
OI	FiO ₂ × P _{aw} × 100 / PaO ₂	>20-25 severe
ECMO		
Recirculation	(S _{pre} - SvO ₂) / (S _{post} - SvO ₂)	<20-30%
O ₂ transfer	Q × (C _{post} O ₂ - C _{pre} O ₂) × 10	mL/min
Flow	Q × 1000 / wt	50-80 mL/kg/min
Post-op valve echo		
EROA	2πr ² × V _{alias} / V _{max}	≥0.40 severe MR
RVol	EROA × VTI _{MR}	≥60 mL severe
MVA (PHT)	220 / PHT	≤1.5 severe MS
E/e'	E / avg e'	>14 ↑ LAP

CVICU / CTICU Hemodynamic Calculations

A working reference: every formula, every variable defined, normal values, and how to think about the number at the bedside

This guide is organized by the physiologic question each calculation answers: How much blood is moving? What is it pumping against? How hard is the ventricle working? Is the RV keeping up? Is oxygen delivery meeting demand? It ends with the graphical representations (waveforms, PV loops, Starling curves) and the classification schemes that tie the numbers together. A companion spreadsheet performs every calculation here.

Units convention. Pressures in mmHg, flows in L/min, hemoglobin in g/dL, saturations as decimals inside formulas (0.98, not 98) unless stated, contents in mL O₂/dL of blood. Indexed values (the trailing "I") divide by body surface area (BSA, m²).

Core variables used throughout

Abbreviation	Meaning	Units / how obtained
HR	Heart rate	beats/min
SBP / DBP	Systolic / diastolic arterial pressure	mmHg; arterial line
MAP	Mean arterial pressure	mmHg; arterial line (measured) or calculated
CVP / RAP	Central venous / right atrial pressure (used interchangeably)	mmHg; CVC or PA catheter proximal port, end-expiration
PASP / PADP	Pulmonary artery systolic / diastolic pressure	mmHg; PA catheter distal port
mPAP	Mean pulmonary artery pressure	mmHg
PAWP (PCWP)	Pulmonary artery wedge (capillary wedge) pressure; estimates LA pressure and LVEDP	mmHg; balloon-occluded, end-expiration
LVEDP	Left ventricular end-diastolic pressure	mmHg; direct (cath) or approximated by PAWP
CO / CI	Cardiac output / cardiac index	L/min; L/min/m ²
SV / SVI	Stroke volume / stroke volume index	mL/beat; mL/beat/m ²
BSA	Body surface area	m ² ; Mosteller: $\sqrt{(\text{height cm} \times \text{weight kg} / 3600)}$
Hb (Hgb)	Hemoglobin concentration	g/dL
SaO ₂ / PaO ₂	Arterial O ₂ saturation / partial pressure	decimal / mmHg; ABG
SvO ₂ / PvO ₂	Mixed venous O ₂ saturation / partial pressure (true mixed venous = PA blood)	decimal / mmHg; PA catheter distal port VBG
ScvO ₂	Central venous O ₂ saturation (SVC blood)	decimal; CVC tip
PaCO ₂ / PvCO ₂	Arterial / mixed venous CO ₂ partial pressure	mmHg

1. Flow: how much blood is moving?

Cardiac output (CO)

$$\text{CO} = \text{HR} \times \text{SV} / 1000$$

Where:

CO Cardiac output, L/min
HR Heart rate, beats/min
SV Stroke volume, mL/beat (+1000 converts mL to L)

Normal / target: 4–8 L/min

Bedside pearl: CO is the product of two things the body trades against each other. Tachycardia can hold CO up while SV falls, until diastolic filling time runs out. A 'normal' CO at HR 130 is a compensating heart, not a healthy one.

Cardiac index (CI)

$$\text{CI} = \text{CO} / \text{BSA}$$

Where:

CI Cardiac index, L/min/m²
BSA Body surface area, m² (Mosteller formula above)

Normal / target: 2.5–4.0 L/min/m²; <2.2 is the classic cardiogenic shock threshold (Forrester); <1.8 with support = severe

Bedside pearl: Indexing lets you compare a 50 kg patient to a 130 kg patient. Beware indexing in morbid obesity: BSA overstates metabolically active mass, so CI can look falsely low.

Stroke volume (SV) and stroke volume index (SVI)

$$SV = (CO \times 1000) / HR$$

$$SVI = SV / BSA = (CI \times 1000) / HR$$

Where:

SV Volume ejected per beat, mL

SVI SV / BSA, mL/beat/m²

Normal / target: SV 60–100 mL; SVI 33–47 mL/m²

Mean arterial pressure (MAP)

$$MAP \approx DBP + \frac{1}{3} (SBP - DBP) = (SBP + 2 \cdot DBP) / 3$$

Where:

SBP – DBP Pulse pressure (PP)

$\frac{1}{3}$ weighting Diastole occupies roughly two-thirds of the cycle at normal HR; the approximation degrades with tachycardia

Normal / target: 70–105 mmHg; usual target ≥ 65 mmHg (higher in chronic HTN, CKD, or with a fixed coronary lesion)

Bedside pearl: Always prefer the monitor's integrated (area-under-the-curve) MAP. It is the most robust number on an arterial line, least affected by over- or under-damping.

Mean pulmonary artery pressure (mPAP)

$$mPAP \approx (PASP + 2 \cdot PADP) / 3$$

Where:

PASP / PADP PA systolic / diastolic pressure, mmHg

Normal / target: mPAP ≤ 20 mmHg (pulmonary hypertension defined as mPAP > 20 mmHg at rest, 2022 ESC/ERS)

Thermodilution CO (Stewart-Hamilton principle)

$$CO = [V_i \times (T_b - T_i) \times K] / \int \Delta T_b(t) dt$$

Where:

V_i Injectate volume (typically 10 mL)

$T_b - T_i$ Blood minus injectate temperature

K Computation constant (catheter- and injectate-specific; entered into the monitor)

$\int \Delta T_b dt$ Area under the thermodilution curve; a smaller area means faster washout, i.e. higher CO

Normal / target: Average three injections within 10% of each other, at the same phase of respiration

Bedside pearl: Thermodilution is **unreliable** with significant TR (indicator recirculates, curve prolonged, CO underestimated), intracardiac shunts, very low output states, and rapid IV fluid infusion during measurement. Continuous CO (CCO) catheters average over several minutes, so they lag acute changes.

2. The Fick principle (including the hemoglobin-based form)

Adolf Fick's principle: the uptake of a substance by an organ equals blood flow times the arteriovenous concentration difference of that substance. Applied to oxygen across the lungs, the substance is O₂, the organ is the whole body, and the flow is cardiac output.

Fick cardiac output (content form)

$$CO = VO_2 / [(CaO_2 - CvO_2) \times 10]$$

Where:

VO₂ Whole-body oxygen consumption, mL O₂/min (measured or assumed, see below)

CaO₂ – CvO₂ Arteriovenous O₂ content difference, mL O₂/dL

$\times 10$ Converts dL to L so the answer comes out in L/min

Fick cardiac output (hemoglobin form)

$$CO = VO_2 / [13.4 \times Hb \times (SaO_2 - SvO_2)]$$

Where:

13.4 1.34 mL O₂/g Hb (Hüfner's constant) × 10 (dL→L conversion), folded into one number

Hb Hemoglobin, g/dL

SaO₂, SvO₂ Arterial and mixed venous saturations as **decimals** (0.98, 0.65)

VO₂ mL O₂/min

Normal / target: Same answer as the content form, minus the dissolved O₂ term

Bedside pearl: This is simply the content form with dissolved O₂ (0.003 × PO₂) dropped. That term is trivial on room air or modest FiO₂, but at very high PaO₂ (post-bypass on FiO₂ 1.0, or hyperbaric conditions) dropping it slightly overestimates CO. It's the version used in the cath lab and the one you can do in your head: with Hb 10, SaO₂ 0.98, SvO₂ 0.60, assumed VO₂ 250 → 250 / (13.4 × 10 × 0.38) ≈ 4.9 L/min.

Estimating VO₂ when you can't measure it ("assumed Fick")

$$\text{Dehmer: } VO_2 \approx 125 \text{ mL/min/m}^2 \times BSA$$

$$\text{Weight-based: } VO_2 \approx 3 \text{ mL/kg/min} \times \text{weight}$$

$$\text{LaFarge (male): } VO_{2I} = 138.1 - 11.49 \cdot \ln(\text{age}) + 0.378 \cdot HR$$

$$\text{LaFarge (female): } VO_{2I} = 138.1 - 17.04 \cdot \ln(\text{age}) + 0.378 \cdot HR$$

Where:

Dehmer Fixed index; simplest, most commonly used

Weight-based ~3 mL/kg/min (rough adult resting value)

LaFarge Adds age, sex, and HR (derived in children and young adults; less validated in older adults)

ln Natural logarithm

Bedside pearl: The assumed VO₂ is the weak link. Critically ill patients can be far from 125 mL/min/m²: fever, shivering, sepsis, and agitation raise it; hypothermia, sedation, and paralysis lower it. Studies show assumed VO₂ errs by >25% in a large fraction of patients. A trend in assumed-Fick CO on the same patient is more trustworthy than any single absolute value. Measured VO₂ (metabolic cart or indirect calorimetry on the ventilator) fixes this.

3. Resistance: what is the heart pumping against?

All vascular resistance formulas are Ohm's law ($R = \Delta P / Q$) applied to a vascular bed: the pressure drop across the bed divided by flow through it. The factor of 80 converts mmHg·min/L (Wood units) to dyn·s·cm⁻⁵.

Systemic vascular resistance (SVR)

$$SVR = 80 \times (MAP - CVP) / CO$$

$$SVRI = 80 \times (MAP - CVP) / CI \text{ (normal 1970-2390 dyn·s·cm}^{-5}\text{·m}^2\text{)}$$

Where:

MAP – CVP Pressure drop across the systemic circulation (inflow minus outflow), mmHg

CO Flow, L/min

80 Unit conversion: 1 mmHg = 1333 dyn/cm², 1 L/min = 16.67 cm³/s → 1333 / 16.67 ≈ 80

Normal / target: 800-1200 dyn·s·cm⁻⁵ (10-15 Wood units)

Bedside pearl: SVR is **calculated, not measured**, so it inherits every error in MAP, CVP, and CO. High SVR + low CI = cold (cardiogenic or hypovolemic). Low SVR + high CI = vasodilatory (sepsis, post-bypass vasoplegia, SIRS). A low SVR with a low CI suggests mixed shock or a failing heart that can't mount a hyperdynamic response.

Pulmonary vascular resistance (PVR)

$$PVR = 80 \times (mPAP - PAWP) / CO$$

$$PVRI = 80 \times (mPAP - PAWP) / CI \text{ (normal 255-285 dyn·s·cm}^{-5}\text{·m}^2\text{)}$$

Where:

mPAP – PAWP Pressure drop across the pulmonary bed (PA in, left atrium out); this is the TPG

Wood units Drop the ×80: PVR (WU) = (mPAP – PAWP) / CO

Normal / target: <250 dyn·s·cm⁻⁵; <2 WU (2022 ESC/ERS threshold for pre-capillary PH is PVR >2 WU)

Bedside pearl: Transplant and LVAD teams think in Wood units. A PVR >3 WU, and especially one that doesn't fall with vasodilator challenge (nitroprusside, milrinone, inhaled NO), raises concern for acute donor RV failure after heart transplant.

Transpulmonary gradient (TPG) and diastolic pulmonary gradient (DPG)

$$\text{TPG} = \text{mPAP} - \text{PAWP} \quad \text{DPG} = \text{PADP} - \text{PAWP}$$

Where:

TPG Total pressure drop across the lungs, mmHg

DPG Diastolic PA pressure minus wedge, mmHg; less flow-dependent than TPG

Normal / target: TPG ≤ 12 mmHg; DPG < 7 mmHg

Bedside pearl: In left-heart failure the PA pressure rises passively with the wedge (isolated post-capillary PH: TPG and DPG normal). When TPG > 12 or DPG ≥ 7 , the pulmonary vessels themselves have remodeled (combined pre- and post-capillary PH), which won't fully reverse when you unload the LV.

4. Ventricular work and cardiac power

Cardiac power output (CPO)

$$\text{CPO} = (\text{MAP} \times \text{CO}) / 451$$

$$\text{CPI} = \text{CPO} / \text{BSA} \text{ (normal } \sim 0.5\text{--}0.7 \text{ W/m}^2\text{)}$$

Where:

MAP $\times$ CO Pressure $\times$ flow = hydraulic power

451 Converts mmHg·L/min to watts ($133.3 \text{ Pa/mmHg} \div 60,000$ to convert L/min to m^3/s)

Normal / target: ~ 1.0 W at rest (0.8–1.2); **< 0.6 W** is the strongest single hemodynamic predictor of in-hospital mortality in cardiogenic shock (SHOCK trial registry analysis, Fincke 2004)

Bedside pearl: CPO integrates pressure and flow into one number. A MAP of 70 on high-dose norepinephrine with a CO of 3.0 gives CPO 0.47 W: the MAP looks fine, but the heart is in trouble. Many shock teams use CPO < 0.6 W as a trigger to consider escalating mechanical circulatory support.

Left ventricular stroke work index (LVSWI)

$$\text{LVSWI} = \text{SVI} \times (\text{MAP} - \text{PAWP}) \times 0.0136$$

Where:

SVI mL/beat/ m^2

MAP – PAWP Pressure the LV generates above its filling pressure

0.0136 Converts mmHg·mL to gram-meters

Normal / target: 50–62 g·m/ m^2 /beat

Bedside pearl: LVSWI is a crude contractility surrogate because it accounts for both the volume moved and the pressure it's moved against. Low LVSWI with high PAWP places the patient on a depressed, flattened Starling curve.

Right ventricular stroke work index (RVSWI)

$$\text{RVSWI} = \text{SVI} \times (\text{mPAP} - \text{CVP}) \times 0.0136$$

Where:

mPAP – CVP Pressure the RV generates above its filling pressure

Normal / target: 5–10 g·m/ m^2 /beat

Bedside pearl: In LVAD candidacy literature RVSWI is often reported *without* the 0.0136 factor (units mmHg·mL/ m^2); values below $\sim 300\text{--}450$ mmHg·mL/ m^2 suggest higher risk of post-implant RV failure. Check which unit a paper uses.

5. Right ventricular indices

The RV is the usual weak link in the CTICU: after LVAD implantation, heart transplant, inferior/RV MI, mitral surgery with pulmonary hypertension, and long bypass runs. These indices try to catch RV failure early.

Pulmonary artery pulsatility index (PAPi)

$$\text{PAPi} = (\text{PASP} - \text{PADP}) / \text{RAP}$$

Where:

PASP – PADP PA pulse pressure: a surrogate for how much stroke volume the RV is generating

RAP Right atrial pressure (CVP)

Normal / target: Higher is better. Concerning: **<0.9** in acute inferior MI (predicts RV failure / mortality); **<1.85** before LVAD implant (predicts post-LVAD RV failure)

Bedside pearl: PAPI works because a failing RV produces a low PA pulse pressure while backing up into a high RAP. It's cheap, available from any PA catheter, and trends well.

RA:PCWP ratio

$$\text{RA:PCWP} = \text{RAP} / \text{PAWP}$$

Where:

RAP / PAWP Right-sided filling pressure relative to left-sided

Normal / target: Normal ~0.5. **>0.63** suggests the RV is failing out of proportion to the LV (post-LVAD RV failure risk); **>0.86** in acute MI suggests RV involvement

Bedside pearl: If CVP is climbing toward or above the wedge, the right heart is the problem, and more volume will usually make it worse.

RV systolic pressure (echo estimate)

$$\text{RVSP} = 4 \times (\text{TR } V_{\text{max}})^2 + \text{RAP}$$

Where:

TR V_{max} Peak tricuspid regurgitant jet velocity, m/s (continuous-wave Doppler)

$4v^2$ Simplified Bernoulli: pressure gradient between RV and RA in systole

RAP Measured CVP or estimated from IVC size/collapsibility

Normal / target: <35–40 mmHg; equals PASP in the absence of pulmonic stenosis or RVOT obstruction

Tricuspid annular plane systolic excursion (TAPSE)

$$\text{TAPSE} = \text{longitudinal displacement of the lateral tricuspid annulus in systole (M-mode)}$$

Normal / target: ≥ 17 mm normal; < 17 mm suggests RV systolic dysfunction

Bedside pearl: TAPSE is angle- and load-dependent and drops after any pericardiotomy, even with preserved RV function, so it underestimates RV performance after cardiac surgery. RV fractional area change (normal $\geq 35\%$) and RV S' (≥ 9.5 cm/s) are complementary.

6. Oxygen transport: supply versus demand

The oxygen chain runs: how much O_2 is in the blood (content) $\rightarrow$ how much is delivered per minute (DO_2) $\rightarrow$ how much the tissues take out (VO_2) $\rightarrow$ what fraction that represents (O_2ER) $\rightarrow$ what's left in the venous blood coming back (SvO_2).

Arterial oxygen content (CaO_2)

$$CaO_2 = (1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)$$

Where:

1.34 Hüfner's constant: mL O_2 bound per gram of fully saturated Hb (values from 1.34 to 1.39 appear in references)

Hb Hemoglobin, g/dL

SaO_2 Arterial saturation as a decimal

0.003 Solubility of O_2 in plasma: mL O_2 /dL/mmHg

PaO_2 Arterial O_2 partial pressure, mmHg

Normal / target: ~17–20 mL O_2 /dL

Bedside pearl: Nearly all oxygen is carried on hemoglobin. Raising PaO_2 from 100 to 400 adds only 0.9 mL/dL; raising Hb from 7 to 8 adds ~1.3 mL/dL. Hb and SaO_2 matter; PaO_2 above ~80 barely does.

Mixed venous oxygen content (CvO_2)

$$CvO_2 = (1.34 \times Hb \times SvO_2) + (0.003 \times PvO_2)$$

Where:

SvO_2 Mixed venous saturation (PA blood) as a decimal

PvO_2 Mixed venous O_2 partial pressure, mmHg

Normal / target: ~12-15 mL O₂/dL

Oxygen delivery (DO₂)

$$DO_2 = CO \times CaO_2 \times 10$$

$$DO_2I = CI \times CaO_2 \times 10$$

Where:

× 10 Converts mL/dL to mL/L

Normal / target: ~900-1100 mL O₂/min; DO₂I 500-600 mL/min/m²

Bedside pearl: Your three levers: CO, Hb, and SaO₂. CO usually has the most room to move. Post-bypass, a DO₂I below ~270-300 mL/min/m² during the pump run is associated with acute kidney injury (goal-directed perfusion literature).

Oxygen consumption (VO₂, reverse Fick)

$$VO_2 = CO \times (CaO_2 - CvO_2) \times 10$$

$$VO_2I = CI \times (CaO_2 - CvO_2) \times 10$$

Where:

CaO₂ - CvO₂ a-v O₂ content difference; normal ~4-5 mL/dL

Normal / target: ~200-250 mL O₂/min; VO₂I 110-160 mL/min/m²

Bedside pearl: Calculated VO₂ shares the CO measurement with DO₂ ("mathematical coupling"), so an error in CO moves both in the same direction. That's one reason supply-dependency is hard to prove from PA catheter data alone.

Oxygen extraction ratio (O₂ER)

$$O_2ER = VO_2 / DO_2 \approx (SaO_2 - SvO_2) / SaO_2$$

Where:

Simplified form Valid when dissolved O₂ is negligible; CO and Hb cancel out

Normal / target: 0.22-0.30 (22-30%). Critical extraction ~0.50-0.60

Bedside pearl: This is the core supply-demand concept. As DO₂ falls, tissues compensate by extracting more, so VO₂ stays flat (supply-independent). Once O₂ER hits its ceiling at the critical DO₂, VO₂ starts falling with DO₂ (supply-dependent), anaerobic metabolism begins, and lactate rises.

Mixed venous (SvO₂) and central venous (ScvO₂) saturation

$$SvO_2 \approx SaO_2 - [VO_2 / (CO \times Hb \times 13.4)]$$

Where:

Rearranged Fick Shows SvO₂ falls when VO₂ rises, or when CO, Hb, or SaO₂ fall

SvO₂ Drawn from PA distal port: mixes SVC, IVC, and coronary sinus blood

ScvO₂ Drawn from SVC (CVC tip): reflects upper body only

Normal / target: SvO₂ 65-75%; ScvO₂ >70%

Bedside pearl: In health ScvO₂ runs 2-3% below SvO₂ (kidneys extract little, so IVC blood is well saturated). In shock the relationship inverts: splanchnic and renal beds extract heavily, IVC saturation drops, and ScvO₂ can overestimate SvO₂ by 5-8%. Also: a **high** SvO₂ (>80%) is not always good news. It can mean failed extraction (sepsis, cyanide or nitroprusside toxicity, mitochondrial dysfunction), a left-to-right shunt, or a wedged catheter sampling arterialized blood.

7. CO₂-derived perfusion markers

Venous-to-arterial CO₂ gap (Pv-aCO₂, "CO₂ gap")

$$\Delta PCO_2 = PvCO_2 - PaCO_2$$

Where:

PvCO₂ Mixed venous (or central venous) PCO₂, mmHg

PaCO₂ Arterial PCO₂, mmHg

Normal / target: ≤6 mmHg. >6 mmHg = flow is inadequate to wash out tissue CO₂

Bedside pearl: CO_2 is far more diffusible than O_2 , so the gap tracks flow even when extraction is impaired. Its key use: a patient with a *normal* ScvO_2 but a gap >6 is still under-perfused (often seen in sepsis and post-bypass vasoplegia). Think of it as the flow detector, and ScvO_2 as the extraction detector.

CO₂ gap to a-v O₂ content ratio

$$\text{Ratio} = (\text{PvCO}_2 - \text{PaCO}_2) / (\text{CaO}_2 - \text{CvO}_2)$$

Where:

Numerator CO_2 gap, mmHg

Denominator a-v O₂ content difference, mL/dL

Normal / target: >1.4 (some use >1.0 - 1.6) suggests anaerobic metabolism (CO_2 produced without matching O₂ consumed)

Bedside pearl: Approximates the respiratory quotient. It identifies patients whose lactate is from true tissue hypoxia, who are more likely to increase VO_2 when you increase DO_2 .

Lactate

Lactate (measured, mmol/L)

Normal / target: <2 mmol/L; clearance ≥ 10 - 20% over 2-6 h is a resuscitation target

Bedside pearl: Post-bypass hyperlactatemia isn't always hypoperfusion: epinephrine (β_2 -driven aerobic glycolysis), hyperglycemia, and liver dysfunction all raise it. Pair lactate with SvO_2 and the CO_2 gap before chasing it with more inotrope.

8. Shunt calculations

Pulmonary-to-systemic flow ratio (Qp:Qs)

$$\text{Qp:Qs} = (\text{SaO}_2 - \text{SmvO}_2) / (\text{SpvO}_2 - \text{SpaO}_2)$$

Where:

Qp / Qs Pulmonary / systemic blood flow

SaO₂ Systemic arterial saturation

SmvO₂ Mixed venous saturation *upstream* of the shunt (usually SVC, or Flamm: $(3 \times \text{SVC} + \text{IVC})/4$)

SpvO₂ Pulmonary venous saturation (often assumed 95-98% or = SaO_2 if no R→L shunt)

SpaO₂ Pulmonary artery saturation

Normal / target: 1.0 (no shunt). >1.5 = hemodynamically significant left-to-right shunt; <1.0 = net right-to-left

Bedside pearl: In the CTICU this is the post-MI VSD calculation, and it matters in adult congenital disease. A saturation "step-up" of $\geq 7\%$ from RA to PA on serial sampling is the clue. Each saturation is used as a fraction (Hb cancels out).

Intrapulmonary shunt fraction (Qs/Qt)

$$\text{Qs/Qt} = (\text{CcO}_2 - \text{CaO}_2) / (\text{CcO}_2 - \text{CvO}_2)$$

Where:

CcO₂ End-capillary O₂ content, assuming $\text{SaO}_2 = 1.0$ and $\text{PO}_2 = \text{PAO}_2$: $(1.34 \times \text{Hb} \times 1.0) + (0.003 \times \text{PAO}_2)$

PAO₂ Alveolar O₂ (alveolar gas equation): $\text{FiO}_2 \times (\text{P}_{\text{atm}} - 47) - \text{PaCO}_2 / 0.8$

47 Water vapor pressure at 37 °C, mmHg

0.8 Respiratory quotient

Normal / target: <5 - 10% . >20 - 30% = significant shunt (atelectasis, ARDS, pulmonary edema)

Bedside pearl: True shunt doesn't respond to FiO_2 ; V/Q mismatch does. If PaO_2 barely moves on FiO_2 1.0, think shunt, and recruit the lung (PEEP, recruitment) or look for an intracardiac R→L shunt (PFO with high RA pressure).

9. Valves and coronary perfusion

Simplified Bernoulli equation

$$\Delta P = 4v^2$$

Where:

ΔP Pressure gradient across an orifice, mmHg

v Peak velocity through it, m/s (CW Doppler)

Bedside pearl: Foundation for every echo gradient: AS, MS, TR-derived RVSP, LVOT obstruction (SAM after mitral repair).

Gorlin formula (aortic valve area)

$$AVA = CO / (HR \times SEP \times 44.3 \times \sqrt{\text{mean gradient}})$$

Where:

AVA Aortic valve area, cm²
CO mL/min here (not L/min)
SEP Systolic ejection period, s/beat
44.3 Gorlin constant: $\sqrt{(2 \times 980 \text{ cm/s}^2)} \times \text{empiric coefficient (37.7 for the mitral valve, with diastolic filling period replacing SEP)}$
mean gradient Mean transvalvular gradient, mmHg
Normal / target: Normal 3–4 cm²; severe AS <1.0 cm² (AVAi <0.6 cm²/m²)

Hakki shortcut

$$AVA \approx CO \text{ (L/min)} / \sqrt{(\text{peak-to-peak gradient})}$$

Where:

Peak-to-peak Peak LV pressure minus peak aortic pressure (cath)

Bedside pearl: Fast, and within ~20% of Gorlin at normal HR. Less accurate with bradycardia or tachycardia.

Continuity equation (echo AVA)

$$AVA = (LVOT \text{ area} \times VTI_{LVOT}) / VTI_{AV}$$

Where:

LVOT area $\pi \times (LVOT \text{ diameter} / 2)^2$, cm²
VTI Velocity-time integral, cm (pulsed-wave in LVOT, continuous-wave through the valve)

Bedside pearl: Conservation of mass: what goes through the LVOT must go through the valve. LVOT diameter is squared, so a 1 mm measurement error produces a large area error.

Coronary perfusion pressure (CPP)

$$CPP = DBP - LVEDP \text{ (or PAWP as LVEDP surrogate)}$$

Where:

DBP Aortic diastolic pressure: the LV is perfused mostly in diastole
LVEDP Back-pressure opposing subendocardial flow

Normal / target: Generally aim >50–60 mmHg (in cardiac arrest, CPR CPP >15–20 mmHg is associated with ROSC)

Bedside pearl: This is why diastolic pressure matters so much in a hypertrophied LV, a freshly grafted heart, or AS: tachycardia shortens diastole and a high LVEDP squeezes the subendocardium at the same time. It's also the rationale for IABP diastolic augmentation.

10. Fluid responsiveness

Pulse pressure variation (PPV)

$$PPV (\%) = 100 \times (PP_{\max} - PP_{\min}) / [(PP_{\max} + PP_{\min}) / 2]$$

$$SVV (\%) = 100 \times (SV_{\max} - SV_{\min}) / SV_{\text{mean}} \text{ (>10-12\% predicts response)}$$

Where:

PP_{max} / PP_{min} Largest and smallest pulse pressure over one respiratory cycle

Normal / target: >12–13% predicts a rise in SV with fluid; 9–13% is the 'gray zone'

Bedside pearl: Valid only with: controlled mechanical ventilation, $V_T \geq 8$ mL/kg, no spontaneous breaths, sinus rhythm, and a closed chest. With an open sternum, low compliance, or RV failure, PPV is unreliable. RV failure can produce high PPV from afterload variation even in a volume-overloaded patient.

Passive leg raise (PLR)

$$\Delta SV \text{ or } \Delta CO \text{ (or LVOT VTI) during } 45^\circ \text{ leg raise from semirecumbent}$$

Normal / target: Increase $\geq 10\%$ in SV/CO/VTI = fluid responsive

Bedside pearl: Works in spontaneously breathing patients and arrhythmias. Must use a real-time flow measure, not BP. CVP as a single value does not predict fluid responsiveness; its trend and its relationship to PAWP are more useful.

11. Vasoactive burden and shock classification

Vasoactive-Inotropic Score (VIS)

VIS = dopamine + dobutamine + 100·epi + 100·norepi + 10·milrinone + 10,000·vasopressin

Where:

Catecholamines Doses in mcg/kg/min
, milrinone

Vasopressin Dose in U/kg/min (convert from U/min by dividing by weight)

Normal / target: No universal normal; higher VIS after cardiac surgery correlates with morbidity and mortality. In adult cardiac surgery, VIS >20–30 in the first 24 h is commonly cited as high risk

Bedside pearl: Originally a pediatric score (Gaies 2010) that's now widely used in adults. It's a common language for 'how much support is this patient on' and a frequent MCS escalation trigger.

Forrester hemodynamic subsets (originally post-MI; still the bedside warm/cold, wet/dry grid):

	PAWP ≤18 (dry)	PAWP >18 (wet)
CI >2.2 (warm)	I: Normal perfusion, no congestion	II: Pulmonary congestion → diuretics, vasodilators
CI ≤2.2 (cold)	III: Hypoperfusion without congestion → volume	IV: Cardiogenic shock → inotropes, MCS

SCAI SHOCK stages (2019, updated 2022):

Stage	Description	Typical hemodynamics
A: At risk	No shock signs; at-risk substrate (large MI, acute HF)	Normal BP, normal perfusion
B: Beginning	Relative hypotension or tachycardia without hypoperfusion	SBP <90 or MAP <60, lactate normal
C: Classic	Hypoperfusion requiring one intervention (pressor, inotrope, MCS)	CI <2.2, lactate ≥2, PAWP >15
D: Deteriorating	Failing to respond to initial interventions; escalation	Worsening lactate, rising support despite therapy
E: Extremis	Cardiac arrest with CPR or ECMO, or refractory collapse	Multiple pressors plus MCS, or ongoing CPR

12. Graphical representations

Frank-Starling curve. Plots SV (or CO, or stroke work) against preload (LVEDV or its pressure proxy, PAWP). On the steep ascending limb, fluid raises SV; on the flat plateau, it only raises filling pressure and causes edema. Inotropes shift the whole curve up and left; a failing ventricle sits on a flatter, lower curve. Fluid responsiveness tests (PPV, PLR) are all asking 'which limb is this patient on?'

Pressure-volume (PV) loop. One cardiac cycle plotted as LV pressure against volume. Four phases: filling (bottom), isovolumic contraction (right side), ejection (top), isovolumic relaxation (left side). Key lines:

Element	What it means
ESPVR (slope = E_{es})	End-systolic pressure-volume relationship; slope is end-systolic elastance, a load-independent contractility index
EDPVR	End-diastolic pressure-volume relationship; a steeper curve means a stiffer ventricle (diastolic dysfunction, hypertrophy, tamponade physiology)
E_a (arterial elastance)	≈ end-systolic pressure / SV; represents afterload as a line on the same graph
E_a/E_{es} (coupling)	Ventricular-arterial coupling; ~1 is optimal. RV-PA uncoupling (E_a/E_{es} rising) is an early sign of RV failure
Loop area	Stroke work
Loop width	Stroke volume (EDV – ESV)

PV loops explain MCS: an Impella pulls blood from the LV throughout the cycle, turning the loop into a triangle and shifting it left (unloaded). VA-ECMO adds retrograde aortic flow, raising afterload; the loop shifts right and up (LV distension), which is why ECMO patients may need venting.

CVP / RA waveform.

Wave	Mechanical event	Abnormality and cause
a	Atrial contraction (follows ECG P wave)	Cannon a: AV dissociation, junctional rhythm, complete heart block. Absent a: atrial fibrillation
c	Tricuspid valve bulging into RA during isovolumic contraction	Often merged with a
x descent	Atrial relaxation and descent of the tricuspid annulus	Blunted in TR
v	Venous filling of RA against a closed tricuspid valve	Large v (c-v wave): severe TR
y descent	Tricuspid opening, RA empties into RV	Steep y: constrictive pericarditis, RV failure. Blunted y: tamponade

Read CVP at end-expiration, at the base of the c wave (or mean of the a wave), which best reflects RV end-diastolic pressure.

PAWP waveform. Same a and v morphology (left atrial), delayed. **Giant v waves** suggest acute severe MR (papillary muscle rupture post-MI, failed mitral repair) and can make the PA tracing look like a wedge, so read the wedge at end-expiration, at the base of the v wave. A wedge higher than PADP points to a measurement error or a large v wave.

Arterial waveform. Upstroke slope roughly reflects contractility (dP/dt). The dicrotic notch marks aortic valve closure; it moves lower and is lost with vasodilation and distal sampling sites. Respiratory variation estimates fluid responsiveness (PPV). Pulsus paradoxus (>10 mmHg systolic drop on inspiration, spontaneous breathing) suggests tamponade. A pulsus alternans pattern indicates severe LV dysfunction. Use a fast-flush test to check damping before trusting any number.

13. CTICU-specific calculations

The sections above are the hemodynamic core shared by every cardiac ICU. A cardiothoracic ICU adds the bypass circuit, lung resection, mechanical ventilation after thoracic surgery, ECMO, post-op valve assessment, and transplant size matching.

Estimated blood volume (EBV)

$$\text{EBV} = \text{weight (kg)} \times 70 \text{ mL/kg (men) or } \times 65 \text{ mL/kg (women)}$$

Where:

70 / 65 Typical adult blood volume per kg; lower in obesity (fat is poorly perfused), higher in athletes

Normal / target: ~5 L in a 70 kg man

Predicted hematocrit on cardiopulmonary bypass

$$\text{Hct}_{\text{CPB}} = (\text{EBV} \times \text{Hct}_{\text{preop}}) / (\text{EBV} + \text{prime} + \text{cardioplegia} + \text{crystalloid})$$

Where:

Hct_{preop} Pre-bypass hematocrit (as a decimal or a percent, used consistently)

Prime Crystalloid/colloid volume priming the circuit, typically 1.0–1.5 L (less with mini-circuits or retrograde autologous priming)

Cardioplegia, crystalloid Additional non-blood volume added before or during the run, mL

Normal / target: Most centers aim to keep on-pump Hct ≥21–24%

Bedside pearl: This is the dilution equation: the patient's red cell mass is fixed, the volume it's spread through grows. It's how perfusion decides before the case whether to add blood to the prime. Small patients with low preop Hct dilute the most.

Allowable blood loss (ABL)

$$\text{ABL} = \text{EBV} \times (\text{Hct}_{\text{initial}} - \text{Hct}_{\text{target}}) / \text{Hct}_{\text{average}}$$

Where:

Hct_{average} $(\text{Hct}_{\text{initial}} + \text{Hct}_{\text{target}}) / 2$

Hct_{target} Lowest acceptable hematocrit for this patient

Pump flow index and oxygen delivery on bypass

Pump flow index = pump flow (L/min) / BSA

$DO_{2I_{CPB}} = \text{pump flow index} \times CaO_2 \times 10$

Where:

Pump flow Arterial pump flow set by perfusion, L/min

CaO_2 From the on-bypass arterial gas and Hb

Normal / target: Pump flow index 2.2–2.4 L/min/m² at normothermia; DO_{2I} on CPB ≥ 272 –280 mL/min/m²

Bedside pearl: Goal-directed perfusion (Ranucci and colleagues) showed AKI risk climbs when on-pump DO_{2I} drops below roughly 270–280 mL/min/m². When the Hct is low, perfusion raises flow to protect DO_{2I} rather than reflexively transfusing.

Heparin and protamine dosing

Heparin (initial) = 300–400 U/kg IV; target ACT >480 s before initiating bypass

Protamine = 1–1.3 mg per 100 U heparin remaining

Where:

ACT Activated clotting time, seconds (baseline ~100–140 s)

Heparin remaining Total given minus estimated metabolism, or measured by heparin-protamine titration (e.g. Hepcon)

Bedside pearl: Heparin resistance (ACT stays low despite adequate dosing) is classically antithrombin deficiency, common after preop heparin infusions. Protamine excess is itself anticoagulant, so more is not better. Watch for protamine reactions (hypotension, pulmonary hypertensive crisis), especially with prior NPH insulin exposure or fish allergy.

Predicted postoperative FEV₁ and DLCO (lung resection)

$ppoFEV_1 = \text{preop } FEV_1 \times (1 - \text{segments resected} / 19)$

$ppoDLCO = \text{preop } DLCO \times (1 - \text{segments resected} / 19)$

Where:

FEV_1 Forced expiratory volume in 1 s, as % predicted

19 Total bronchopulmonary segments (RUL 3, RML 2, RLL 5, LUL 3, lingula 2, LLL 4)

Segments resected Functional (unobstructed) segments being removed; e.g. lobectomy RLL = 5

$VO_{2\text{max}}$ Peak oxygen consumption on cardiopulmonary exercise testing, mL/kg/min

Normal / target: $ppoFEV_1$ and $ppoDLCO$ >60% = low risk; either <60% → exercise testing; <30–40% = high risk. $VO_{2\text{max}}$ >20 mL/kg/min safe for resection, <10 mL/kg/min prohibitive

Bedside pearl: Use the same formula for $ppoDLCO$. If a tumor already obstructs segments, they contribute nothing preop, so removing them costs nothing; the perfusion-scan method handles this better than simple segment counting.

Driving pressure and static compliance

Driving pressure (ΔP) = P_{plat} – PEEP

Static compliance (C_{stat}) = $V_T / (P_{\text{plat}} - \text{PEEP})$

Where:

P_{plat} Plateau pressure, cmH₂O (inspiratory hold)

PEEP Total PEEP, cmH₂O

V_T Tidal volume, mL

Normal / target: ΔP <15 cmH₂O; ventilated compliance ~50–80 mL/cmH₂O

Bedside pearl: Driving pressure is V_T scaled to the size of the ventilated lung, and it tracks ARDS mortality better than V_T or plateau alone (Amato 2015). During one-lung ventilation the ventilated lung is half the size, so the same V_T produces a much higher ΔP . Most centers use 4–6 mL/kg PBW on OLV.

Dead space fraction (Bohr-Engelhoff)

$V_D/V_T = (PaCO_2 - P_{\bar{E}}CO_2) / PaCO_2$

Where:

$P_{\bar{E}}CO_2$ Mixed expired CO₂ (volumetric capnography), mmHg — not end-tidal

Normal / target: 0.2-0.3; >0.57 in early ARDS predicts mortality

Bedside pearl: A rising dead space after cardiac surgery should prompt a search for PE, low cardiac output (poor pulmonary perfusion), or overdistension from excessive PEEP.

Oxygenation index (OI)

$$OI = (FiO_2 \times \text{mean airway pressure} \times 100) / PaO_2$$

Where:

FiO₂ As a decimal
Mean airway pressure P_{aw}, cmH₂O (from the ventilator)

Normal / target: Higher is worse; adult values >20-25 indicate severe hypoxemic failure and help frame ECMO candidacy

Bedside pearl: Better than P/F for ventilated patients because it penalizes the airway pressure needed to achieve the oxygenation.

ECMO recirculation fraction (VV)

$$R = (S_{pre}O_2 - SvO_2) / (S_{post}O_2 - SvO_2)$$

Where:

S_{pre}O₂ Saturation of blood entering the oxygenator (drainage limb)
S_{post}O₂ Saturation leaving the oxygenator (return limb), usually ~100%
SvO₂ True systemic venous saturation before mixing with return blood (estimated from a site the return jet doesn't reach)

Normal / target: As low as possible; >20-30% is clinically significant

Bedside pearl: Recirculated blood is oxygenated blood that goes straight back into the drainage cannula without reaching the patient. Suspect it when the pre-oxygenator saturation is high but the patient is hypoxemic; fixes include repositioning cannulas (increase separation), lowering flow, or a dual-lumen cannula.

Oxygenator O₂ transfer and ECMO flow

$$VO_{2,ECMO} = Q_{ECMO} \times (C_{post}O_2 - C_{pre}O_2) \times 10$$

$$ECMO \text{ flow (mL/kg/min)} = Q_{ECMO} \times 1000 / \text{weight}$$

Where:

Q_{ECMO} Circuit blood flow, L/min
C_{post}O₂, C_{pre}O₂ O₂ content leaving and entering the oxygenator: $1.34 \times Hb \times S + 0.003 \times PO_2$

Normal / target: Flow ~50-80 mL/kg/min (VV typically 60-80; VA titrated to perfusion and LV distension). Failing oxygenator: falling post-oxygenator PO₂ (<150-200 mmHg on FiO₂ 1.0) or rising transmembrane pressure

Bedside pearl: Blood flow controls oxygenation, sweep gas controls CO₂. In VV ECMO, arterial saturation depends on the ratio of ECMO flow to native cardiac output: as native CO rises (fever, agitation), a smaller fraction of venous return passes through the circuit.

PISA: effective regurgitant orifice area (MR)

$$EROA = 2\pi r^2 \times V_{alias} / V_{max,MR}$$

$$\text{Regurgitant volume} = EROA \times VTI_{MR}$$

Where:

r PISA hemisphere radius, cm (from the aliasing boundary to the orifice)
V_{alias} Aliasing velocity (Nyquist limit set by baseline shift), cm/s
V_{max,MR} Peak MR jet velocity by CW Doppler, cm/s
VTI_{MR} Velocity-time integral of the MR jet, cm

Normal / target: Severe primary MR: EROA ≥0.40 cm², regurgitant volume ≥60 mL

Bedside pearl: The flow converging on the orifice forms hemispheric shells of equal velocity; flow through one shell equals flow through the orifice. Post-repair, any residual MR worse than mild, or SAM with LVOT obstruction, needs attention before leaving the OR.

Mitral valve area by pressure half-time

$$\text{MVA} = 220 / \text{PHT}$$

Where:

PHT Pressure half-time: time for the transmitral gradient to fall to half its peak, ms

220 Empirical constant

Normal / target: Normal 4–6 cm²; severe MS ≤1.5 cm²

Bedside pearl: Unreliable immediately after balloon valvotomy and with altered LA or LV compliance (e.g. severe AR, diastolic dysfunction).

E/e' (LV filling pressure)

$$\text{E/e'} = \text{mitral E velocity} / \text{average mitral annular e' velocity}$$

Where:

E Early diastolic transmitral inflow velocity, cm/s

e' Early diastolic tissue Doppler annular velocity (average of septal and lateral), cm/s

Normal / target: Average E/e' >14 suggests elevated LA pressure

Predicted heart mass (PHM) for heart transplant size matching

$$\text{PHM} = \text{predicted LV mass} + \text{predicted RV mass}$$

$$\text{LV mass (g)} = a \times \text{height}^{0.54} \times \text{weight}^{0.61}$$

$$\text{RV mass (g)} = a \times \text{age}^{-0.32} \times \text{height}^{1.135} \times \text{weight}^{0.315}$$

$$\text{PHM mismatch (\%)} = (\text{donor PHM} - \text{recipient PHM}) / \text{recipient PHM} \times 100$$

Where:

Height Meters

Weight Kilograms

Age Years

a (LV) 6.82 women, 8.25 men

a (RV) 10.59 women, 11.25 men

Normal / target: Donor undersizing worse than about –30% PHM mismatch is associated with worse graft survival

Bedside pearl: PHM outperforms weight-matching because it accounts for sex and age, which strongly affect ventricular mass. A small male donor heart into a larger female recipient can be well matched by PHM even if weights differ. UNOS uses recipient PHM as the reference for the % mismatch.

Predicted total lung capacity (pTLC) for lung transplant size matching

$$\text{pTLC (men)} = 7.99 \times \text{height} - 7.08 \quad \text{pTLC (women)} = 6.60 \times \text{height} - 5.79$$

Where:

Height Meters

pTLC Liters (ECSC/Quanjer 1993 reference equations)

Normal / target: Donor/recipient pTLC ratio near 1.0 (roughly 0.8–1.2 commonly accepted, varying with recipient diagnosis)

Bedside pearl: Oversized lungs are usually tolerated better in obstructive disease (hyperexpanded chest) than in restrictive disease. Oversizing is associated with lower primary graft dysfunction risk in several registry analyses; marked undersizing with more.

Risk models. STS PROM (Society of Thoracic Surgeons Predicted Risk of Mortality) and EuroSCORE II are the standard cardiac surgical risk calculators. Both are multi-variable logistic models with dozens of inputs, so use the official online calculators rather than a formula.

14. Worked example: putting the oxygen chain together

Scenario: Post-CABG, 4 hours. HR 110, MAP 64, CVP 14, PAWP 18, CO 3.5 L/min (thermodilution), BSA 2.0 m², Hb 10, SaO₂ 0.98, PaO₂ 100, SvO₂ 0.55, PvO₂ 30, PaCO₂ 40, PvCO₂ 49.

Calculation	Working	Result	Interpretation
CI	3.5 / 2.0	1.75 L/min/m ²	Low
CaO ₂	$1.34 \times 10 \times 0.98 + 0.003 \times 100$	13.4 mL/dL	Low (anemia)
CvO ₂	$1.34 \times 10 \times 0.55 + 0.003 \times 30$	7.5 mL/dL	
DO ₂	$3.5 \times 13.4 \times 10$	470 mL/min	Roughly half of normal
VO ₂	$3.5 \times (13.4 - 7.5) \times 10$	~209 mL/min	Preserved, being maintained by extraction
O ₂ ER	209 / 470	0.44	Near its ceiling
CO ₂ gap	49 – 40	9 mmHg	Flow is inadequate
SVR	$80 \times (64 - 14) / 3.5$	1143	Normal-high
CPO	$64 \times 3.5 / 451$	0.50 W	Below 0.6 W, concerning

Synthesis: Low-output state with extraction approaching its limit and an elevated CO₂ gap. This is cold, with PAWP sitting right at the 18 mmHg cutoff (Forrester III/IV border). The priorities are increasing CO (inotrope, rule out tamponade, check RV function with PAPI and echo) and considering whether Hb support would meaningfully raise DO₂. Adding volume to a PAWP of 18 is unlikely to help.

Normal ranges vary modestly between references and institutions. Interpret every calculated value in the context of the measurement's reliability and the whole patient.