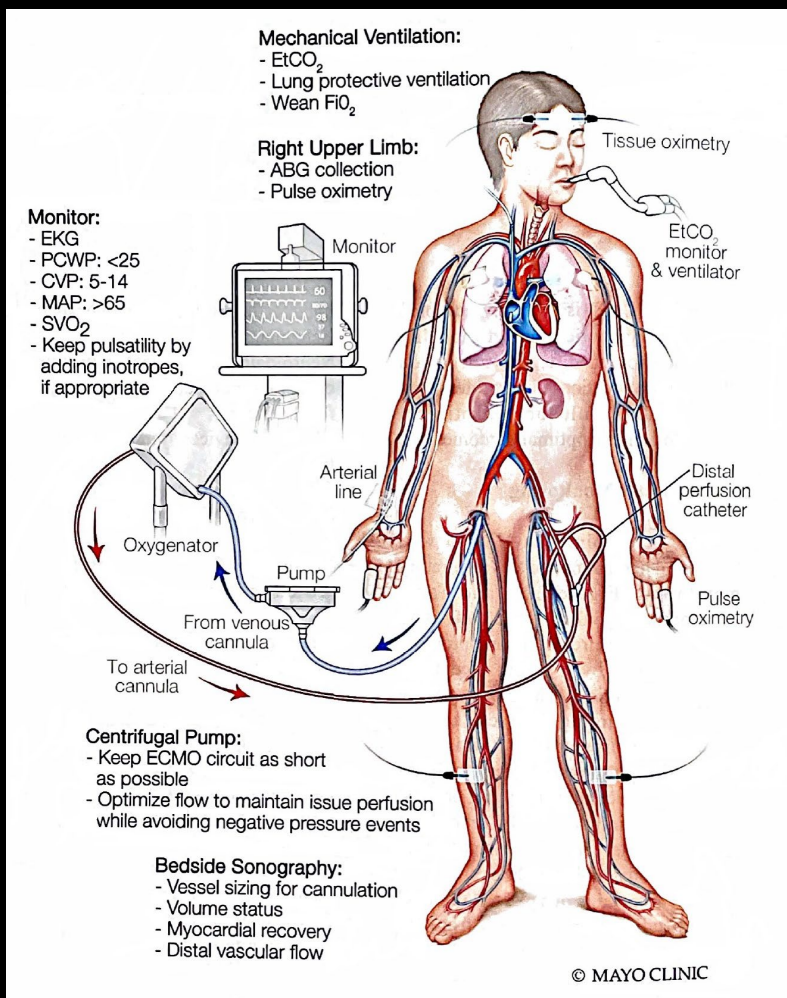


Hemodynamic monitoring in ECMO patients

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Hemodynamic monitoring

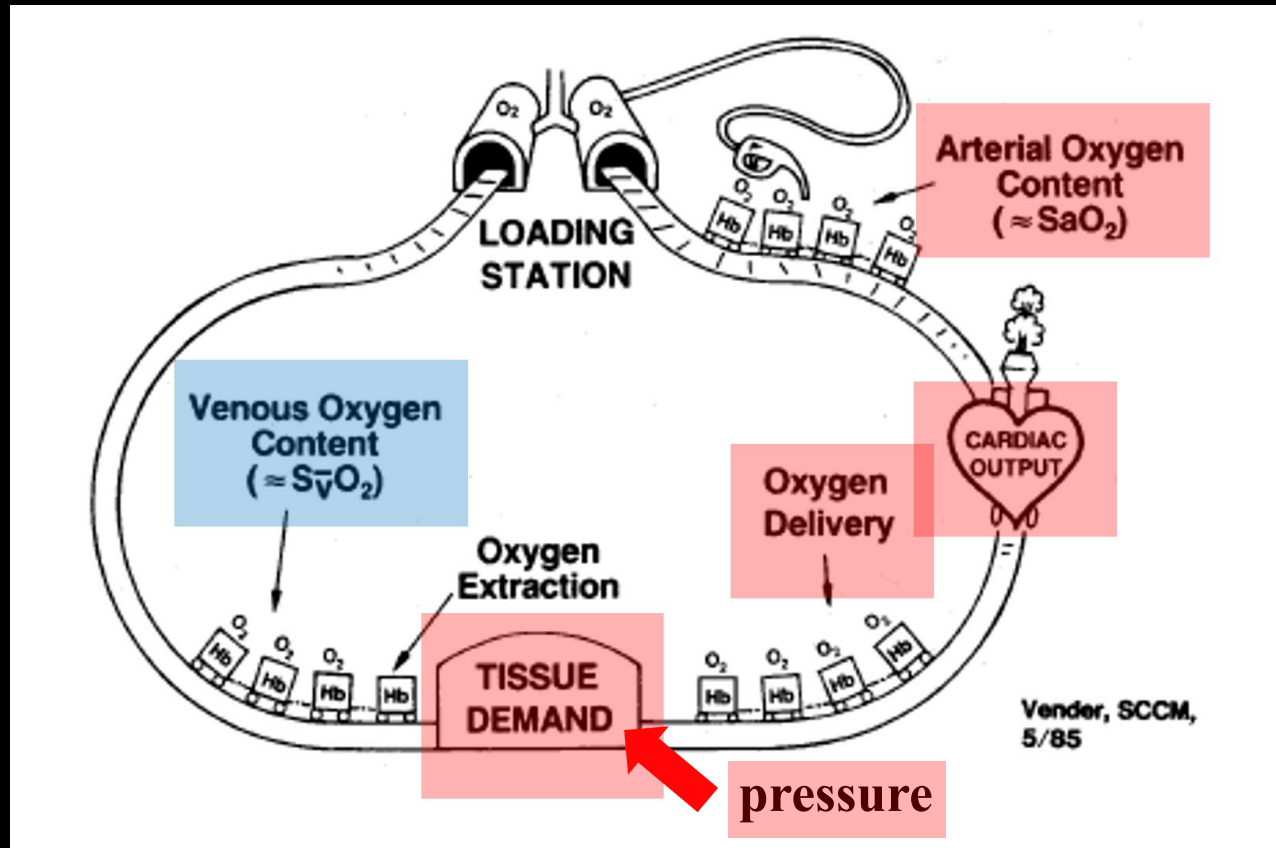


Parameter	Monitor for	Treatment
Rhythm	Dysrhythmia	Antiarrhythmics
MAP	ECMO flow, SVR	Vasoconstrictor
Pulsatility	Thrombus, pulmonary edema	Flow, inotrope, venting
Flow	Pre/afterload, RPM	Volume, vasodilator
Gas exchange	ABGA, sweep gas, oxygenator	Sweep, VAV ECMO
Oxygen delivery	Flow, Hb, SaO ₂ , SvO ₂ , lactate	Flow, transfusion, sedative
Limb ischemia	Pulse, color, NIRS	Distal perfusion
Anticoagulation	aPTT, ACT	Heparinization
Temperature	Normo/hypothermia	Heat exchanger

Ultimate goal of ECMO

- Mechanical support of cardiopulmonary function
 - heart : pumping blood to the organs and tissues for delivery of nutrients and oxygen in blood
 - lung : gas exchange for delivery of oxygen from the lungs to the blood
- Oxygen delivery (DO_2) to vital organs
 - in proportion to oxygen consumption (VO_2)
- Prevention of multi-organ failure
- Bridge to recovery or transplantation (heart \pm lung)

Oxygen : delivery and consumption



1. Oxygen content

2. ECMO flow

3. Volume

4. Pressure

5. SvO_2

Oxygen delivery (DO_2)

- The amount of O_2 delivered to peripheral tissue each minute
- Arterial oxygen content (CaO_2) \times cardiac output (CO)
- Oxygen content = $(Hb \times 1.38 \times SaO_2) + (0.0031 \times PaO_2)$
= O_2 bound + O_2 dissolved
- Monitoring : Hb, SaO_2 , PaO_2 , ECMO flow
 - CBC : RBC transfusion
 - ABGA : adjustment of ECMO FiO_2
 - ECMO flow : optimization (RPM, volume replacement, vasopressor)
 - appropriate DO_2 in proportion to VO_2

Oxygen consumption ($\dot{V}O_2$)

- The volume of O_2 consumed per minute
- $\dot{V}O_2$ in normal resting adult : 3~5cc/kg/min
- Controlled by tissue metabolism
 - $\dot{V}O_2 \uparrow$: seizure, infection, hyperthermia, catecholamine & thyroid hormones \uparrow
 - $\dot{V}O_2 \downarrow$: rest, paralysis, hypothermia
- Monitoring : depth of sedation (RASS), body temperature
 - agitation, delirium : sedative \pm muscle relaxant
 - fever : antipyretics, heater unit (PLS), heat exchanger

Relationship : $\dot{D}O_2$ and $\dot{V}O_2$

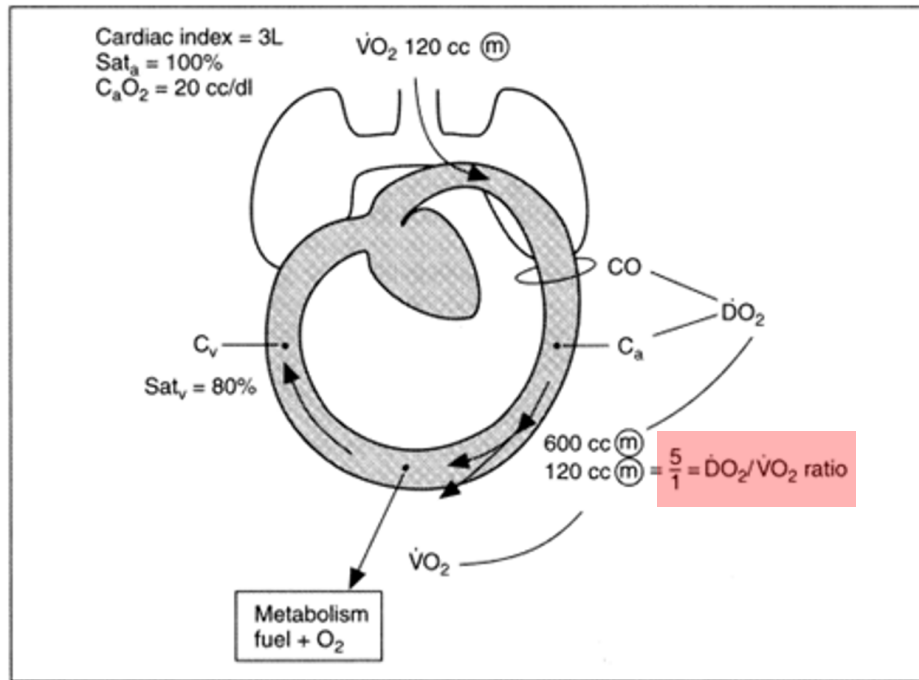


FIGURE 1-1. Oxygen kinetics. Oxygen delivery ($\dot{D}O_2$) is the product of cardiac output (CO) times the arterial oxygen content (C_a). Oxygen delivery is normally four to five times oxygen consumption ($\dot{V}O_2$). (C_v = venous oxygen content; $\text{(m)} = /min/m^2$; Sat_a = arterial saturation; Sat_v = venous saturation.)

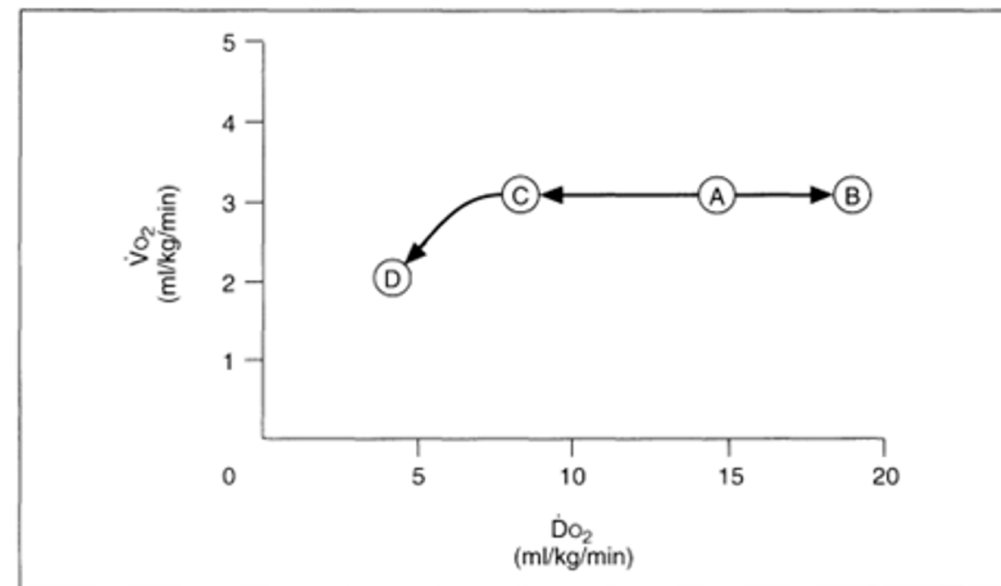
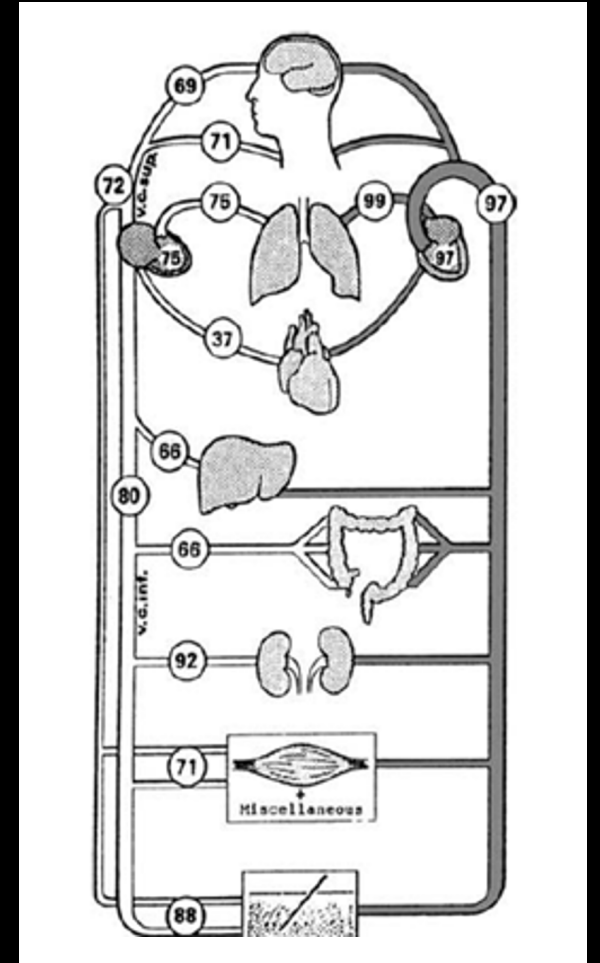


FIGURE 1-11. The normal relationship between $\dot{V}O_2$ and $\dot{D}O_2$. The normal point (A) is shown as $\dot{V}O_2$ 120 cc/m²/min and $\dot{D}O_2$ 600 cc/m²/min. If $\dot{D}O_2$ is increased by transfusion (B), $\dot{V}O_2$ remains constant. If $\dot{D}O_2$ is progressively decreased (A to C), $\dot{V}O_2$ remains constant until the ratio of $\dot{D}O_2 / \dot{V}O_2$ falls below 2:1 (C to D).

SvO₂ : ratio of DO₂ to VO₂

- Venous oxygen content
 - mixed venous blood oxygen saturation
- $DO_2/VO_2 = 5:1$
- In an average adult
 - $DO_2 : 1000\text{ml}/\text{min}$, $VO_2 : 200\text{ml}/\text{min}$
- The amount of O₂ extracted : 20% of delivery
- 80% of O₂ : in venous blood → return to heart
- The oxygen saturation of mixed venous blood : 80%



SvO_2 : ratio of DO_2 to VO_2

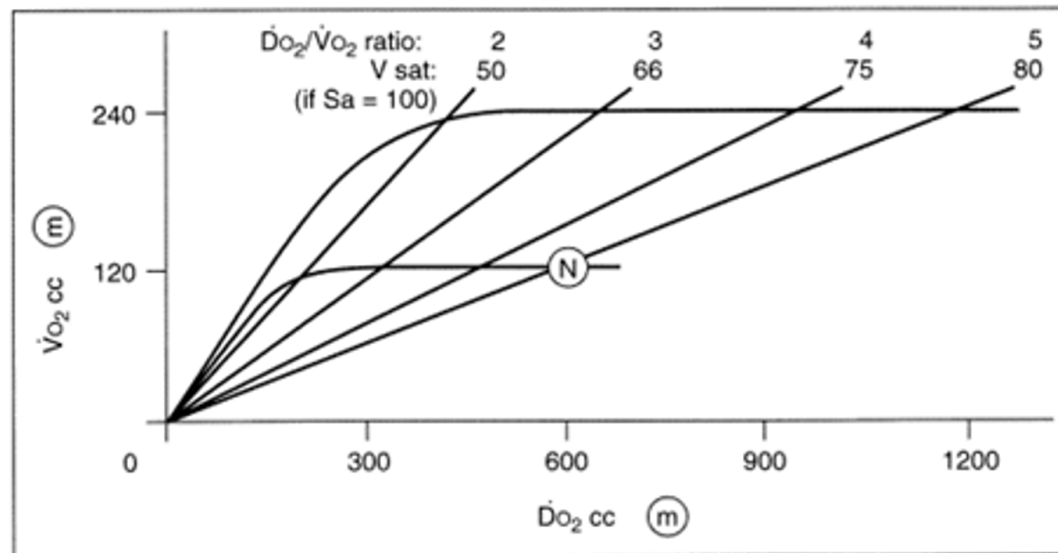
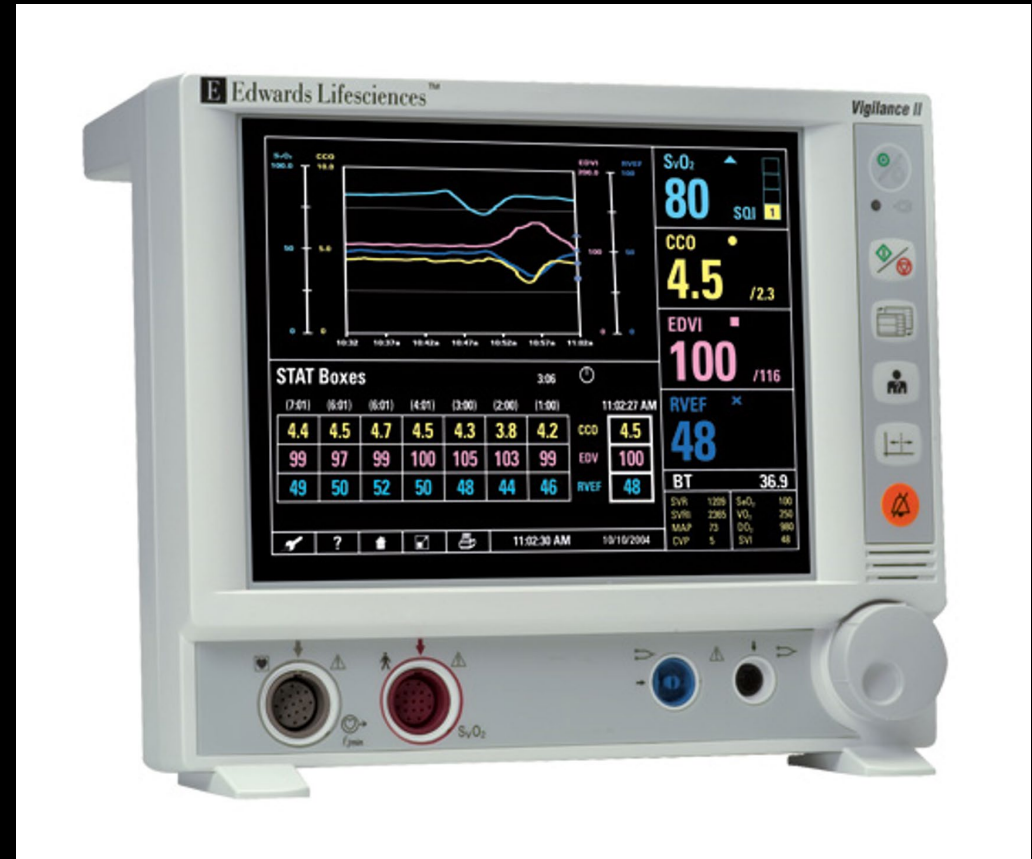
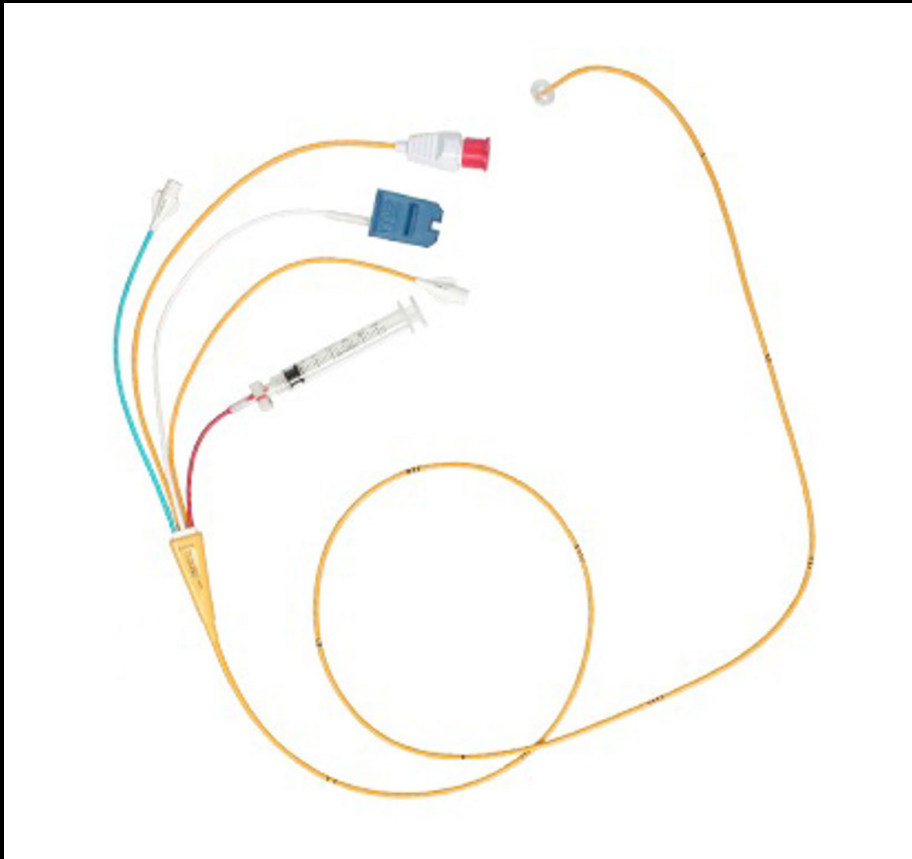
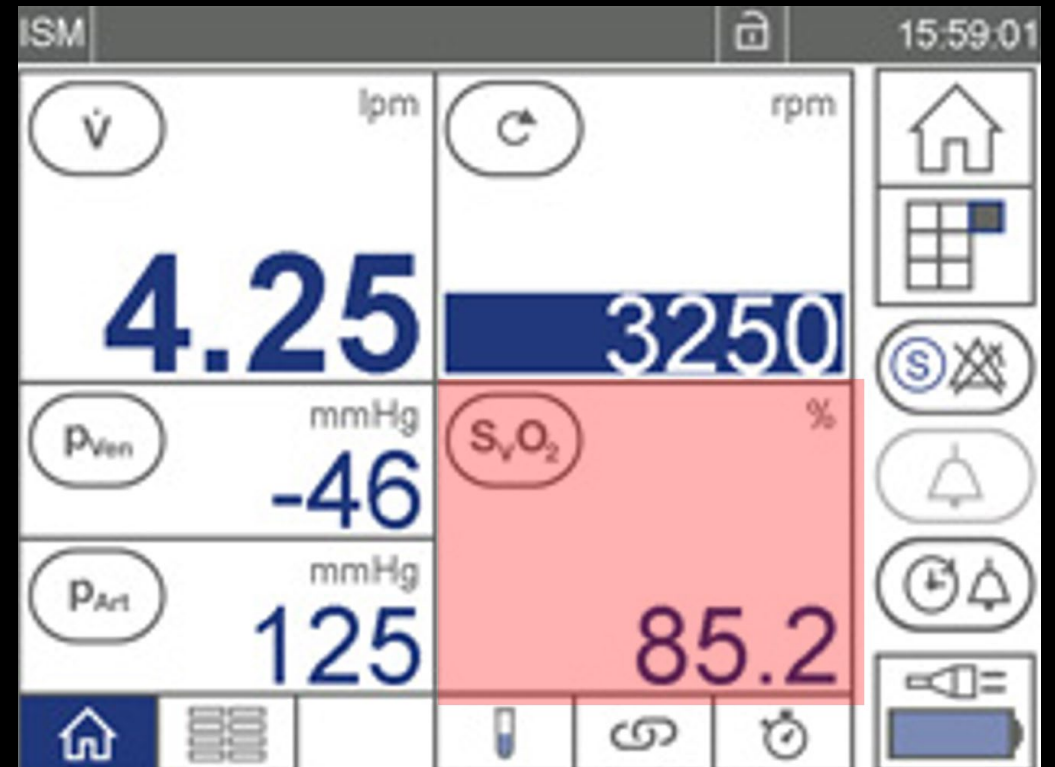


FIGURE 1-12. $\dot{D}O_2/\dot{V}O_2$ relationships during normal metabolism (as in Figure 1-11) and during hypermetabolism. During normal, hypometabolic, or hypermetabolic states the normal ratio of delivery to consumption is 5:1. This results in 80% venous saturation ($V\ sat$) if the arterial blood is 100% saturated. The isobar for the 5:1 ratio is demonstrated in this diagram, as well as the isobar for the 4:1, 3:1, and 2:1 ratios. Corresponding levels of venous saturation are shown. A state of decreasing oxygen consumption in which consumption is supply dependent occurs when the ratio is less than 2:1. (\textcircled{m} = /min/m^2 ; S_a = arterial saturation.)

SvO₂ monitoring



SvO₂ monitoring



SvO_2 : venous oxygen content



Earn not enough



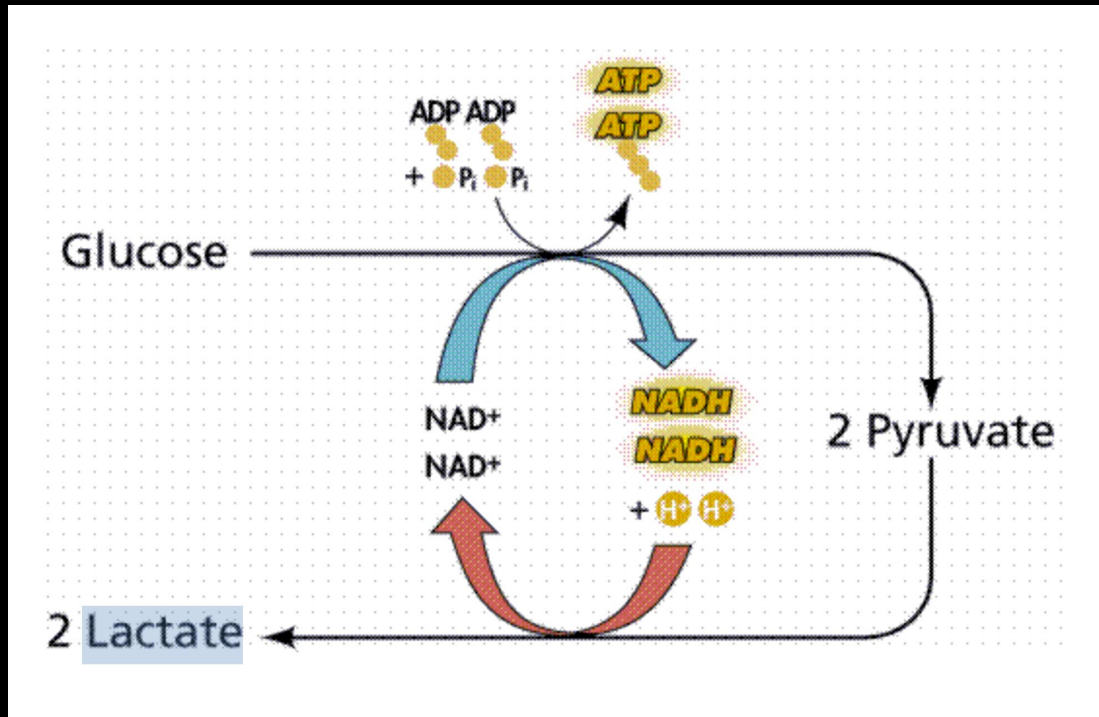
Spend too much

If SvO_2 is bank statement at the end of the month, the balance of income and expenditure is appropriate ?

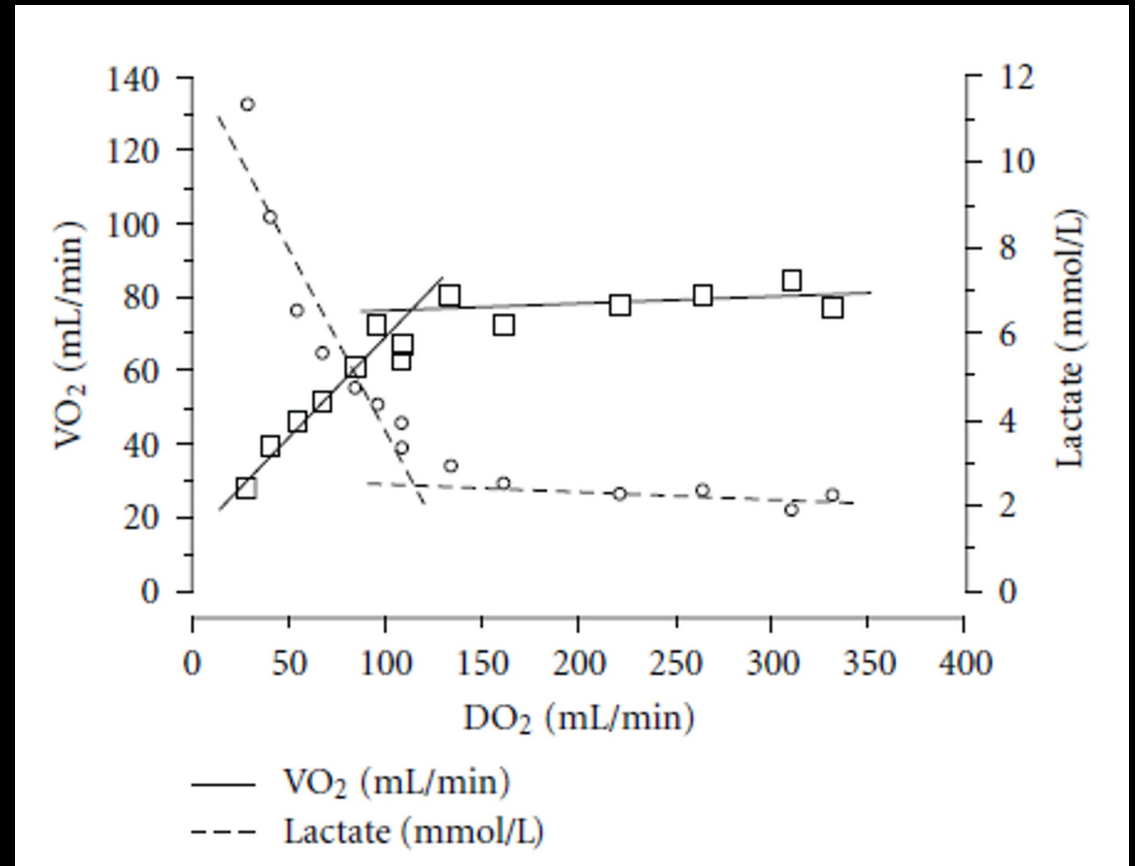
Lactate : DO_2/VO_2 balance



Lactate : DO_2/VO_2 balance



Inadequate DO_2 slows mitochondrial metabolism \rightarrow anaerobic metabolism \rightarrow pyruvate is converted to lactate



Lactate : predictor of mortality

Stephen Trzeciak
R. Phillip Dellinger
Michael E. Chansky
Ryan C. Arnold
Christa Schorr
Barry Milcarek
Steven M. Hollenberg
Joseph E. Parrillo

Serum lactate as a predictor of mortality in patients with infection

- 1,177 patients with primary diagnosis of infection and serum lactate
- Lactate levels were divided into low (0–2 mmol/L), intermediate (2.1–3.9 mmol/L), and high (>4.0 mmol/L)
- Lactate level of 4 mmol/L or more was found to be highly specific (89%–99%) for predicting the acute phase of death and in-hospital death

(Intensive Care Med. 2007 Jun;33(6):970-7)

LACTATE CLEARANCE AND SURVIVAL FOLLOWING INJURY

David Abramson, MD, Thomas M. Scalea, MD, Robyn Hitchcock, MD, Stanley Z. Trooskin, MD,
Sharon M. Henry, MD, and Joshua Greenspan, MD

- 76 patients with trauma and hemorrhagic shock
- No death when lactate levels returned to normal within 24 hours
- 86% of patients died when lactate levels remained after 48 hours

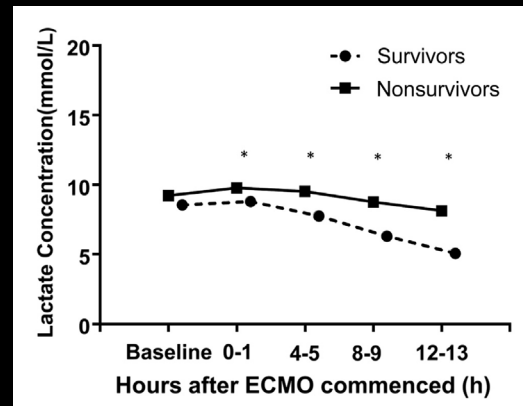
(J Trauma. 1993 Oct;35(4):584-8)

Lactate : predictor of mortality

The early dynamic behavior of lactate is linked to mortality in postcardiotomy patients with extracorporeal membrane oxygenation support: A retrospective observational study

Cheng-Long Li, MD,^a Hong Wang, MD, PhD,^a Ming Jia, MD,^a Ning Ma, MD, PhD,^b and Xiao-Tong Hou, MD, PhD^a

- N=123, VA-ECMO
- Refractory post-cardiotomy cardiogenic shock
- Significant difference in lactate clearance between survivors and non-survivors

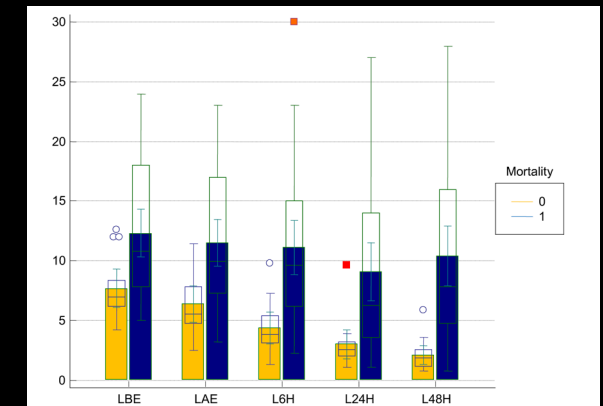


(J Thorac Cardiovasc Surg 2015;149:1445-50)

Does lactate clearance prognosticates outcomes in ECMO therapy: a retrospective observational study

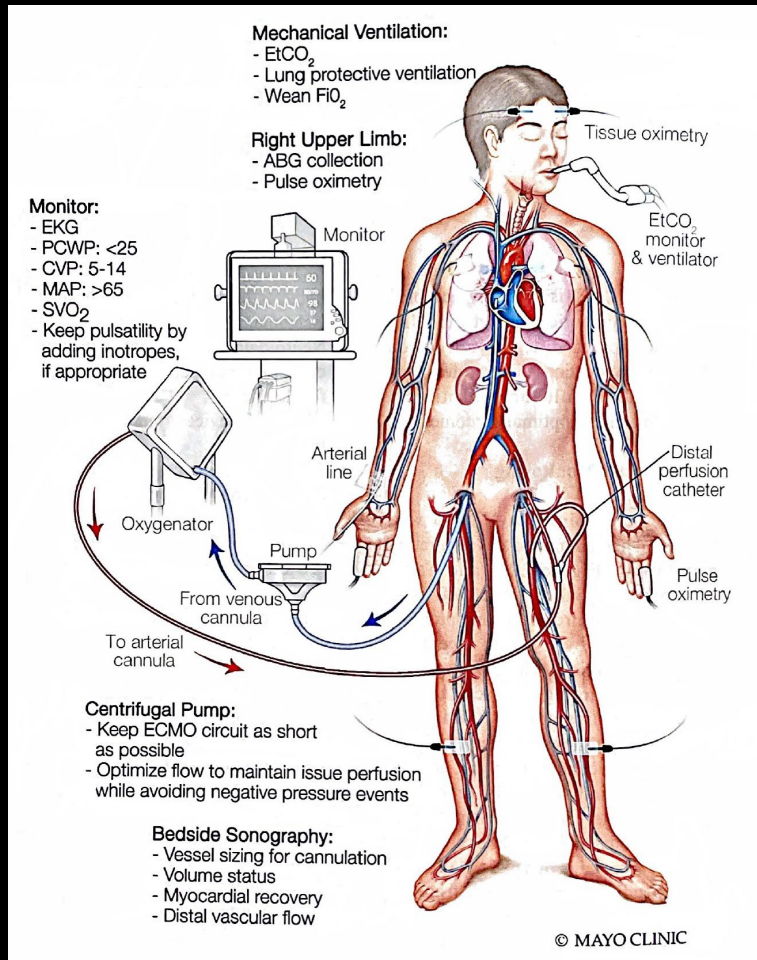
İbrahim Mungan^{*}, Dilek Kazancı, Şerife Bektaş, Derya Ademoglu and Sema Turan

- N=48, VA-ECMO
- Cardiogenic shock
- Significant correlation between dynamic changes in lactate levels and 30-day mortality



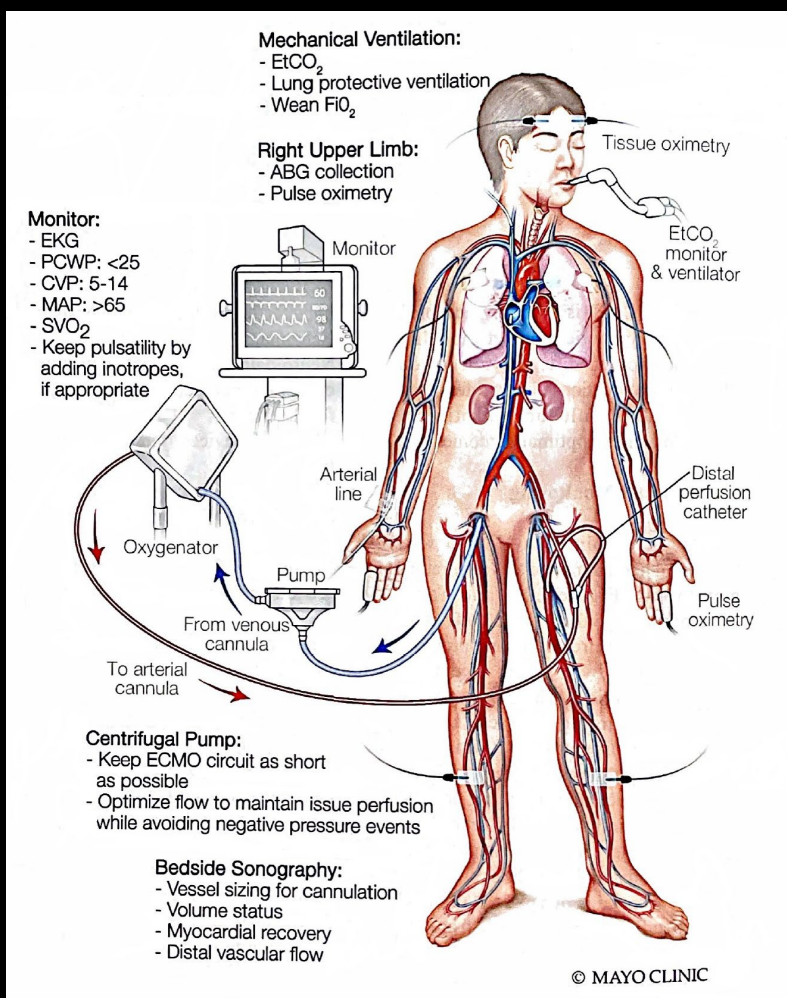
(BMC Anesthesiol. 2018 Oct 24;18(1):152)

Hemodynamic monitoring



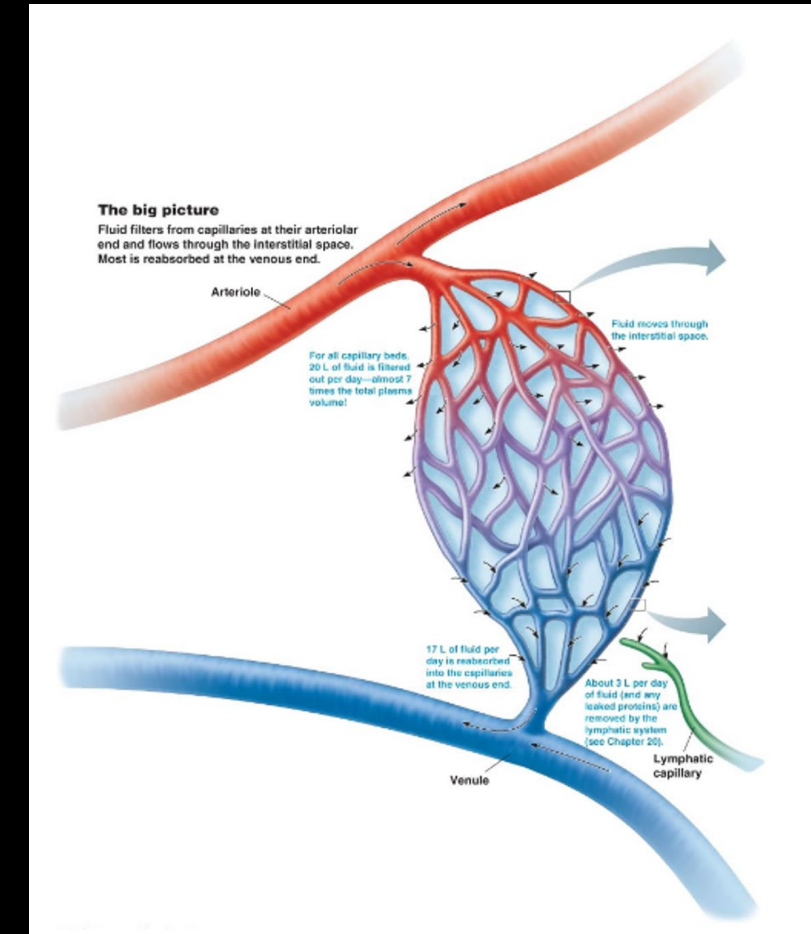
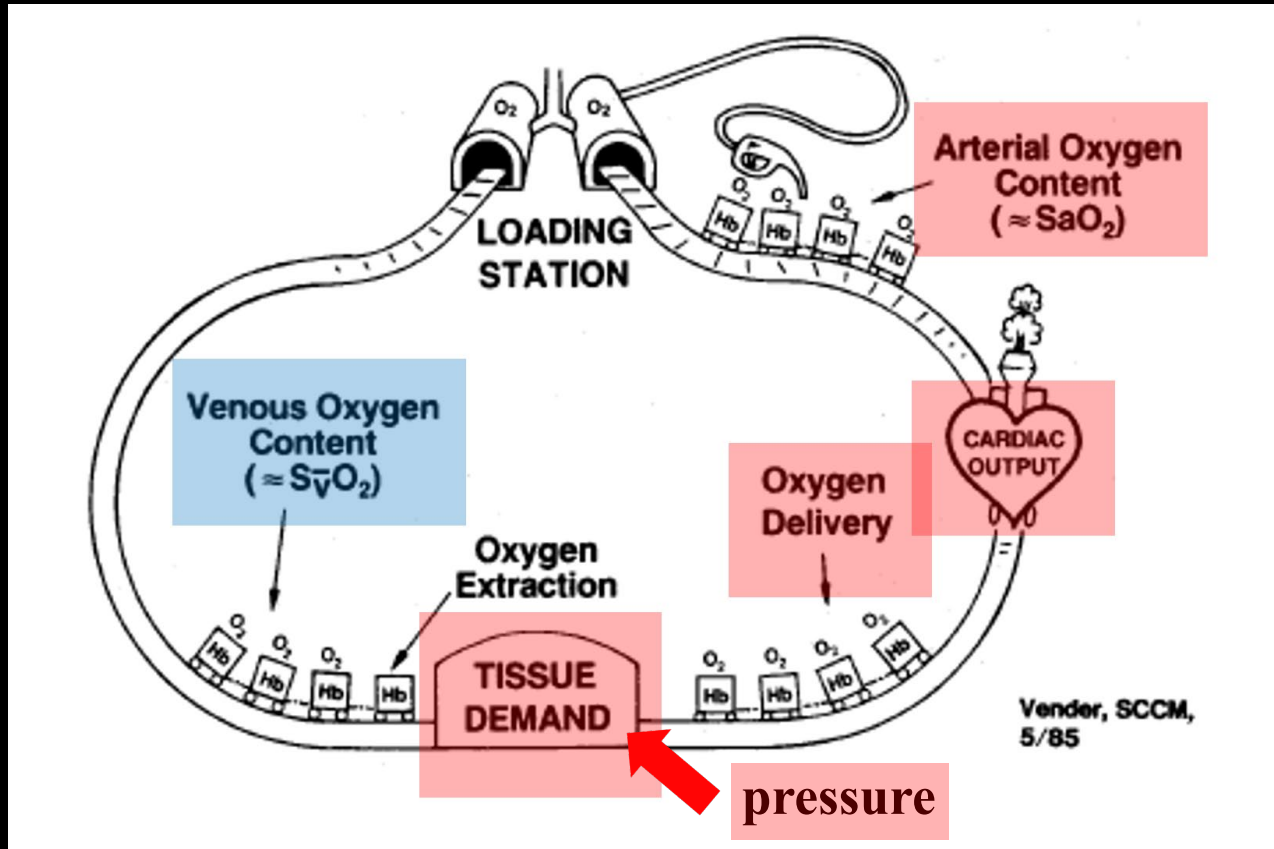
Parameter	Monitor for	Treatment
Oxygen delivery: SvO ₂ and lactate	Decreased SvO ₂ and increasing lactate suggest inadequate O ₂ delivery (DO ₂ = CaO ₂ × CO)	<ol style="list-style-type: none"> 1. Transfuse 2. Ensure adequate gas exchange 3. Increase ECMO flow
	<ol style="list-style-type: none"> 1. Hemoglobin 2. SaO₂ 3. PaO₂ 4. ECMO flow 	
	Excessive O ₂ consumption	<ol style="list-style-type: none"> 4. Heat exchanger, antipyretics 5. Consider agents such as dexmedetomidine
	<ol style="list-style-type: none"> 1. Febrile 2. Agitation 	

Hemodynamic monitoring



Parameter	Monitor for	Treatment
Rhythm	Dysrhythmia	Antiarrhythmics
MAP	ECMO flow, SVR	Vasoconstrictor
Pulsatility	Thrombus, pulmonary edema	Flow, inotrope, venting
Flow	Pre/afterload, RPM	Volume, vasodilator
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Limb ischemia	Pulse, color, NIRS	Distal perfusion
Anticoagulation	aPTT	Heparinization
Temperature	Normo/hypothermia	Heat exchanger

Tissue perfusion : pressure



Systemic arterial BP for tissue perfusion : MAP

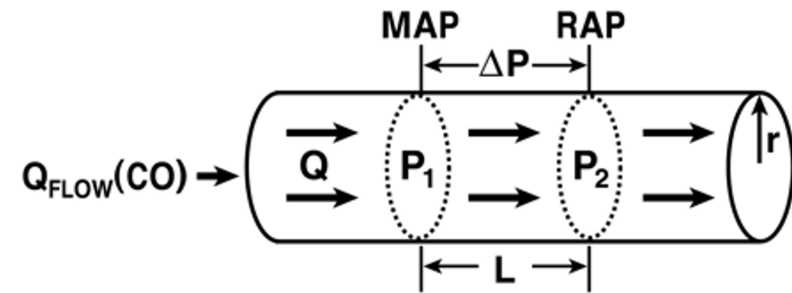
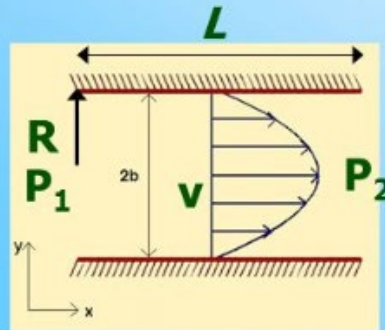
Tissue perfusion : pressure

- **Rate of flow** : the volume of fluid which passes through a given surface per unit time (m^3/s)

- **Poiseuille's equation** :

$$\text{Rate of flow} = \frac{\Delta V}{\Delta t} = \frac{\pi R^4 (P_1 - P_2)}{8\eta L}$$

η : viscosity of the fluid



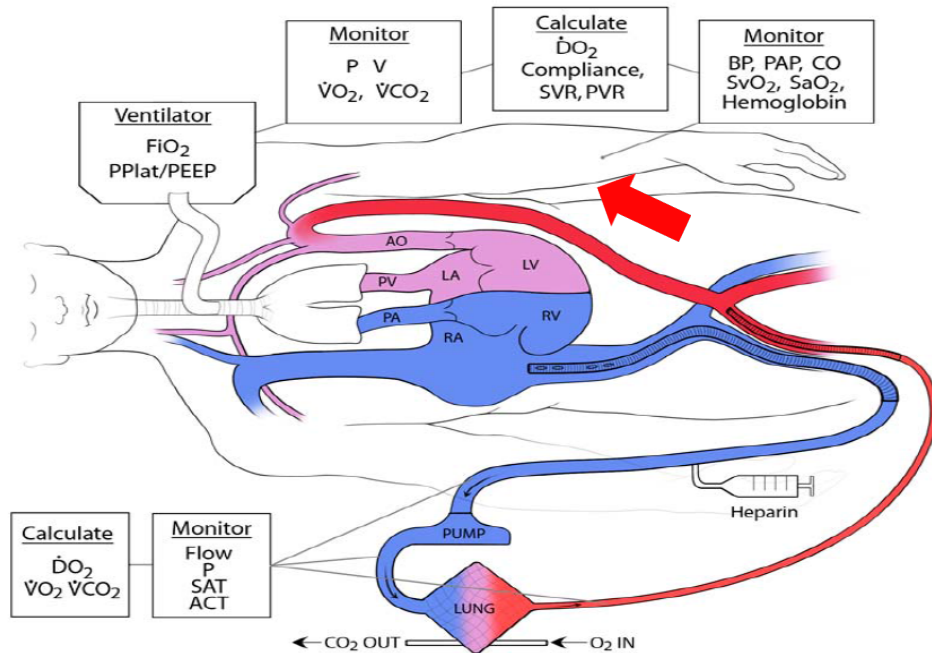
$$Q_{\text{FLOW}} = \frac{P_1 - P_2}{R = \frac{8\mu L}{\pi r^4}} \quad \text{CO} = \frac{\text{MAP} - \text{RAP}}{\text{SVR}}$$

- Blood flow = perfusion pressure / systemic vascular resistance (SVR)
- Perfusion pressure = blood flow \times SVR
- Perfusion pressure = mean arterial pressure (MAP) – systemic venous pressure
- $\text{MAP} \propto \text{ECMO flow} \times \text{SVR}$ (by vasoconstrictor)

Physiology : peripheral VA–ECMO

- Drainage of venous blood from RA → return of oxygenated blood to femoral artery
 - partial bypass
 - retrograde ECMO flow + anterograde cardiac output ejected from LV
- Effect on hemodynamics
 - drainage of blood from RA → RAP & CVP ↓ → RV ejection into lung ↓ → venous return to LA ↓
- LVEDV & LVEDP ↓
 - myocardial stretch ↓ → ventricular unloading
 - coronary blood flow ↑ by coronary perfusion pressure ↑ = diastolic BP – LVEDP ↓
 - myocardial rest
- Promotion of heart recovery

Afterload : peripheral VA–ECMO



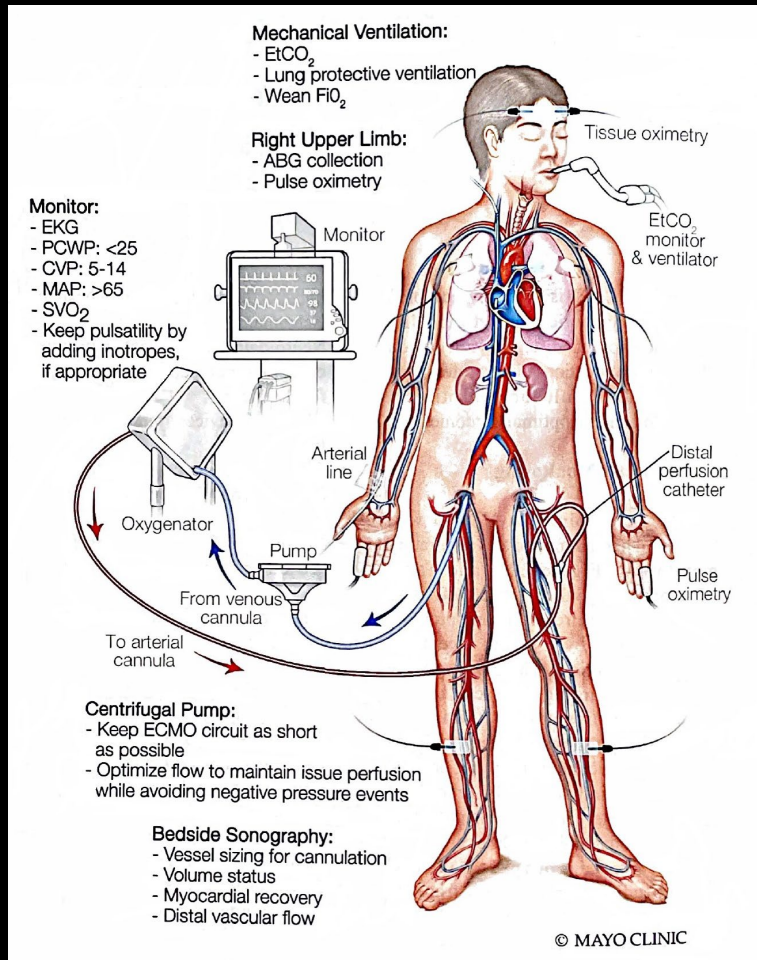
VA: Venoarterial access via the femoral vessels

- Retrograde blood flow into the aorta
 - not always problematic
 - only in case of excessive ECMO flow
- Inadequate LV systolic function to open AV → loss of native ejection → arterial pulse pressure ↓ → retention of blood in LV → LVEDP & LVEDV ↑
- Stagnation of blood → LV thrombosis
- LV distension & coronary blood flow ↓ → myocardial ischemia → myocardial recovery ↓
- LA hypertension → pulmonary edema
 - requirement for LV decompression (venting)

Tissue perfusion : pressure

During VA support hemodynamics are controlled by the blood flow (pump flow plus native cardiac output), and vascular resistance. Because the pulse pressure is low the mean systemic arterial pressure will be somewhat lower than normal pressure (40 to 50 mmHg for a newborn, 50 to 70 mmHg for a child or adult). In addition, patients placed on ECLS for cardiac support are on high doses of pressors when ECLS is begun. As these drugs are titrated down, resistance falls and systemic pressure falls proportionately. If the systemic perfusion pressure is inadequate (low urine output, poor perfusion) pressure can be increased by adding blood or low doses of pressor drugs. Systemic vasodilatation requiring pressor drugs is common in patients in septic shock. Although the mean arterial pressure may be low, systemic perfusion may be completely adequate. Systemic perfusion is best measured by mixed venous blood saturation. Assuming SaO_2 is over 95% venous saturation greater than 70% indicates systemic oxygen delivery is adequate even though the pressure may be low. If systemic oxygen delivery is not adequate (venous saturation less than 70%) increase the pump flow until perfusion is adequate. If extra blood volume is required to gain extra flow, consider the relative advantages of blood and crystalloid solution.

Hemodynamic monitoring



Parameter	Monitor for	Treatment
MAP	<p>Hypotension (MAP = CO × SVR)</p> <ol style="list-style-type: none"> 1. Inadequate ECMO flow 2. Inadequate SVR 	<ol style="list-style-type: none"> 1. Increase ECMO flow 2. Vasoconstrictor
Pulsatility	<p>decreased pulse pressure on arterial waveform caused by</p> <ol style="list-style-type: none"> 1. Poor myocardial function 2. Excessive VA-ECMO support <p>May result in LV thrombosis, myocardial ischemia, pulmonary edema</p>	<ol style="list-style-type: none"> 1. Inotropes 2. decrease ECMO flow 3. LV decompression (venting)

Summary

- Hemodynamic monitoring in ECMO patients
 - ultimate goal of ECMO support : oxygen delivery to vital organs
 - SvO₂ : balance between DO₂ and VO₂
 - lactate : result of anaerobic metabolism d/t inadequate DO₂
 - MAP : tissue perfusion pressure for oxygen delivery
 - pulse pressure : adequate LV afterload, requirement for LV decompression
- Optimal hemodynamic status in ECMO : should be individualized
 - according to various monitoring parameters
 - optimal balance between DO₂ and VO₂

Thank you for your attention !