



ECMO GOOD, BAD, UNKNOWN



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Disclosures

Speaker and Consultant Abiomed

Surgical Advisory Board Abiomed





ECMO INTRODUCTORY COURSE OBJECTIVES

- Appreciate the history of ECMO
- Recognize major types of ECMO, their indications and contraindications (venovenous (VV) and venoarterial (VA))
- Identify the components of the ECMO circuit
- Describe basic physiology of venovenous (VV) and venoarterial (VA) ECMO.
- Describe the process of ECMO weaning for VV and VA ECMO
- Identify common problems and major complications of ECMO.
- Comprehensively assess the ECMO patient with appropriate monitoring techniques.

ECMO INTRODUCTION ECMO INDICATIONS AND CONTRAINDICATIONS

LET'S START WITH A CASE....

39 F two weeks post partum, G2P2 presents to an outside ED with 3 days of worsening malaise, SOB, rhinorrhea, non-productive cough

- ED work up including chest xray was unremarkable
- She was discharged home with URI diagnosis and symptomatic treatment
- She returned to the ED the next day.....

Past Med/Surg History: Unremarkable. G2P2, 2 weeks post parturn term vag delivery, healthy female, uncomplicated, breast feeding

Social History: Flight attendant, off on maternity leave, (-) ETOH/illicit drugs/tobacco

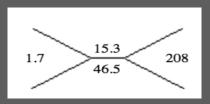
Medications: Fe supplement and prenatal MV

Allergies: None

T 36.6 °C HR140 RR29 BP104/80 Sat 92 % BIPAP 12/5 100% BMI 27.4

- Gen: WDWN, severe distress, speaks in 1-2 word sentences
- HEENT: MMM, using accessory muscles (-) JVD
- CVS: tachycardic no murmur
- RESP: B rhonchi, R>L with decreased BS RLL
- ABD: soft, NTND
- **EXT:** (-)edema, rash; wwp, brisk cap refill

R Radial	R Brachial	A-Line Draw	A-Line Draw
7.25 *L	7.21 *L	7.20 *L	7.16 *L
47 H	50 H	69 *H	68 *H
59 L	55 L	64	65
20.2	19.7	26.1	23.7
88	82	90	89
-7.0	-8.4	-3.3	-6.1
0.3		0.3	
Ventilator	Ventilator	Ventilator	Ventilator
A/C	A/C	A/C	A/C
16	20	25	25
100.0	100.0	100.0	100.0
400	400	400	400
12	14	14	14
35064	35064	35064	35064



		. ,
138	103	11 123
4.1	21	1.07
· '		`

Lactate: 1.5

Trop: 0 AST: 68 ALT: 140 Alk Phos: 99

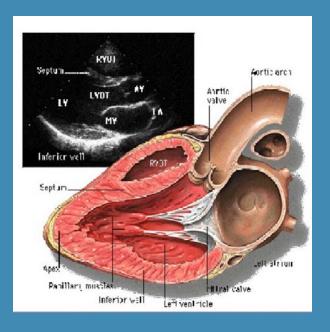
T.Bili: .5

D.Bili: .1

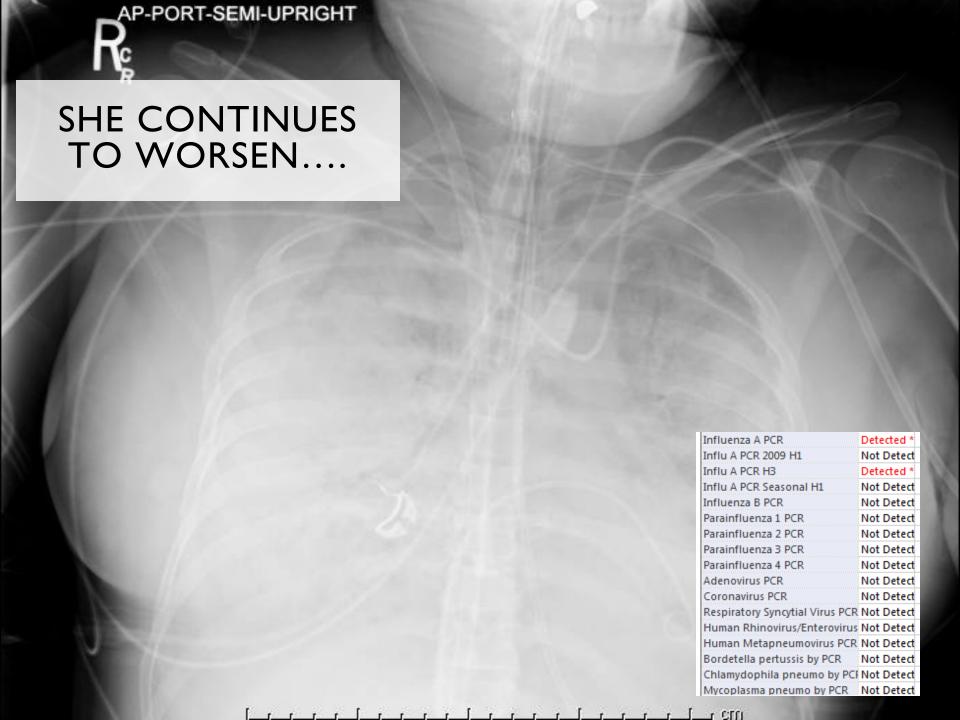
Alb: 3.9

INR: 1.3

ON 2/9 SHE DEVELOPED CARDIOGENIC SHOCK......



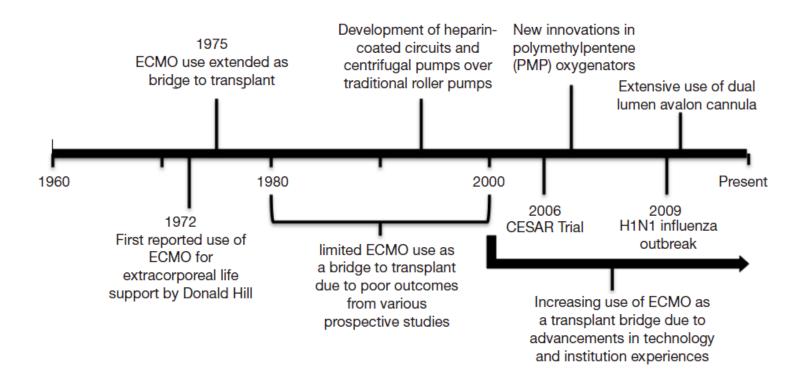




WHAT IS ECMO?

Extracorporeal membrane oxygenation (ECMO) is a *temporary* mechanical support system used to aid heart and lung function in patients with severe respiratory and/or cardiac failure

Essentially creates a dual circulation **ECMO** circuit and Native circulation to meet the patients oxygenation and perfusion needs



HISTORY OF ECMO

1975 – Robert Bartlett MD First successful use of ECMO in neonatal patient w/ severe respiratory distress, UC Irvine





Esperanza "Hope", Age I Day, 1975

Esperanza, Age 21, w/ Dr. Bartlett

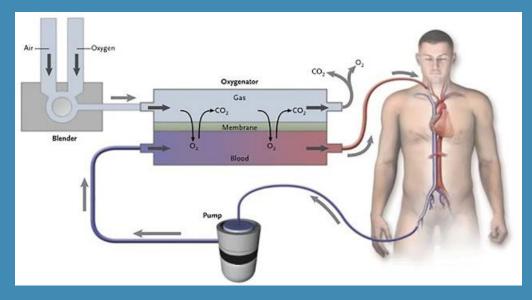
1971 – J Donald Hill MD and Maury Bramson BME. First successful ECLS Patient; Survived after trauma and ARDS – "ECMO" x 75hrs; Santa Barbara, CA



PRINCIPAL GOAL OF ECMO

Siphon off as much venous circulation as possible, pump it through an artificial oxygenator where CO₂ is removed and O₂ is added, then return it back to either venous or arterial systems

(VV or VA)



Zwischenberger JB, Bartlett RH. Extracorporeal life support: an overview. In: ECMO Extracorporeal Cardiopulmonary Support in Critical Care. 3rd ed. Ann Arbor, MI: Extracorporeal Life Support Organization; 2005:1-4

TYPES OF ECMO

Veno-venous (VV)

 For lung support when oxygenation or CO2 removal are inadequate despite both traditional and rescue therapies

Veno-arterial (VA)

- For advanced heart failure w/ bridge to transplant or long term VAD
- Rescue therapy for cardiogenic shock and impending end organ failure
- Inability to separate from cardiopulmonary bypass
- Other: Triple cannulation strategies (VV, VPa, VA, VVA, VAV, VAPa), LVAD/ECMO, Impella/ECMO., ECPR.



INDICATIONS FOR ECMO

- SEVERE but reversible respiratory failure (Murray Score)
 - Hypoxemic respiratory failure with PaO2 to/ FIO2 <100mmHg
 - Hypercapnic respiratory failure arterial pH <7.20
- Refractory cardiogenic shock
- Cardiac arrest
- Failure to wean from cardiopulmonary bypass
- Bridge to long term VAD with possibility of transplant (BiVAD)

TABLE 2. Murray score¹⁸

Variable	Score					
	0	1	2	3	4	
PaO ₂ /FiO ₂ (on 100% oxygen) in mm Hg	≥300	225-299	175-224	100-174	<100	
CXR (quadrant)	Normal	1	2	3	4	
PEEP (cm H ₂ O)	≤5	6-8	9-11	12-14	≥15	
Compliance (mL/cm H ₂ O)	≥80	60-79	40-59	20-39	≤19	

Abbreviations: CXR = chest X-ray; FiO_2 = fraction of inspired oxygen; PaO_2 = partial pressure of oxygen; PEEP = positive end-expiratory pressure

MURRAY SCORE

MURRAY SCORES > 2 OR P/F RATIOS < 15 MMHG SHOULD BE CONSIDERED FOR TRANSFER TO AN ECMO CENTER



R Radial	R Brachial	A-Line Draw	A-Line Draw
7.25 *L	7.21 *L	7.20 *L	7.16 *L
47 H	50 H	69 *H	68 *H
59 L	55 L	64	65
20.2	19.7	26.1	23.7
88	82	90	89
-7.0	-8.4	-3.3	-6.1
0.3		0.3	
Ventilator	Ventilator	Ventilator	Ventilator

For our patient:

- P/F ratio is 89 (PaO2 89/100% FiO2 or 1), so 4 points
- All 4 quadrants of the lung are involved, so 4 points
- PEEP was set at 18 cmH₂O, > 15 cmH₂O is 4 points
- Driving pressure (compliance) is 400 ml/18 PEEP = 22 ml/cmH₂O is 3 points

MURRAY SCORE = 3.8/4

ELSO Indications



- 1. Hypoxic respiratory failure
 - a. Consider if P:F < 150 (> 90% FiO2) +/- Murray score2-3 (50% mortality risk)
 - b. Indicated if P:F < 100 (> 90% FiO2) +/- Murray score 3-4 despite >6 hours optimal care (80% mortality)
- 2. CO2 retention despite high Pplat (>30 cm H2O)
- 3. Severe air leak syndromes
- 4. Need for intubation in a patient on lung transplant list
- 5. Immediate cardiac/respiratory collapse (PE, blocked airway, unresponsive to optimal care)

INDICATIONS FOR VA ECMO

Cardiogenic shock/Severe Cardiac Failure due to almost any cause:

ACS

Cardiac arrhythmic storm

Sepsis w/ profound cardiac depression

Drug/Tox-mediated profound cardiac depression

Myocarditis

Pulmonary Embolism

Cardiac Trauma

Acute Anaphylaxis

Chronic Cardiomyopathy:

Bridge to long-term VAD

Bridge to transplant

Bridge to decision

Peri-procedural

Post Cardiotomy

Inability to wean from cardiopulmonary bypass after cardiac surgery

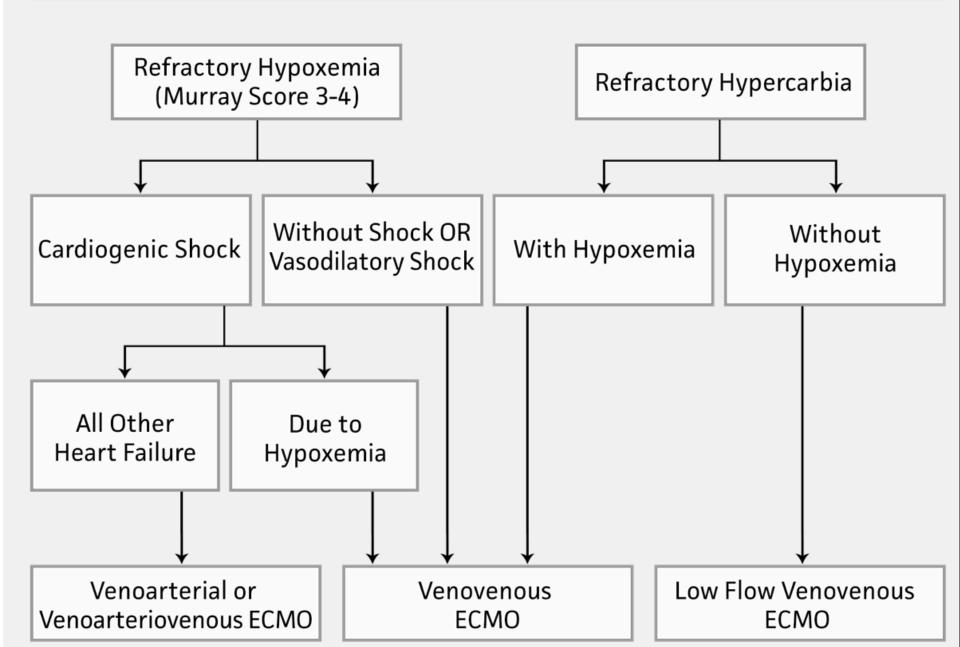
Post Heart Transplant

Primary graft failure

Peri-procedural Support

High risk percutaneous cardiac interventions

Extracorporeal Life Support Srategies for Patients with Refractory Hypoxemia or Hypercarbia



ADDITIONAL ISSUES TO CONSIDER

- Is the patient either TOO sick or not sick enough for ECMO?
- Is the current management optimized?
- What are the benefits? Bridge to ???
- What are the risks and drawbacks?
- Special considerations:
 - Patient/family consent
 - Patient wishes
 - Ethical considerations for weaning
 - Functionality
 - Need for transport (and added risks?)

Table 1. Indications and Contraindications for ECMO in Severe Cases of ARDS.*

Indications

Severe hypoxemia (e.g., ratio of Pao_2 to Fio_2 <80, despite the application of high levels of PEEP [typically 15–20 cm of water]) for at least 6 hr in patients with potentially reversible respiratory failure;

Uncompensated hypercapnia with acidemia (pH <7.15) despite the best accepted standard of care for management with a ventilator

Excessively high end-inspiratory plateau pressure (>35-45 cm of water, according to the patient's body size) despite the best accepted standard of care for management with a ventilator

Relative contraindications

High-pressure ventilation (end-inspiratory plateau pressure >30 cm of water) for >7 days

High F_{10_2} requirements (>0.8) for >7 days

Limited vascular access

Any condition or organ dysfunction that would limit the likelihood of overall benefit from ECMO, such as severe, irreversible brain injury or untreatable metastatic cancer

Absolute contraindication

Any condition that precludes the use of anticoagulation therapy:

CONTRAINDICATIONS TO ECMO

ELSO Contraindications



- No absolute contraindications
- Conditions associated with poor outcome include:
 - Mechanical ventilation at high settings (FiO2 > .9, P-plat > 30) for 7 days or more
 - Major pharmacologic immunosuppression (absolute neutrophil count <400/mm3)
 - CNS hemorrhage that is recent or expanding
 - Non recoverable co-morbidity (major CNS damage/terminal malignancy)
 - Increasing risk with age

CONTRAINDICATIONS TO ECMO

PREDICTORS OF POOR OUTCOMES

- Advanced patient age (> 65 years)
- Length of pre-ECMO mechanical ventilation
- Diagnosis
- Complications while on ECMO

EVIDENCE BASED APPROACH TO ECMO

VA-ECMO VS. CONVENTIONAL MECHANICAL VENTILATION IN SEVERE ARDS

90 patients randomized, stopped for futility.

Extracorporeal Membrane Oxygenation in Severe Acute Respiratory Failure

A Randomized Prospective Study

Warren M. Zapol, MD; Michael T. Snider, MD, PhD; J. Donald Hill, MD; Robert J. Fallat, MD; Robert H. Bartlett, MD; L. Henry Edmunds, MD; Alan H. Morris, MD; E. Converse Peirce II, MD; Arthur N. Thomas, MD; Herbert J. Proctor, MD; Philip A. Drinker, PhD; Philip C. Pratt, MD; Anna Bagniewski, MA; Rupert G. Miller, Jr, PhD

• Nine medical centers collaborated in a prospective randomized study to evaluate prolonged extracorporeal membrane oxygenation (ECMO) as a therapy for severe acute respiratory failure (ARF). Ninety adult patients were selected by common criteria of arterial hypoxemia and treated with either conventional mechanical ventilation (48 patients) or mechanical ventilation supplemented with partial venoarterial bypass (42 patients). Four patients in each group survived. The majority of patients suffered acute bacterial or viral pneumonia (57%). All nine patients with pulmonary embolism and six patients with posttraumatic acute respiratory failure died. The majority of patients died of progressive reduction of transpulmonary gas exchange and decreased compliance due to diffuse pulmonary inflammation, necrosis, and fibrosis. We conclude that ECMO can support respiratory gas exchange but did not increase the probability of long-term survival in patients with severe

(JAMA 242:2193-2196, 1979)

was launched to determine how useful it was. We describe a randomized, prospective, and collaborative study of the effect of several days of bypass with a membrane artificial lung on the chances that adults will survive severe ARF.

When this study was conceived, membrane oxygenators had been used for long-term bypass of normal animals'; the results were promising. By 1974, 150 patients suffering from ARF of varying causes and severity had undergone bypass; approximately 10% to 15% had survived. For some, the incidence of survival was even

Zapol, Warren M., et al. "Extracorporeal membrane oxygenation in severe acute respiratory failure: a randomized prospective study." *Jama* 242.20 (1979): 2193-2196.

ECMO VS. CONVENTIONAL MV IN SEVERE ARDS

Randomized Clinical Trial of Pressure-controlled Inverse Ratio Ventilation and Extracorporeal CO₂ Removal for Adult Respiratory Distress Syndrome

ALAN H. MORRIS, C. JANE WALLACE, RONALD L. MENLOVET, TERRY P. CLEMMER, JAMES F. ORME, JR., LINDELL K. WEAVER, NATHAN C. DEAN, FRANK THOMAS, THOMAS D. EAST, NATHAN L. PACE, MARY R. SUCHYTA, EDUARDO BECK, MICHELA BOMBINO, DEAN F. SITTIG, STEPHAN BÖHM, BARBARA HOFFMANN, HAYO BECKS, SAMUEL BUTLER, JAMES PEARL, and BRAD RASMUSSON

Pulmonary and Critical Care Division, Department of Medicine, and the Statistical Data Center, LDS Hospital, and the Pulmonary and Critical Care Division and the Division of Cardiology, Department of Medicine, the Division of Epidemiology and Biostatistics, Department of Family and Preventative Medicine, and the Department of Anesthesiology, University of Utah School of Medicine, Salt Lake City, Utah

The impact of a new therapy that includes pressure-controlled inverse ratio ventilation to poreal CO $_2$ removal on the survival of patients with severe ARDS was evaluated in a ran clinical trial. Computerized protocols generated around-the-clock instructions for man oxygenation to assure equivalent intensity of care for patients randomized to the nev those randomized to the control, mechanical ventilation limb. We randomized 40 patients who met the ECMO entry criteria. The main outcome measure was survival at 30 days a Survival was not significantly different in the 19 mechanical ventilation (42%) and 21 nev poreal) (33%) patients (p = 0.8). All deaths occurred within 30 days of randomization. vival was 38% (15 of 40) and was about four times that expected from historical data (p =

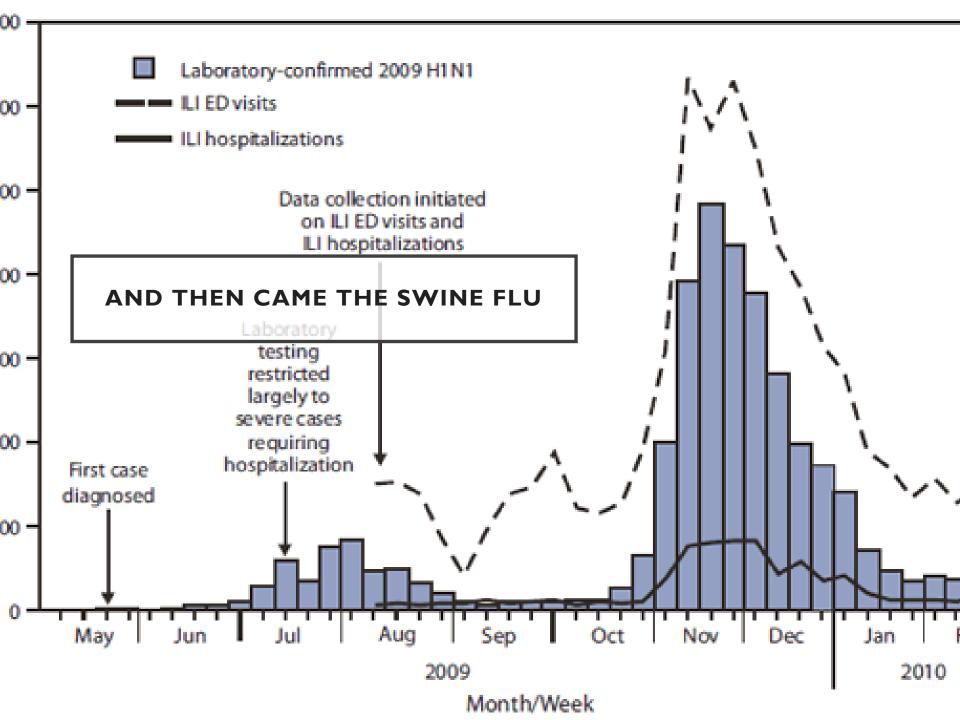
poreal treatment group survival was not significantly different from other published survival rates after extracorporeal CO₂ removal. Mechanical ventilation patient group survival was significantly higher than the 12% derived from published data (p = 0.0001). Protocols controlled care 86% of the time. Average PaO₂ was 59 mm Hg in both treatment groups. Intensity of care required to maintain arterial oxygenation was similar in both groups (2.6 and 2.6 PEEP changes/day; 4.3 and 5.0 FiO₂ changes/day). We conclude

that there was no significant difference in survival between the mechanical ventilation and the extracorporeal CO₂ removal groups. We do not recommend extracorporeal support as a therapy for ARDS. Extracorporeal support for ARDS should be restricted to controlled clinical trials. **Morris AH, Wallace CJ,**

Menlove RL, Clemmer TP, Orme JF Jr, Weaver LK, Dean NC, Thomas F, East TD, Pace NL, Suchyta MR, Beck E, Bombino M, Sittig DF, Böhm S, Hoffmann B, Becks H, Butler S, Pearl J, Rasmusson B. Randomized clinical trial of pressure-controlled inverse ratio ventilation and extracorporeal CO₂ removal for Adult Respiratory Distress Syndrome. Am J Respir Crit Care Med 1994; 149:295–305.

40 patients, no difference in survival

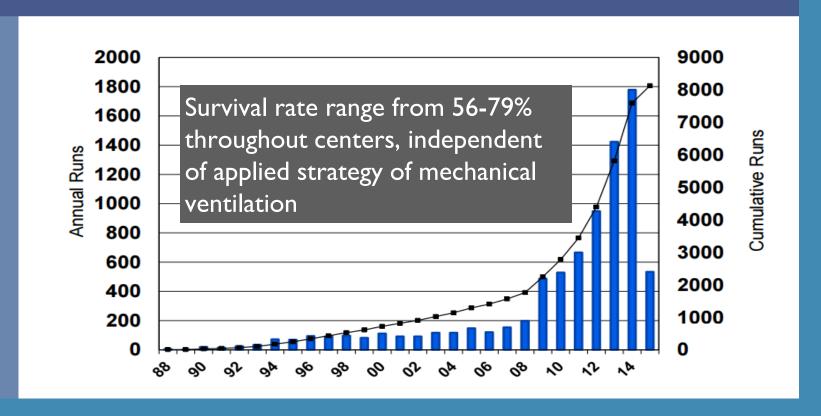
Morris, A. H., et al. "Randomized clinical trial of pressure-controlled inverse ratio ventilation and extracorporeal CO2 removal for adult respiratory distress syndrome." *American journal of respiratory and critical care medicine* 149.2 (1994): 295-305.



ECMO IN HINI INFLUENZA ASSOCIATED ARDS

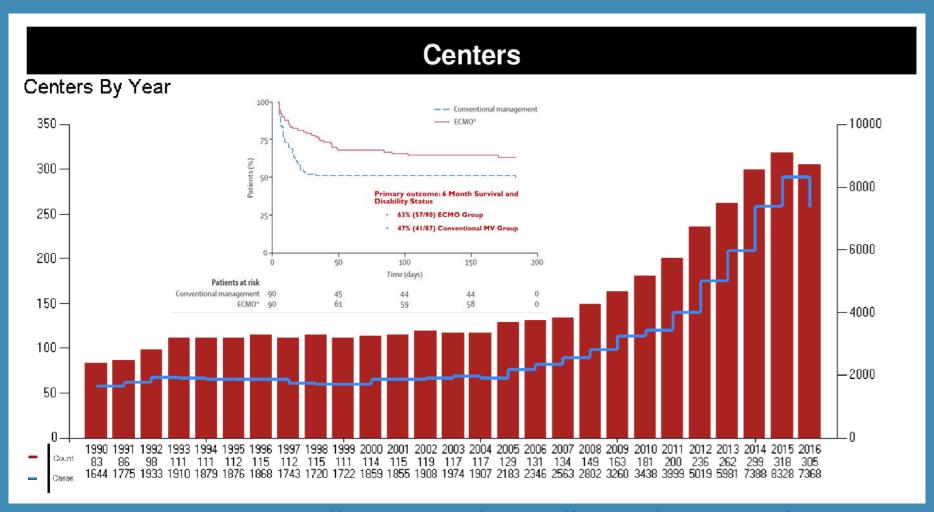


Registry Trends: Adult Respiratory Failure Cases Rising!



Davies, Andrew, et al. "Extracorporeal membrane oxygenation for 2009 influenza A (HINI) acute respiratory distress syndrome." JAMA: the journal of the American Medical Association 302.17 (2009): 1888-1895.

SURVIVAL AFTER ECMO



https://www.elso.org/Registry/Statistics/InternationalSummary.aspx



EFFICACY AND ECONOMIC ASSESSMENT OG CONVENTIONAL VENTILATORY SUPPORT VS ECMO: THE CESAR TRIAL

	ECMO group (n=90)*	Conventional m	nagement					
Alive at 6 months or discharged alive	57 (63%)	46 (51%)						
Follow-up information available†								
Full information	52 (58%)	32 (36%)						
Incomplete information from GP or hospital	5 (6%)	8 (9%)						
Information about death and disability status only	0	3 (3%)						
Alive but no further information available	0	3 (3%)						
EQ-5D								
Follow-up information available	57 (63%)	40 (44%)						
Problems with mobility				ECMO gro	up			Conventional
None	30 (33%)	19 (21%)		(n=90)*			(n=87)	management group (n=87)
Some	26 (29%)	19 (21%)			5 1 1 11			
Confined to bed	0	2 (2%)		Mean cost	Probability of survival	′	Mean cost	Mean cost Probability of survival
Data missing	1 (1%)‡	0			to 6 month	S	S	
Problems with self care			C	672.070				
	42 (470/)	26 (29%)	Scenario 1: base case‡	£73 979	0.63		£33 435	
None	42 (47%)	20 (25%)						
None Some problems washing or dressing	42 (47%) 13 (14%)	11 (12%)	Scenario 2: QALYs gained	£57534	0.63		£36688	£36688 0.47
			Scenario 2: QALYs gained at 6 months with costs based on NHS tariffs§	£57534	0.63		£36688	130088 0-4/

ECMO SURVIVAL

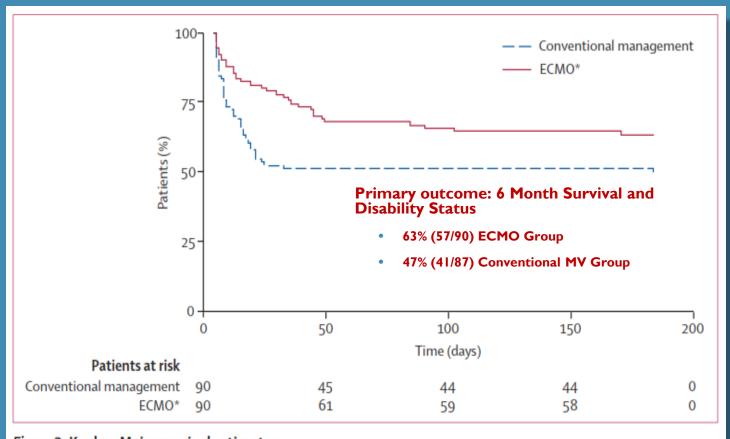


Figure 2: Kaplan-Meier survival estimates

THEN NEJM

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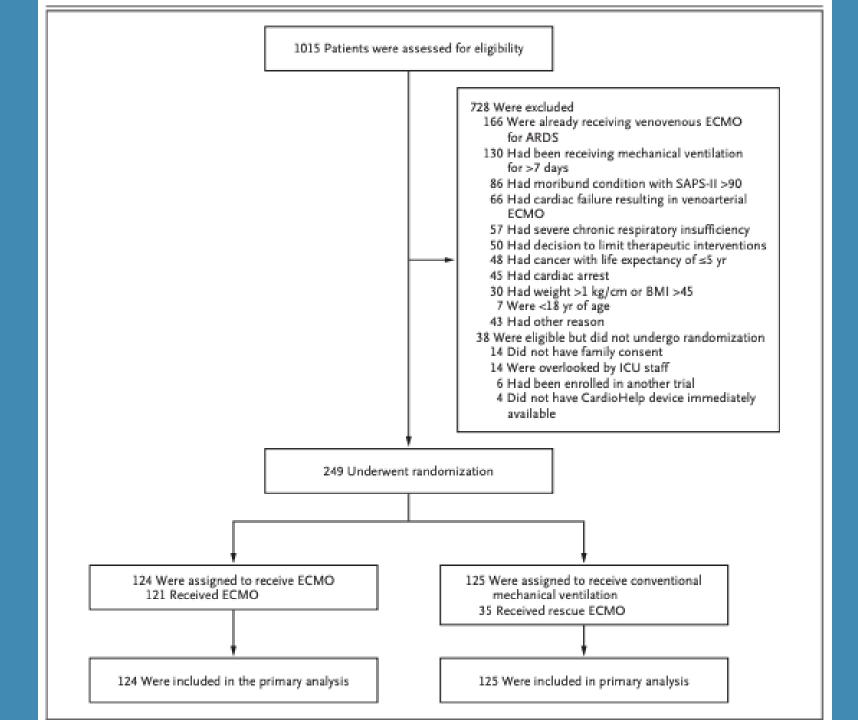
Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome

A. Combes, D. Hajage, G. Capellier, A. Demoule, S. Lavoué, C. Guervilly, D. Da Silva, L. Zafrani, P. Tirot, B. Veber, E. Maury, B. Levy, Y. Cohen, C. Richard, P. Kalfon, L. Bouadma, H. Mehdaoui, G. Beduneau, G. Lebreton, L. Brochard, N.D. Ferguson, E. Fan, A.S. Slutsky, D. Brodie, and A. Mercat, for the EOLIA Trial Group, REVA, and ECMONet*

THEN NEJM

RESULTS

At 60 days, 44 of 124 patients (35%) in the ECMO group and 57 of 125 (46%) in the control group had died (relative risk, 0.76; 95% confidence interval [CI], 0.55 to 1.04; P=0.09). Crossover to ECMO occurred a mean (±SD) of 6.5±9.7 days after randomization in 35 patients (28%) in the control group, with 20 of these patients (57%) dying. The frequency of complications did not differ significantly between groups, except that there were more bleeding events leading to transfusion in the ECMO group than in the control group (in 46% vs. 28% of patients; absolute risk difference, 18 percentage points; 95% CI, 6 to 30) as well as more cases of severe thrombocytopenia (in 27% vs. 16%; absolute risk difference, 11 percentage points; 95% CI, 0 to 21) and fewer cases of ischemic stroke (in no patients vs. 5%; absolute risk difference, -5 percentage points; 95% CI, -10 to -2).



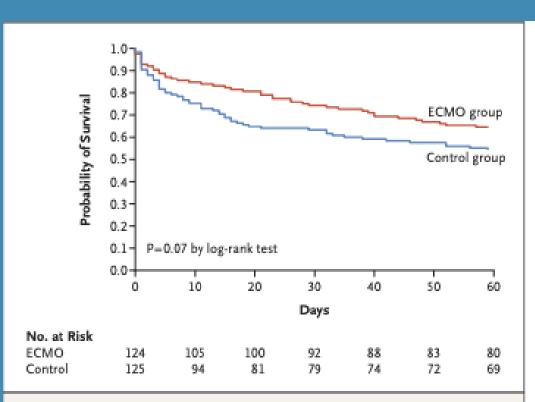


Figure 2. Kaplan-Meier Survival Estimates in the Intention-to-Treat Population during the First 60 Days of the Trial.

35 patients Medical therapy crossed over to ECMO

mean days until cross over 6.5days

20 of 35 crossover patients died on ecmo

Lesson Learned

DONT WAIT TOO LONG

CROSSOVER HIGH MORTALITY



Extracorporeal Membrane Oxygenation for 2009 Influenza A(H1N1) Acute Respiratory Distress Syndrome

Table 1. Comparison of Patients With Influenza A Who Received ECMO and Those Who Received Mechanical Ventilation But Without ECMO at ECMO Centers^a

Parameter	ECMO (n = 61)		
Age, median (IQR), y	36 (27-45)	44 (31-54)	.02
Maie sex	29 (48)	63 (47)	.54
BMI, median (IQR)	29 (23-36)	29 (24-37)	.92
Chronic lung disease	18 (30)	35 (26)	.64
APACHE III comorbidity ^b	5 (8)	30 (23)	.02
Pregnancy or postpartum	10 (16)	12 (9)	.21
Diabotos mollitus	9 (15)	23 (17)	.64
H1N1 positive	56 (92)	107 (80)	.05
At ICU admission Mechanical ventilation	53 (87)	117 (88)	.80
Vasopressor	35 (57)	46 (34)	.02
Renal replacement ther	rapy 5 (8)	9 (7)	.95
Duration or length of stay, median (IQR), d			
Mechanical ventilat	ion 18 (9-27)	8 (4-14)	.001
ICU	22 (13-32)	12 (7-18)	.001
Hospital	28 (15-43)	20 (13-31)	.07
Mortality in ICU	14 (23)	12 (9)	.01
in hospital	14 (23)	17 (13)	.06

- 4,950patientshospitalizedfor influenza252 patients
 - at ECMO centers in ICU



Referral to an Extracorporeal Membrane Oxygenation Center and Mortality Among Patients With Severe 2009 Influenza A(H1N1)

No. of Deaths/ Total No. of Patients (%)				
	ECMO-Referred	Non-ECMO-Referred	RR (95% CI)	P Value
Matching method Propensity score	18/75 (24.0)	35/75 (46.7)	0.51 (0.31-0.84)	.008
GenMatch	18/75 (24.0)	38/75 (50.7)	0.47 (0.31-0.72)	.001
Individual	14/59 (23.7)	31/59 (52.5)	0.45 (0.26-0.79)	.006



THE CHEER TRIAL

Table 2.
Cardiac arrest and treatment details.

Arrest characteristics	All N = 26	Survivors N = 14	Non-survivors N = 12	P value
ECMO inserted, n (%)	24 (92)	12 (86)	12 (100)	0.41
Median time from ECPR team arrival to initiation of ECMO, min (IQR)	20 (15– 30)	16 (15–19)	30 (24–35)	0.01
Median time from collapse to initiation ECMO, min (IQR)	56 (40– 85)	40 (27–57)	78 (48–101)	0.02
Location of ECMO (n = 24)				
Emergency Department, n (%)	13 (50)	6 (43)	7 (58)	
Intensive Care Unit, n (%)	7 (27)	3 (21)	4 (33)	0.40
Coronary catheterization laboratory, n (%)	3 (12)	2 (14)	1 (8)	
Hospital ward, n (%)	1 (4)	1 (7)	0 (0)	
ST elevation on initial ECG, n (%)	9 (35)	5 (36)	4 (33)	0.75
Initial post arrest laboratory values				
pH	6.9 (6.7– 7.1)	7.0 (6.8– 7.1)	6.8 (6.7–7.0)	0.02

Photo: E-CPR Equipment

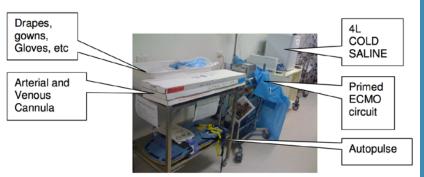


Table 3.
Outcomes and complications.

		Survivors	Non-survivors	P
Outcomes	All $N = 26$	N = 14	N = 12	value
Survival to hospital discharge, n (%)	14 (54)			
CPC 1-2, n (%)	14 (54)	14 (100)		
ROSC, n (%)	25 (96)	14 (100)	11(92)	0.27
Wean off ECMO ^a	13/24 (54)	12/12 (100)	1 (7)	0.01
Median time on ECMO, days (IQR)	2 (1-5)	3 (1.8-5)	1 (1-5)	0.32
Median time in ICU, h (IQR)	134 (39– 291)	230 (118–320)	30 (4–134)	0.01
Median hospital length of stay, days (IQR)	13 (1.3–22)	20 (12–26)	1 (1–8)	<0.01
Bleeding, n (%)	18 (70)	10 (71)	8 (67)	0.79
Renal replacement therapy, n (%)	10 (39)	4 (29)	6 (50)	0.29
Peripheral vascular issues, n (%)	10 (39)	5 (36)	5 (42)	0.75
Stroke, n (%)	6 (23)	2 (14)	4 (33)	0.25

 ${\tt ECMO-extra}\ corporeal\ membrane\ oxygenation,\ CPC-cerebral\ performance\ category,\ ROSC-return\ of\ spontaneous\ circulation.$

a From patients requiring ECMO.

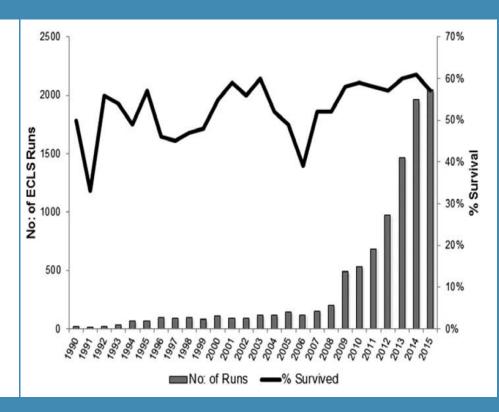


ECLS 2016 DATA

Table 1. ECLS Cases and Survival to Discharge

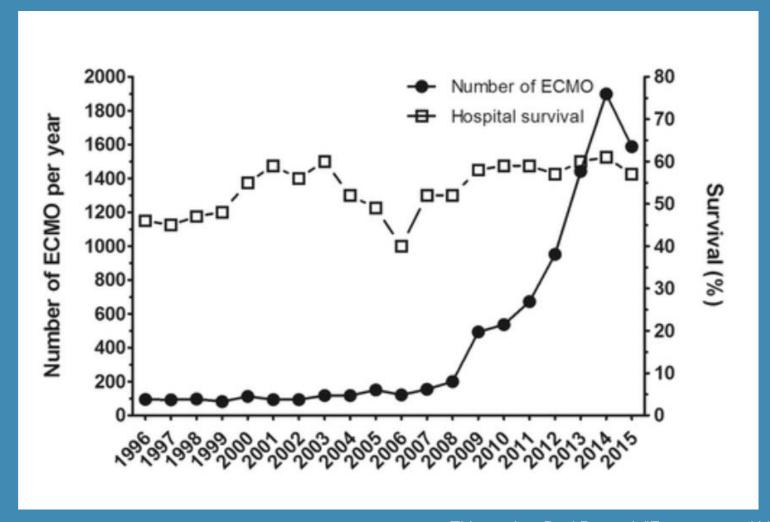
			9
	No. Cases	Survived ECLS, N (%)	Discharged, N (%)
Neonatal			
Respiratory	29,153	24,488 (84)	21, 545 (74)
Cardiac	6,475	4,028 (62)	2,695 (42)
ECPR	1,336	859 (64)	547 (41)
Pediatric			. ,
Respiratory	7,552	5,036 (67)	4,371 (58)
Cardiac	8,374	5,594 (67)	4,265 (51)
ECPR	2,996	1,645 (55)	1,232 (41)
Adult			
Respiratory	10,601	6,997 (66)	6,121 (58)
Cardiac	9.025	5,082 (56)	3,721 (41)
ECPR	2,885	1,137 (39)	848 (29)
Total	78,397	54,866 (70)	45,345 (58)

ECLS, extracorporeal life support; ECPR, ECLS to support cardiopulmonary resuscitation.





ECLS 2016 DATA





SURVIVAL IN PROLONGED ECMO

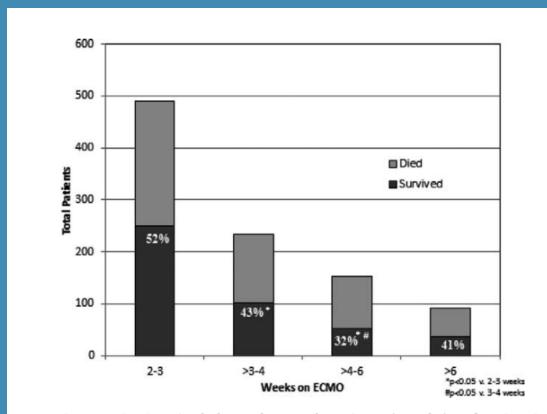


FIGURE 1. Survival based on duration (weeks) of ECMO (% = percent survived per week(s) range). The Fisher exact test was used for the pairwise comparisons.

Posluszny, Joseph, et al. "Outcome of adult respiratory failure patients receiving prolonged (≥ 14 days) ECMO." *Annals of surgery* 263.3 (2016): 573-581.



SURVIVAL IN PROLONGED ECMO

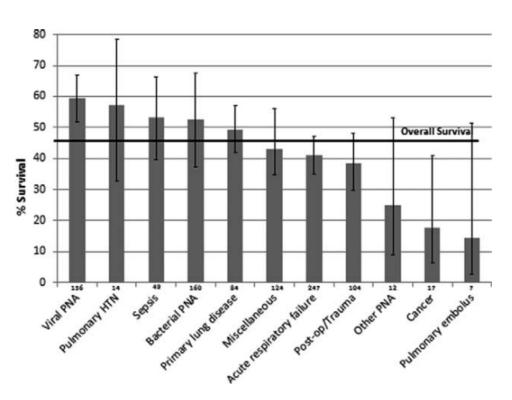
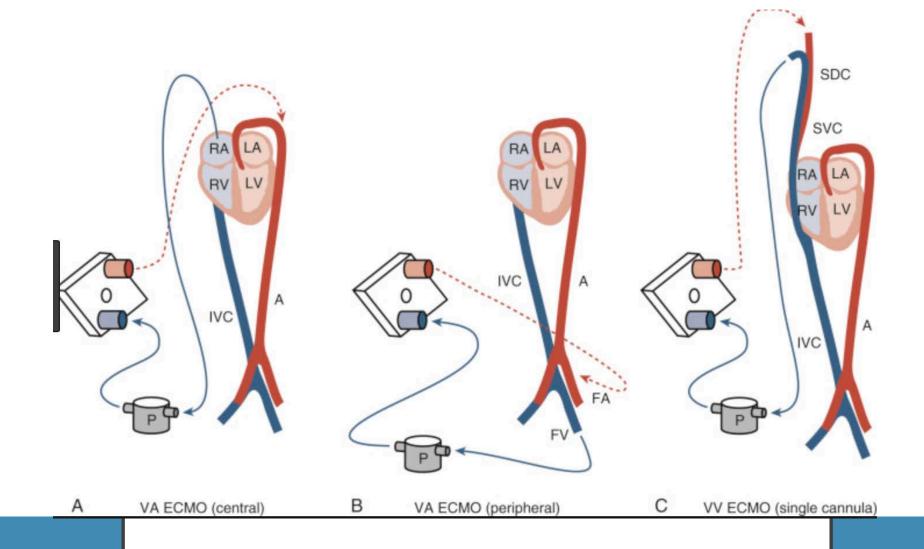


FIGURE 5. Survival based on primary diagnosis. The horizontal line indicates overall survival (45%). The number patients with the corresponding diagnosis are listed below each bar.

PREPARING TO INITIATE ECMO

THE ECMO CIRCUIT

- Pumps
- Membrane oxygenators
- Tubing
- Circuit pressures
- Monitoring systems
- Other



THE ECMO CIRCUIT

THE PUMP HEAD

- Initial pumps were roller or peristaltic type
 - Preload dependent
 - Depended on gravity drainage
- Afterload independent
 - Prone to high pressures
 - Risk of occlusion is circuit rupture
- Flow = stroke volume x heart rate
- Linear increase in output with increased RPM





ROLLER OR PERISTALTIC PUMP



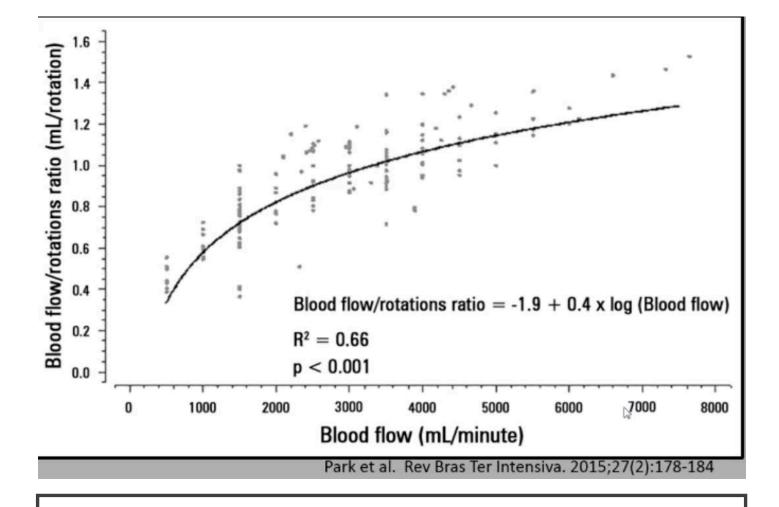
- Diameter of tubing and distance traveled → stroke volume x rpms = CO
- Afterload independent
 - Can clamp arterial line and pump keeps turning
 - May lead to rupture



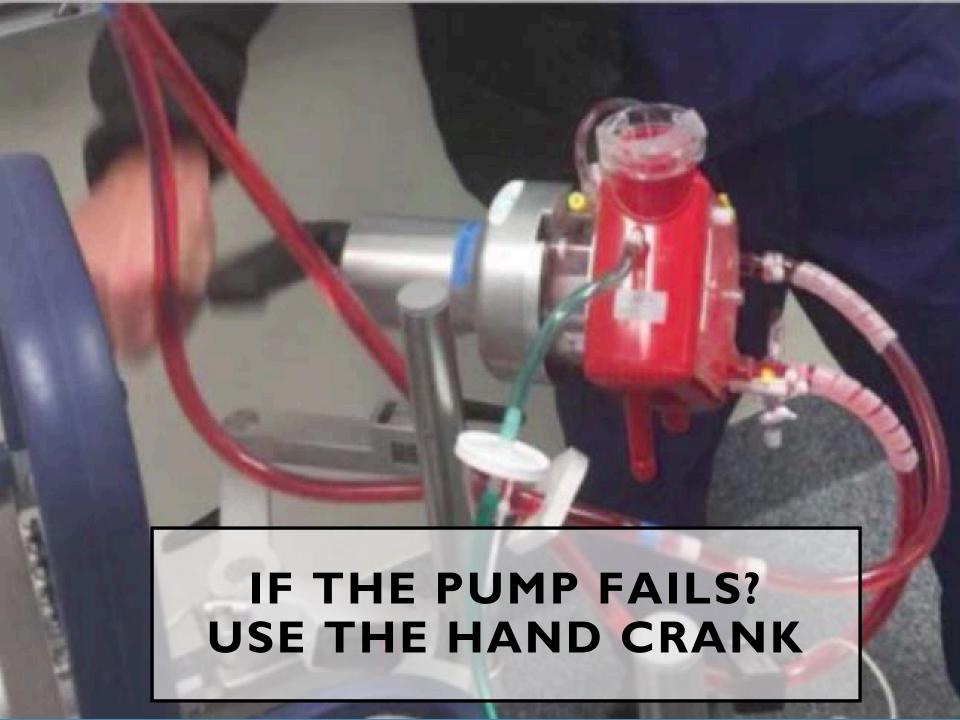
CENTRIFUGAL "MODERN" PUMPS



- Preload dependent
 - Generate negative pressure
- Afterload sensitive
 - Pump output decreases with resistance
 - No circuit rupture
- Non-occlusive
 - Retrograde flow can occur
- Magnetically coupled to the motor (vs levitated)
- Non linear relationship with RPMs

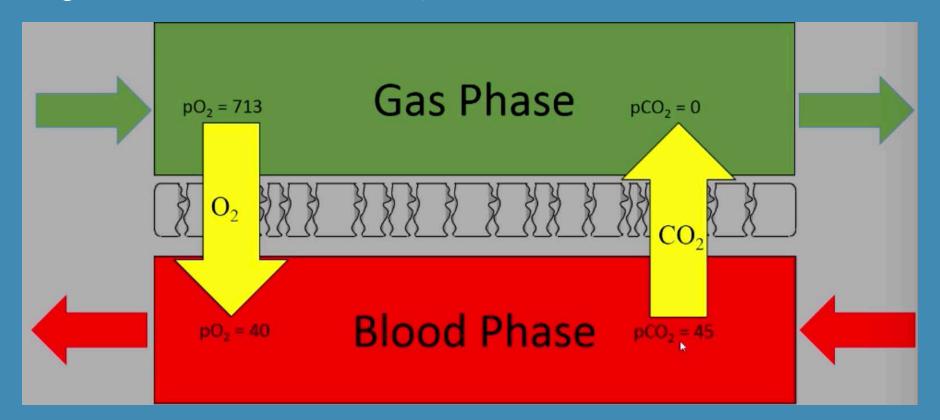


NON LINEAR RELATIONSHIP BETWEEN RPMS AND FLOWS



MEMBRANE OXYGENATOR

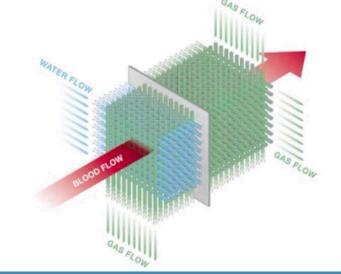
 Need something physically to separate the blood from the gas. This diminished hemolysis



MEMBRANE OXYGENATOR







http://maryland.ccproject.com/2016/12/03/ecmo-boot-camp-day-1/

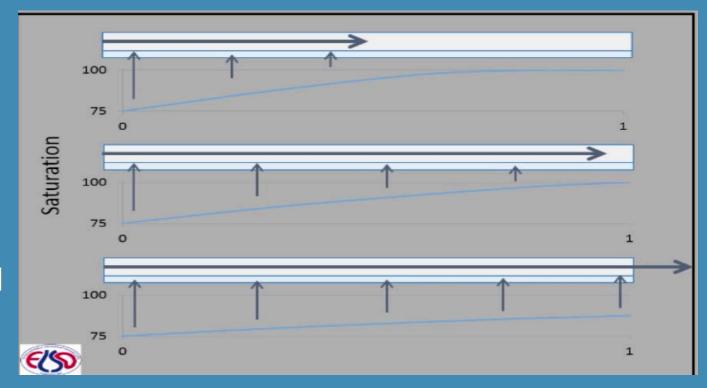
POLYMETHYLPENTENE (PMP) HOLLOW FIBER OXYGENATORS

- Low resistance
- Biocompatible
- More efficient gas exchange
- Smaller pores = less plasma leakage than older oxygenators
- Nanoporous membrane



RATED FLOW

- Maximum flow rate in which saturation will increase from 75% to 95% (with Hb 12%)
- Quadrox-I rated flow is 7 liters/minute



TUBING

- 3/8 diameter
- Proprietary tubing of unclear value
 - Heparin
 - Biocompatible
- Protein layer develops rapidly



OXYGEN BLENDER AND FLOW METER

- Flowmeter
 - Adjust sweep gas flow
 - Increase sweep, decrease CO₂
- Oxygen blender
 - Adjust FiO₂
 - Don't really wean like on the vent
 - Some programs only use 100%



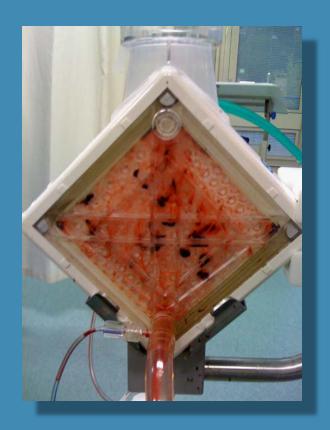
CIRCUIT PRESSURES

- With a centrifugal pump, pressures are always negative
- Determined by drainage cannula size, patient volume status, pump RPM
- Changes may reflect tamponade, tension PTX, intrathoracic pressure changes, cannula position, line kinks
- Limits ~ -90-100 mmHg
 - Excess neg pressure can cause cavitation and hemolysis
- Chatter?
 - Decrease RPMs
 - Give volume



CIRCUIT PRESSURES

- Pressure drop across the oxygenator?
 - Δ P
- Changes with flow
- Usually 20-50
- Trend not absolute number
- Resistance in membrane
 - Clots
 - Fibrin Strands



MONITORING

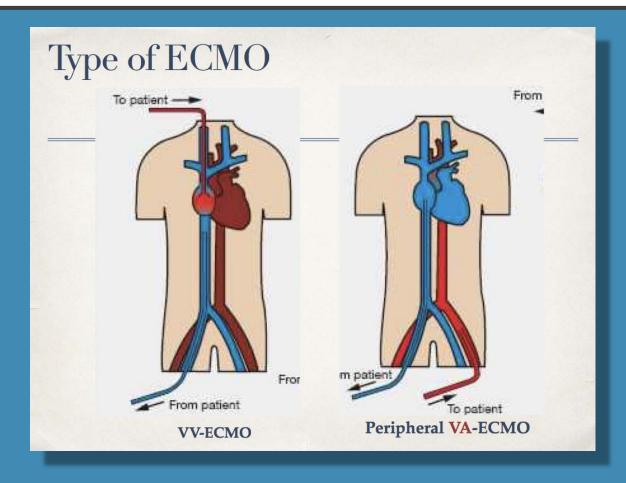
- Change in ∆ P from baseline is an early warning sign of circuit issues
- Increased ∆ P
 - Increased pre membrane, decreased post membrane
 - Obstruction in flow in membrane
- No change in ∆ P
 - Increased pre-membrane and increased post-membrane
 - Increased resistance between membrane and patient
 - Obstruction
 - Cannula size
 - Hypertension (in VA ecmo)

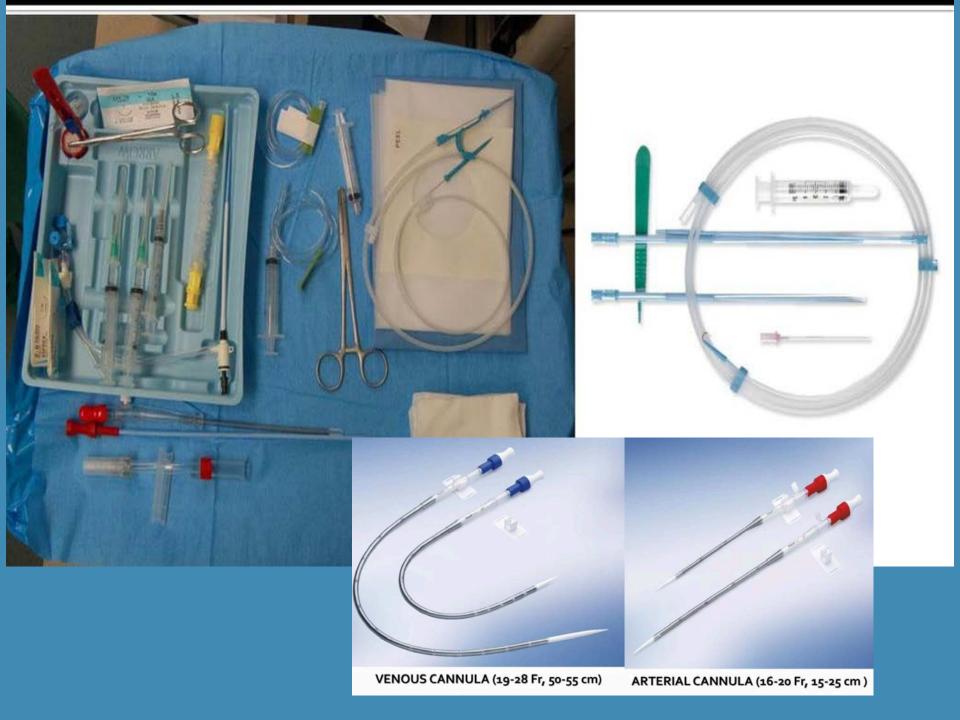
MONITORING

- Increased negative pressure
 - Hypovolemia
 - RPM too high
 - Occlusion on venous side (clot, kink)
 - Cannula malposition
- MINIMUM sustained blood flow is > 2L/min and prevents clot formation in circuit and/or patient
- MAXIMUM heater settings < 37 degrees celcius
 - Turn off heater when patient's desired temperature is reached

ECMO CANNULATION AND PHYSIOLOGY

TYPES OF ECMO



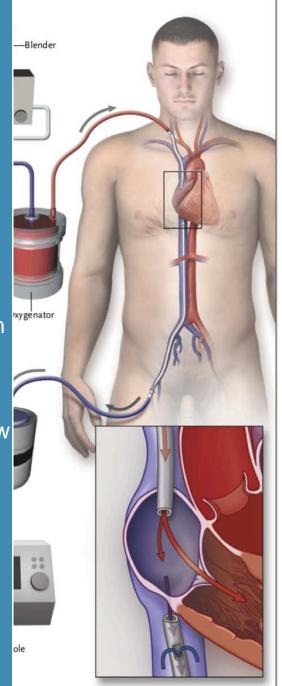


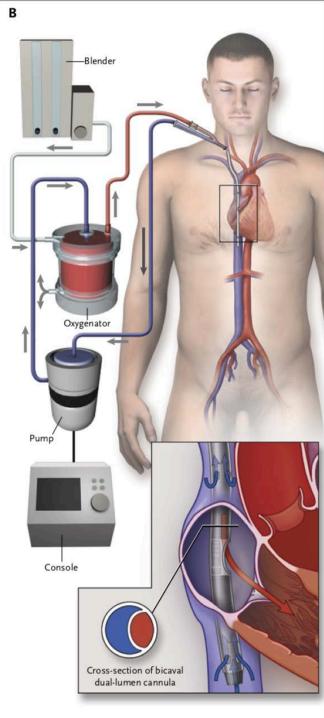
CANNULATION FOR VV ECMO

Blood is drained from and returned to the venous circulation

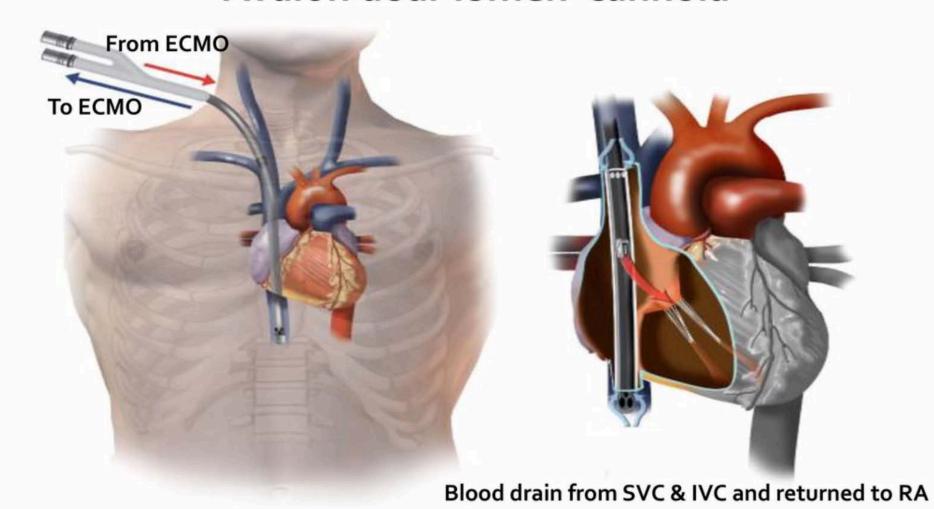
Does not provide hemodynamic support

Goal is to rest the lungs and allow time for healing or as a bridge to transplant





Avalon dual-lumen cannula



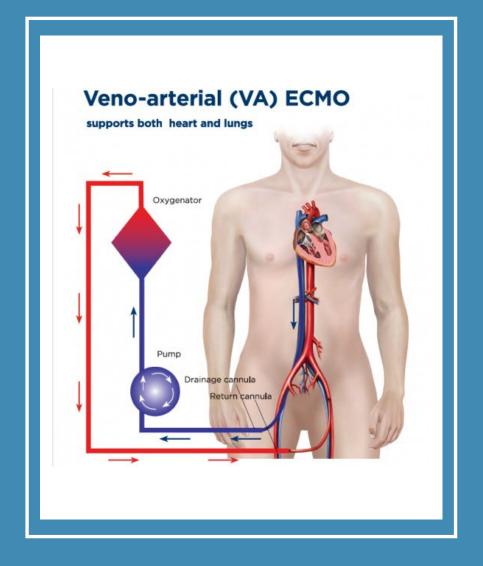
CANNULATION FOR VA ECMO

Provides hemodynamic and pulmonary support

Heart and lungs are bypassed

Venous drainage/arterial return

Nonpulsatile

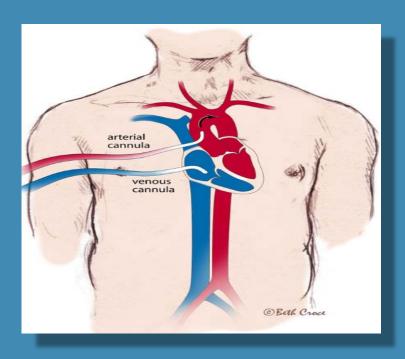


VA - CENTRAL CANNULATION

Blood extracted from IVC or R. Atrium

Blood returned to ascending aorta
Used more after cardiac surgery
Open vs. Closed

Better oxygenated antegrade flow



VA - PERIPHERAL CANNULATION

Fem $V \rightarrow$ Fem A.

Fem V – Carotid A.

Fem V → Axillary A.

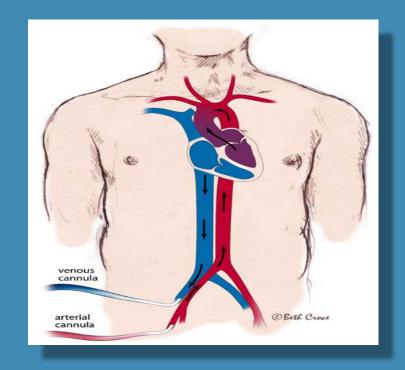
Better for emergent situations

Less invasive

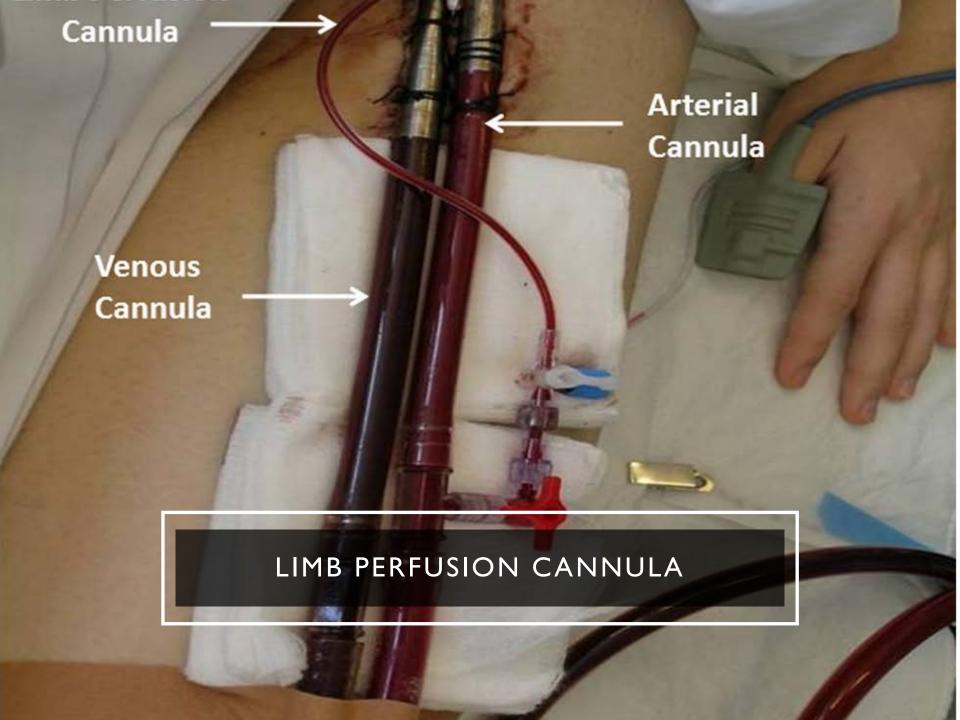
Faster insertion

Relies upon retrograde flow Admixing in aortic arch

Risk of Limb Ischemia



1. http://www.slideshare.net/oliflower/ecmo-in-nz-by-mcguinness



VENOARTERIA L (VA) ECMO CHALLENGES

Differential Hypoxia

- Risk of poor cerebral, upper extremity or R-Sided perfusion from admixing
- Monitor R. Femoral ABG vs. R. Radial ABG

Reduced oxygenated blood flow through coronary arteries

- Consider IABP to provide flow to coronaries
- Consider VAV ECMO for bad lungs

Can increase LV Preload and thereby O2 Demand

- Inotropes to maintain LV Ejection
- Placement of LV Drain

Poor perfusion to distal limb in Fem. Artery cannulation

Separate arterial perfusion cannulation to reduce ischemia risk

VENO-ARTERIAL-VENOUS (VAV) ECMO

For Differential Hypoxia

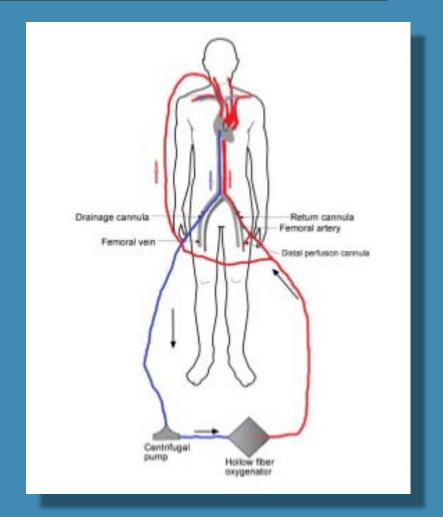
Peripheral (femoral) VA cannulation

Additional return cannula to Subclavian Vein

Perfuses venous side with oxygenated blood

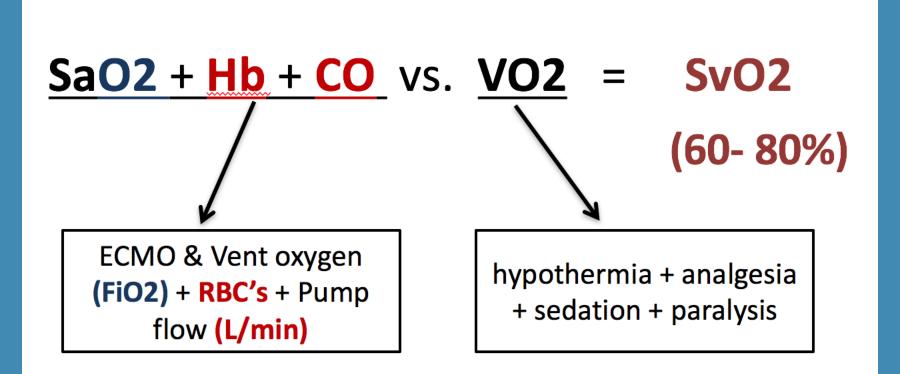
Combines VA + VV

Very limited evidence



Choi, Joon Hyouk, et al. "Application of veno-arterial-venous extracorporeal membrane oxygenation in differential hypoxia." *Multidisciplinary respiratory medicine* 9.1 (2014): 1.

Oxygen Delivery and Consumption



Oxygen Delivery and Consumption

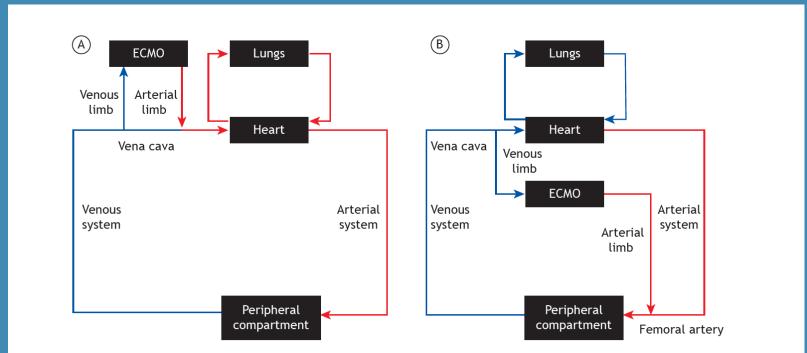


Figure 1. Basic extracorporeal membrane oxygenation (ECMO) configurations. Panel A shows the venovenous ECMO configuration, in which the extracorporeal system is in series with the lungs, providing only respiratory support. Panel B shows the venoarterial ECMO configuration, in which the extracorporeal system is in parallel with the heart and lungs, providing respiratory and cardiovascular support.

NURSING CONSIDERATIONS DURING CANNULATION

- Maintain strict infection control
- Restrict access to essential personnel
- Remove unnecessary invasive lines
- Ensure that all required invasive access are present (NG tube, core temperature probe, etc)
- Secure ET tube
- Ensure crash trolly in close proximity
- Ensure that fecal softeners are prescribed
- Prepare and position patient

IMPORTANT NURSING CONSIDERATIONS DURING CANNULATION

- Clip hair on the proposed side with an electric razor
- Position bed so that Echo machine, ECMO trolley and sterile field can be positioned
- Routine assessments:
 - HR, SaO2, SBP, MAP
- Hourly assessment:
 - Neurovascular checks, urine output, core temperature, ventilator parameters, CVP and sedation assessment

OTHER NURSING POINTS

- Only change the dressing on the cannula if significant exudates or not intact and secure
- Requires two nurses for dressing changes, preferably during the day shift
- Pull dressing off TOWARD the insertion site
- Avoid NG or other interventions that can cause bleeding once patient is initiated on ECMO

BLOOD WORK AND OTHER DIAGNOSTICS

- Current type and match
- Daily electrolytes, magnesium, LFTs, LDH and haptoglobin
- CBC twice per day
- Daily blood cultures if fever spike
- Pre and post oxygenator ABG
- ACT every 2 hours x 24 hours
- APTT every 6 hours, target 60-80 s

ECMO COMPLICATIONS

Hemorrhage

Surgical site hemorrhage

Systemic anticoagulation - Pulmonary, intrathoracic, GI, RP

Thrombosis

Systemic thrombus

Circuit thrombus – life-threatening

Hemolysis

Check plasma free Hgb levels

Thrombocytopenia

Transfuse as needed

Heparin-induced thrombocytopenia (HIT)

Use direct antithrombin agents – Argatroban, Bivalrudin



ECMO COMPLICATIONS

Neurologic

Intracranial Hemorrhage (most fatal)

Ischemia or Stroke

Seizures

Infectious

Underlying sepsis

ECMO Circuit- Foreign Body

Cardiovascular

Hypertension

Arrhythmias

Gastrointestinal

Ischemia

Hemorrhage

Metabolic

Fluid-shifting

Medication range derangements

Alteration in Kidney/Liver

Function

Mechanical

Clots in circuit

Oxygenator failure

Consumption coagulopathy

Pulmonary embolus

Systemic emboli

Makdisi, George, and I-wen Wang. "Extra Corporeal Membrane Oxygenation (ECMO) review of a lifesaving technology." *Journal of thoracic disease* 7.7 (2015): E166.

ECMO COMPLICATIONS

Venoarterial ECMO Without Routine Systemic Anticoagulation Decreases Adverse Events

Katherine L. Wood, MD, Brian Ayers, MBA, Igor Gosev, MD, Neil Kumar, MD, Amber L. Melvin, MD, Bryan Barrus, MD, Sunil Prasad, MD*



DOI: https://doi.org/10.1016/j.athoracsur.2019.08.040

Results

From May 2011 through January 2018, there were 203 eligible patients supported on VA-ECMO, 35% (75) were not anticoagulated. Overall complication rates were significantly lower for the no anticoagulation group (57% vs 76%, p=0.007) including a trend toward fewer hemorrhagic complications (53% vs 63%, p=0.178) without increased risk of thrombosis (13% vs 21%, p=0.147). The anticoagulated group required more transfusions of packed red blood cells (12.8 vs 1.09, p=0.002) and platelets (3.0 vs 1.3, p=0.009) and showed a higher incidence of HIT (8% vs 0%, p=0.015). There was no difference in overall mortality (72% vs 62%, p=0.165).

Conclusions

The absence of routine systemic anticoagulation for patients supported on VA-ECMO is not associated with higher mortality, pump failure, or thrombotic complications. Patients had a lower requirement for blood product transfusions, and there was no incidence of HIT. VA-ECMO patients without other indications for anticoagulation can be treated without systemic anticoagulation during their VA-ECMO course.

ECMO COMPLICATIONS VA - SPECIFIC

Cannulation - Related

Vessel perforation

Arterial dissection

Distal ischemia

Incorrect location

Pseudoaneurysm development

Cardiac Thrombosis

Secondary to retrograde flow and intraventricular stasis

Coronary/Cerebral Hypoxia

Differential hypoxia from admixing

ETHICAL CONSIDERATIONS

"Bridge to nowhere" for patients who are poor candidates for device placement or transplantation

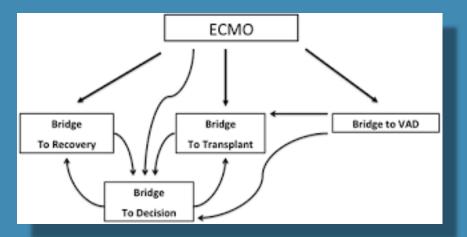
Little data on long-term complications

Cardiopulmonary recovery without neurologic recovery

Anxiety, Depression, PTSD in survivors

Prolonged ICU Lengths of Stay

Financial cost and resource utilization





https://en.wikipedia.org/wiki/Bridge_to_Nowher e_(San_Gabriel_Mountains)

ECMO WEANING

VV ECMO WEANING

Criteria to begin weaning VV ECMO

- Resolution of underlying acute lung injury and patient meets typically weaning parameters for ventilator weaning
- Weaning from V-V ECMO is performed by progressively decreasing the FGF ("Sweep") to the oxygenator. and increasing ventilator (or patient) effort to maintain adequate CO2 clearance.
- Once sweep or FGF is 0 L/min for a period of 4 to 24 hours, the patient is effectively 'off ECMO'

NOTE: Circuit blood flow does not need to be decreased to zero and should be maintained so that circuit does not clot

VA ECMO WEANING

When are VA patients ready to start weaning?

- Etiology of cardiac failure is compatible with myocardial recovery
- ECMO flows ≤2-3L/min
- Pulsatile arterial waveform present for >24h
- MAP> 60 mmHg in the absence of "highdose" inotropes/vasopressors
- Major metabolic disturbances have resolved
- Lung function is not severely impaired

VA ECMO WEANING PROCEDURE

VA Weaning trial:

- Takes about 1 hour and Echocardiographer (either TTE or TEE)
- IV heparin bolus: If not coagulopathic, then small bolus of IV heparin (1000-2,500 units to prevent clotting at low circuit blood flows
- Record echo variables inlcuiding:
 - Aortic VTI TDSa mitral annulus LVEF TAPSE
- Drop ECMO flow in 0.5l/min increments for five minutes at a time
 - Do not reduce below 1-1.5L/min
- The following hemodynamic variables are observed and recorded at each increment as well:
- MAP, HR, CVP, AP (if pulmonary arterial catheter is present) SpO₂

Successful weaning = No significant deterioration in the hemodynamic variables and improvements in cardiac function as assessed by echocardiography

UNSUCCESSFUL WEANING FROM VA ECMO?

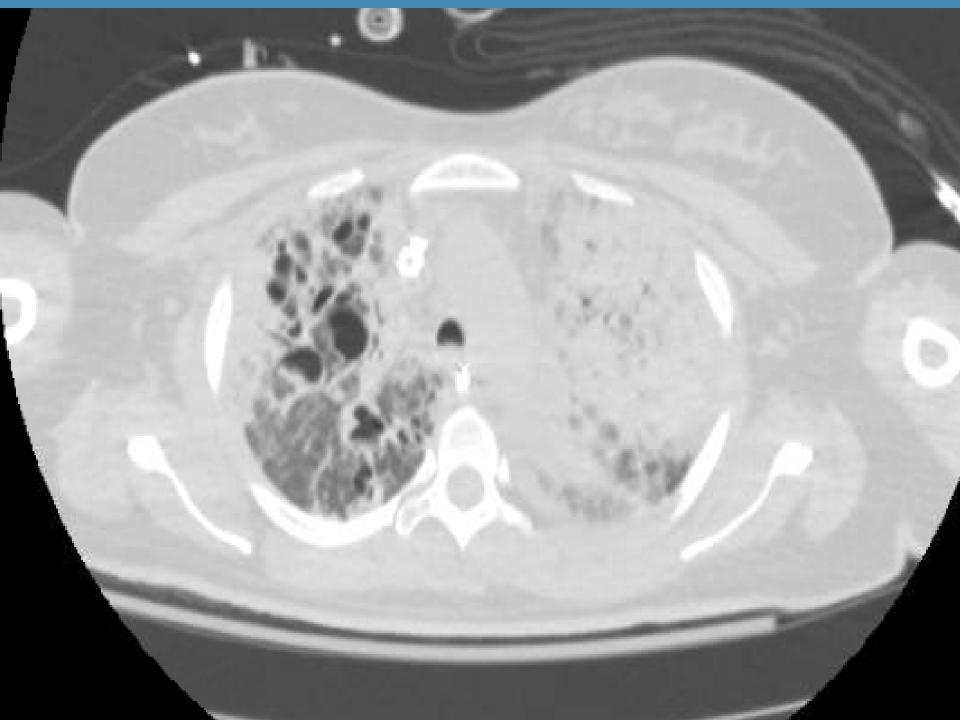
- If any hemodynamic instability or objective changes during the weaning trial...
 - Turn the ECMO circuit blood flow setting back up to where it was prior to the weaning study and reduce the ventilator support back to where it was pre-study.
 - Wait 48 hours before attempting another weaning study.

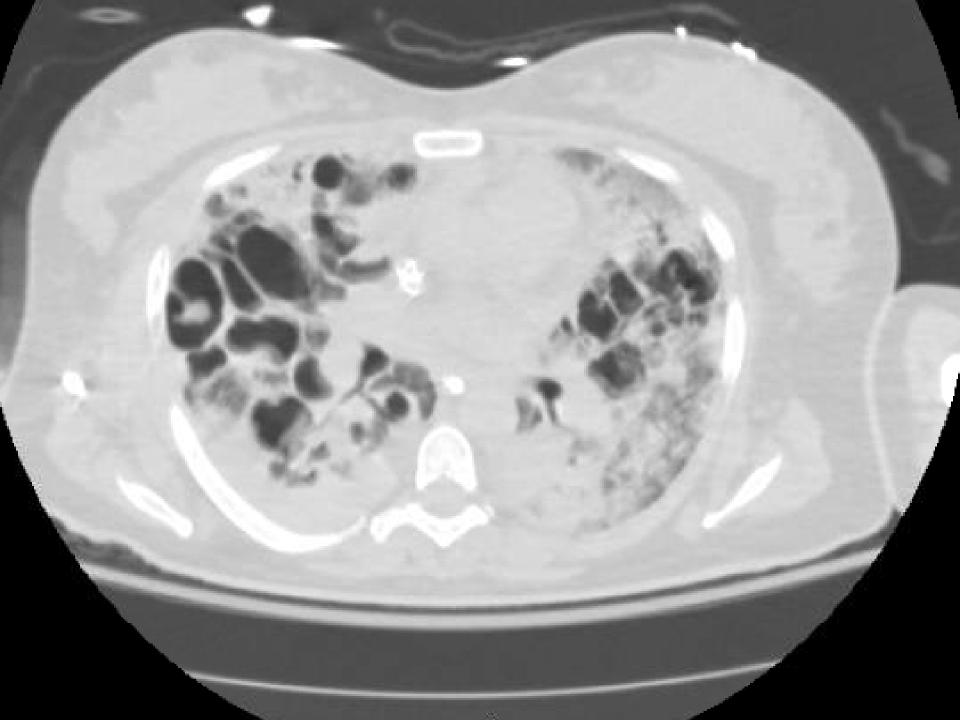
CASE REVISITED

Ecmo Day 33

- Necrotizing pneumonia with pseudomonas on BAL
 - Treated with multiple antibiotics, tamiflu, and one dose of peramivir
- Developed RLE compartment syndrome and went to OR emergently for 4 compartment fasciotomy
- Acute renal failure requiring CRRT
- Transitioned to VV ecmo
- S/p tracheostomy
- Awake, working with PT, ?
 Pulmonary recovery and ? Role for transplantation.







ECMO TROUBLESHOOTING

CASE ONE

A 47 yo M with influenza and severe ARDS is on VV ECMO.

An ABG from his right arterial aline demonstrates an PaO2 of 80%. This correlates with his SaO2 as measured by pulse oximetry on his left index finger.

You're asked to troubleshoot...

What do you address first?

Fresh gas flow (FGF) Blender Flow calibrations

Most important first step with VV ECMO is to verify adequate oxygen delivery and appropriate circuit blood flow. When ECMO is initiated, FGF and ECMO blood flow are 1:1 and are verified by ABG

FRESH GAS FLOW (FGF) AND BLENDER

Ensure that the fresh gas tubing is connected to the inlet from the fresh gas flowmeter

- FGF is titrated to the target PaCO2
- When patient is hypoxemic, must make sure FGF has not been excessively decreased by inspecting the flow meter
 - Don't confuse high and low flow sides
 - Read from the center of the ball on the flow meter.
 - With rotaflow, usually set at 100% and kept there even during weaning

FLOW CALIBRATIONS

- Turn the pump speed (rpm) to zero while the circuit is clamped
- Hold down the '0' button on the PLS or pressing the zero indicator button on the HLS
- Remember to then increase pump speed to 1000 rpm before slowly releasing the clamp over 3-5 seconds and then continuing to slowly increase the rpm until target flow is achieved
- Target blood flows are chosen to achieve adequate arterial oxygenation in the setting of lung protective ventilator strategy

NEXT STEP? CHECK FOR ACCESS INSUFFICIENCY

- If FGF, blender settings and flow calibrations are wnl, the NEXT STEP is to check for ACCESS INSUFFICIENCY
- Unstable circuit flows
- Rising negative pressures (D P) (if using the HLS system)
- "Chatter", "chugging", kicking or swinging of the venous drainage line

ACCESS INSUFFICIENCY

There is NO access insufficiency if ECMO blood flow steadily increases with increasing rpm

There IS access insufficiency if ECMO blood flow decreases once a threshold rpm is exceeded

If present, access insufficiency must be corrected before you do anything else

ACCESS INSUFFICIENCY

- Access insufficiency occurs when the suction pressure at the access cannula exceeds venous return.
- Inflow is interrupted due to partial or complete occlusion of the inlet ports of the access cannula by the walls of the collapsible vein.
- After a few seconds, ongoing venous return fills up the vein again and the cannula ports reopen to function once more.
- This cycle repeats itself resulting in unstable, fluctuating ECMO flows (shown in L/min on the ECMO console) despite a stable pump speed (rpm).

ACCESS INSUFFICIENCY

- Suspect access insufficiency if:
 - Unstable circuit flows
 - Increasingly negative pressures (if using the HLS system)
 - Hemolysis (e.g. plasma free-hemoglobin level is >0.10 g/dL or rising LDH and haptoglobin

CAUSES OF ACCESS INSUFFICIENCY

- Hypovolemia/ hemorrhage
- Poorly sited access cannula (too low)
- Excessive pump speed (rpm setting)
- Patient coughing or straining
- Positional (e.g. after turning the patient)
- Acute vasodilatation (e.g. sedation bolus)

- Increased intra-abdominal pressure
- High output cardiac failure (e.g. septic shock)
- Cardiac tamponade
 Thrombosis at cannula
 access site
- Worsening cardiac function

CASE TWO

The patient in case one is still hypoxemic after insuring FGF, blender settings, flow calibrations and in the absence of access insufficiency..

What next?

PRE OXYGENATOR BLOOD GAS TO CHECK SVO2

Need to consider the possibility of high oxygen consumption or recirculation.

- Low SvO2 (<60%) suggests HIGH OXYGEN CONSUMPTION. Response?
 - Tolerate lower SaO2
 - Target a higher hhemoglobin to increase oxygen delivery (e.g. Hb >100 g/L)
 - Improve oxygenation through the lungs (e.g. try nitric oxide or prostacyclin)
 - Consider neuromuscular blockade to decrease oxygen demand
 - Use targeted temperature management to cool the patient and decrease oxygen demand

PRE OXYGENATOR BLOOD GAS TO CHECK SVO2

- High SvO2 (>80%) suggests RECIRCULATION
 - Flow from the return cannula is entering the access cannula, rather than being distributed to the patient.
 - Check cannula positions
 - Is there displacement of the cannula at the insertion site
 - Check position of tips on x-ray
 - The tip of the access cannula should be 8 to 15cm apart to minimize recirculation.

CHECKING THE MEMBRANE OXYGENATOR

Confirm **oxygen flow** to the oxygenator:

Ensure that the cap on the oxygen inlet where oxygen tubing is connected was removed prior to connection

Feel the fresh gas flow at the inlet to the oxygenator with your hand

Check that oxygen tubing is securely connected

Check that the oxygen blender is set to 100% oxygen

Set the fresh gas flow of oxygen (aka "sweep gas") at a minimum flow rate of 2 L/min

Check for clotting:

Visually inspect for increased clot in the oxygenator using a torch

Check for rising D-dimer (suggests clotting) and plasma free hemoglobin (suggests hemolysis)

Check the **transmembrane pressure**:

This should be <10 mmHg/L of ECMO flow (and is normally around 5 mmHg/L)

A TMP >40-50mmHg at any time is highly suspicious for oxygenator thrombosis

CHECKING THE MEMBRANE OXYGENATOR

- Oxygenator failure is a recognized phenomenon
- Life of an oxygenator is ~ 2-3 weeks
- Circuit failure is characterized by a gradually decreasing post oxygenator pO2
- The best response is a timely circuit change

CASE THREE

A 40-year-old man is receiving VA ECMO support following an out-of hospital cardiac arrest. He is also receiving continuous renal replacement therapy (CRRT) for an acute kidney injury.

You notice that the effluent bags of the CRRT have developed a reddish hue.

What do you do?



HEMOLYSIS

This patient is likely hemolyzing

- This affects 1 in 5 ECMO patients and shouldn't occur unless there is a problem in the circuit
- Hemolysis is associated with acute kidney and multiorgan dysfunction in ECMO patients
- Check plasma free hemoglobin (the most sensitive test for hemolysis and should be < 0.1 g/dL).
 - If PFHb not available, check LDH, K+, bilirubin and haptoglobin
- ECMO-related causes include:
 - Access insufficiency
 - Pump head thrombosis
 - Oxygenator thrombosis

CASE FOUR

A "Code Blue" is called on a 45 yo F on VV ECMO for ARDS due to influenza.

How should you respond?

What is the most likely cause of her arrest?

CODE BLUE/CARDIAC ARREST IN VV ECMO PATIENT

- Cardiac arrest is associated with a sudden loss of native cardiac output during ECMO support, typically due to a dysrhythmia, most commonly:
 - Ventricular fibrillation or tachycardia
 - Ventricular standstill
 - Asystole
- Unlike VA ECMO, VV ECMO is completely dependent on native cardiac output.
 - START COMPRESSIONS and other therapy by normal ACLS algorithm
 - CONTINUE ECMO even at low flows
 - Stopping ECMO will worsen hypoxemia during arrest and decrease chances of ROSC

CODE BLUE/CARDIAC ARREST IN VV ECMO PATIENT

Always exclude other possibilities:

- Hyper/hypokalemia
- Pneumothorax
- Cardiac tamponade
- Hemorrhage
- Air embolus

CODE BLUE/CARDIAC ARREST IN VV ECMO PATIENT

- IF NO CLEAR CAUSE, CHECK THE ECMO CIRCUIT for:
- Power supply failure: Alarm should sound
- Lack of oxygen supply: Is O2 cylinder empty after transport? Is the oxygenator connected to the wall supply
- Oxygenator failure: Usually gradual and associated with decreasing post-oxygenator PaO2 and an increasing pressure drop (the difference between pre-oxygenator pressure and post-oxygenator pressure)
- Cannula migration: Can cause either low flows or significant recirculation

CASE FIVE

A 36 yo M with necrotizing pneumonia on VV ECMO has recurrent access insufficiency.

An ultrasound demonstrates large right pleural effusion compressing his SVC and right atrium.

What do you do?



BLEEDING RIS

- Risk of bleeding with any procedure performed on an ECMO patient is significant and potentially life threatening.
- Hemorrhage is the most common complication of ECMO, due to the need to anticoagulate the circuit
- All interventions require a careful weighing of the risks and benefits
- CT surgery recommends VATS?
 - Most ECMO centers hold anticoagulation for 12 hours prior to and 24 hours after major procedures.
 - The patient is stable for the first 24 hours, but then has sudden loss
 of 1 liter of blood via chest tube.

MAJOR LIFE THREATENING BLEEDING ON ECMO

- Stop heparin and consider reversal with protamine (though this risks circuit thrombosis, it may be essential in life-threatening hemorrhage)
- Activate the massive transfusion protocol
- Use a blood warmer for rapid fluid administration via large bore access (e.g. swan sheath or RICC line) with early use of blood products
- Consult the on call transfusion hematologist
- Assess clotting profile, and correct coagulopathy and other bleeding diatheses if present (e.g. platelets, INR, hypocalcemia, and potentially reversible pro-hemorrhagic medications)
- Correct acidosis and avoid hypothermia
- CT surgery or IR may need to pursue additional interventions such as packing, embolization or pneumonectomy

CASE SIX

A 62 yo M is supported with peripheral V-A ECMO. He presented 3 days ago with a STEMI, the percutaneous coronary intervention was complicated by pulmonary aspiration requiring intubation. Despite revascularization he had progressive cardiogenic shock and was placed emergently on V-A ECMO. The circuit is running without any problems but he is now hypoxemic with an arterial O2 saturation of 82%.

What should you do to assess him?

DIFFERENTIAL HYPOXEMIA

Patients on V-A ECMO have retrograde blood flow in the aorta,

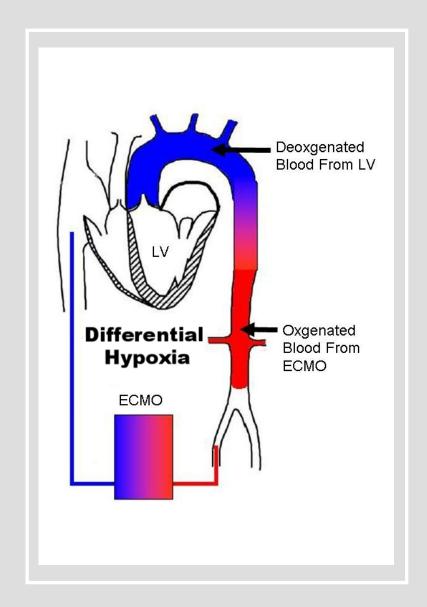
- Blood moves up the aorta toward the heart from the ECMO circuit
- When there is intrinsic cardiac activity there is also anterograde blood flow entering the aorta from the heart.
- The transition point where opposing flows meet in the aorta will vary
 - Affected by both ECMO blood flow and the patient's cardiac output
 - In the presence of pulmonary disease, the blood leaving the heart may be poorly oxygenated due to impaired gas exchange in the lungs; the transition point occurs more distally and the blood is more poorly oxygenated
 - SpO2 on the right radial aline is monitored as an early indicator that the transition point is moving distally because the right subclavian artery arises from the brachiocephalic artery, which is a proximal branch of the aorta

DIAGNOSIS OF DIFFERENTIAL HYPOXEMIA

- Differential hypoxemia is a CLINICAL diagnosis, but there are some pre-requisities
 - Patient on peripheral VA ECMO (doesn't occur on central VA ecmo)
 - Patient must have significant intrinsic cardiac output (indicated by good pulsatility present on arterial trace)
 - Patient must have coexistant pulmonary dysfunction resulting in impaired oxygenation of blood flowing through the pulmonary circulation (indicated by imaging of the lungs, such as a chest xray, showing relevant pathology)

AND

Markedly lower oxygen saturation (SpO2) between the right arm compared to the left arm or between upper and lower limbs (the so-called "North-South phenomenon"). If in doubt this may need ABGs to confirm.



WHY CAN'T YOU HAVE DIFFERENTIAL HYPOXEMIA IN CENTRAL VA ECMO

- In central VA ECMO the return cannula is sutured into the proximal aorta
- VA ECMO characterized by predominately anterograde blood flow down the aorta
- May see a drop in global oxygen saturation with increasing amount of deoxygenated blood ejected from the heart
 - But significant differences in oxygen saturation between upper and lower body will not occur
- FYI: In a central VA ECMO configuration, the right upper extremity pulse oximetry (or arterial line) reflects ECMO circuit function while the left upper extremity pulse oximetry (or arterial line) reflects native respiratory function

NORTH SOUTH SYNDROME (AKA HARLEQUIN SYNDROME)

Due to flow competition between the recovering heart and the ECMO circuit

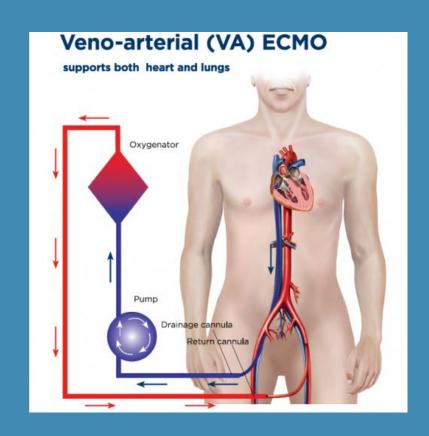
Saturation in the upper part of the body is lower that that of the lower half

High cardiac output from the recovering native heart prevents the oxygenated blood from retrograde ECMO flow in the distending aorta from fully perfusing the upper part of the body

If pulmonary function is also impaired:

"Blue Head": dexxoygenated blood to the upper part

"Red Legs": hyperoxygenated blood to the lower part



ACLS IN VA ECMO

Shockable VT/VF

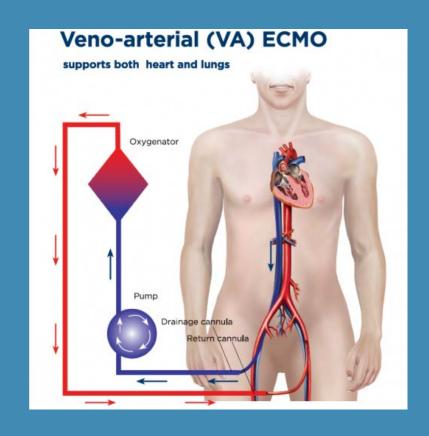
 Defibrillation — 150J (some suggest 100J initial for VT on VA ECMO)

Amiodarone 300mg IV after the third shock

Avoid 1mg adrenaline boluses, consider titrating oppressors to target MAP

Non-shockable Asystole/PEA

Avoid 1mg adrenaline boluses, consider titrating vasopressors and/or inotropes to target MAP



SUMMARY AND WRAP UP

