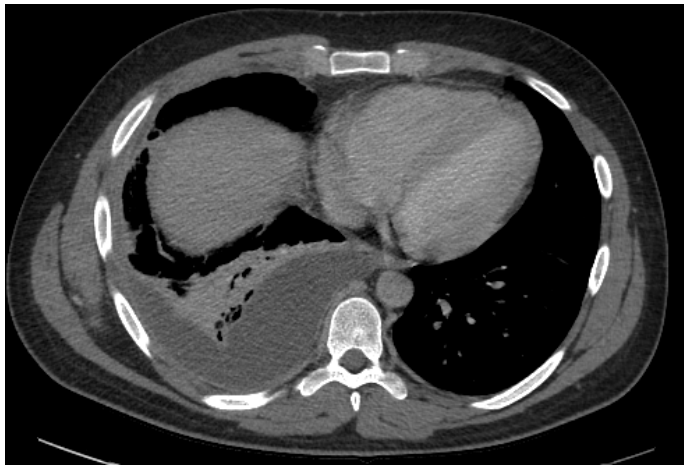
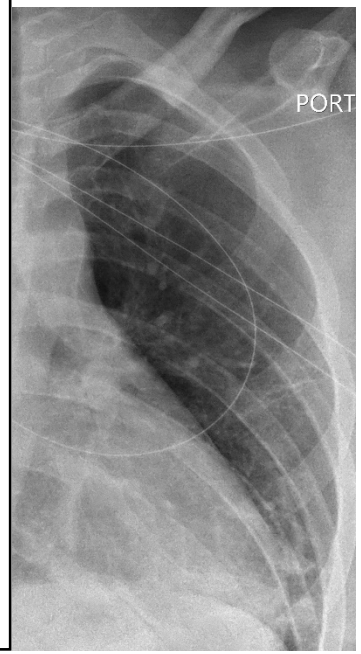
33M +CANNABIS/+TOB: R EMPYEMA S/P R VATS DECORTICATION

PRE-OP



D/c home POD 2



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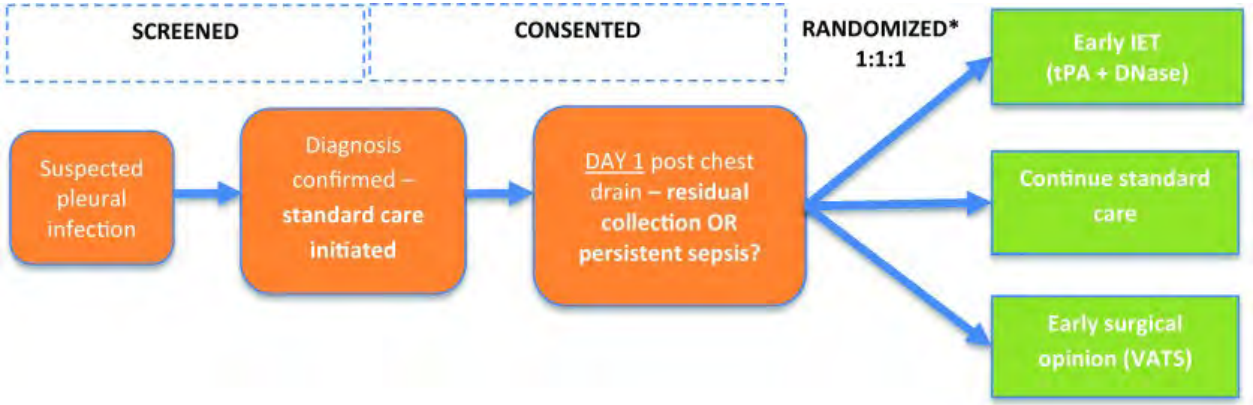
MIST-3: EARLY VATS OR IET IN PLEURAL INFECTION

- N= 19 IET and 20 VATS
- **0% conversion rate** VATS to thoracotomy
- **VATS trended towards dec LOS**
- **High readmission rates for all**
- Conclusion: Must do a larger study (IET vs. VATS)
  - IET **or** VATS



	Standard Care ($n = 21$)	IET ($n = 19$)	VATS ($n = 20$)
Further hospital admission	5 (23.8%)	5 (26.3%)	6 (30.0%)
Further intervention			
Surgery	1 (4.8%)	0	1 (5.0%)
Chest drain	0	1 (5.3%)	1 (5.0%)
Other	1 (4.8%)	1 (5.3%)	0

Am J Respir Crit Care Med. 2023

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RCT: EFFECT OF INTRAPLEURAL FIBRINOLYTIC THERAPY VS. SURGERY FOR COMPLICATED PLEURAL INFECTION

- Pilot RCT of 20 patients w/ empyema (32 months to enroll!!)
- Feasibility study: 100% enrollment and completion
- **30% in IPFT failed**: inc pain, hemothorax, increased loculations
- LOS: IPFT 11 days vs. 5 days VATS decort (P=0.08)
- 0% mortality in both groups

Table 2. Clinical and Outcome Details Overall and Stratified by Treatment Group

Outcome	Treatment group[a]			*P* value, IPFT vs surgery
	Overall (n = 20)	IPFT (n = 10)	Surgery (n = 10)	
Treatment outcomes				
Radiographic improvement, %				
<50	6 (30)	3 (30)	3 (30)	
50-75	6 (30)	2 (20)	4 (40)	.80
>75	8 (40)	5 (50)	3 (30)	
Treatment failure	1 (5)	1 (10)	0	>.99
Crossover treatment	2 (10)	2 (20)	0	.46

JAMA Netw Open. 2023

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FIBRINOLYTICS – ARE THEY ALL BENIGN?

- 16.1% significant bleeding
- 5.4% needed VATS for emergent bleeding

RESEARCH NOTE Open Access

Clinical efficacy and bleeding outcomes of tissue plasminogen activator and dornase alfa in pleural space infection with once daily concurrent administration: a retrospective cohort study

Chuan Jiang[1*], Meng Xie[2], Kelly Cervellione[2] and Craig Thurm[1]

QDAY

Bleeding outcomes	
Pleural bleeding	9 (16.1)
Pleural bleeding requiring ≥ 2U pRBC transfusion	5 (8.9)
Gastrointestinal bleeding	0 (0)
Intracranial bleeding	0 (0)
Other bleeding	0 (0)
Mean hemoglobin loss (SD), g/dL	3.5 (1.1)
Median units of pRBC transfusion in patients transfused (IQR)	2 (2–5)
Surgical intervention for pleural bleeding, n (%)	3 (5.4)
30 day mortality due to pleural bleeding, n (%)	0 (0)

BMC Res Notes 2020

KP NCAL DATA – CHEST TUBES AND FIBRINOLYTICS

- ~ 5.5% of patients got MIST-2 dosing
- Extreme variability in dosing, most Qday dosing, by PULM/HBS/IR/Surgery
- Weekends and nights – very inconsistent
- 37% of patients required > 1 tube
- 15.8% > 3 tubes!!!

TPMG Healthcare delivery science grant

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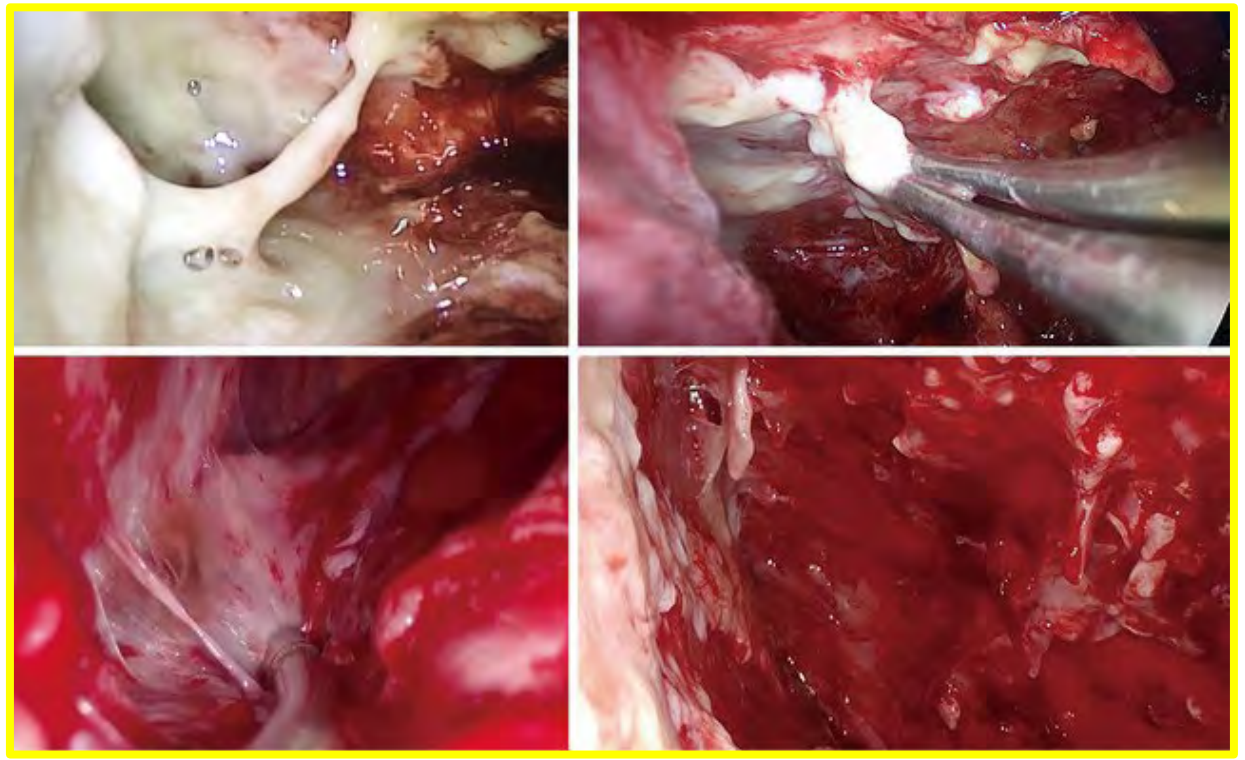
TAKE HOME POINTS: PRO EARLY VATS

- **Early VATS Decortication** should be first line treatment for complex pleural infections
  - Dec LOS, Chest tube duration
- Fibrinolytics are NOT BENIGN treatments
- Multidisciplinary approach **EARLY** on is the key (HBS, Pulm, Surg, ID, Radiology, IR)



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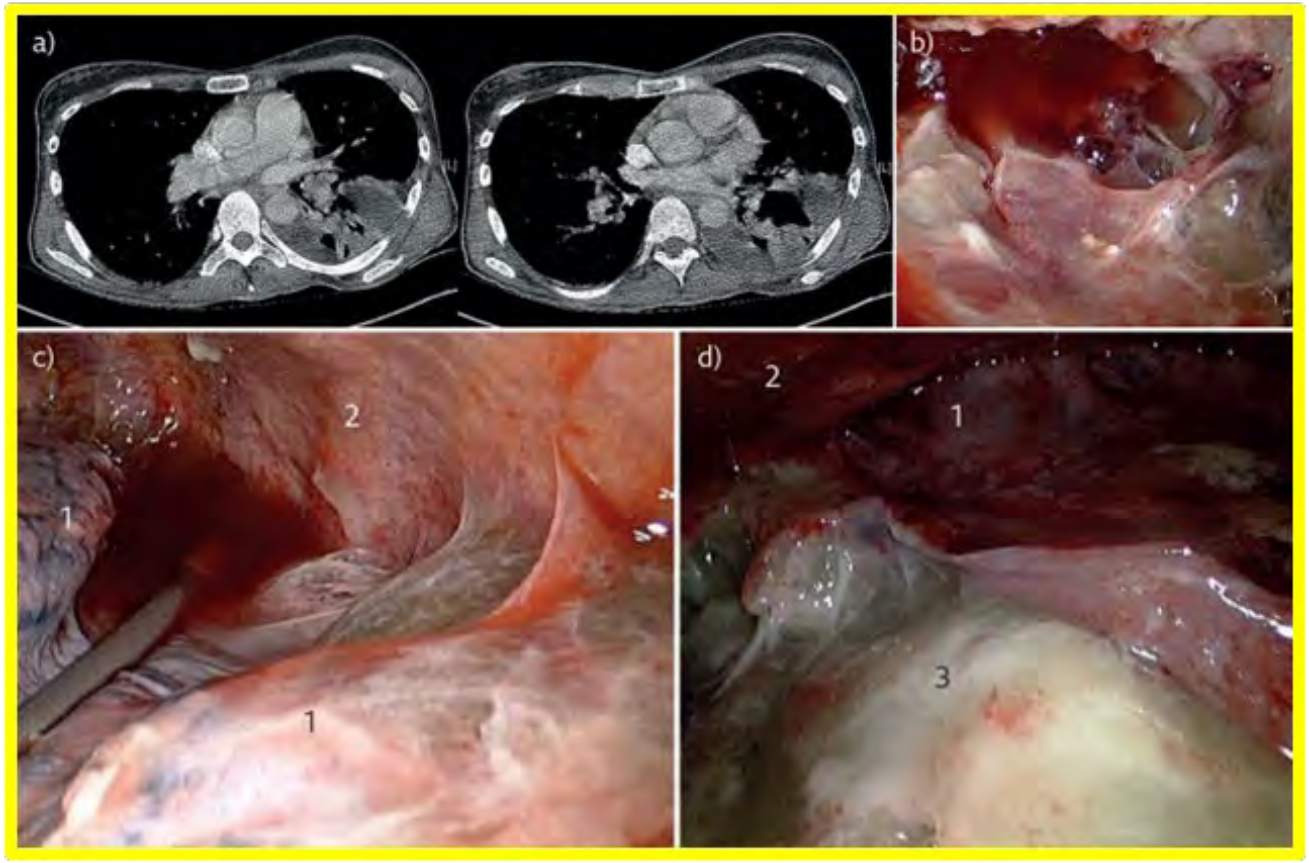
FUTURE DIRECTIONS

- DICE Trial – RCT
- VATS Decortication vs. IR guided Chest Tube Insertion with Fibrinolytics for the Management of Empyema
- Ontario, Canada
- End of 2025 recruitment



Subotic et al. *Breathe* 2018

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NOW FOR THE COUNTERPOINT

@JVelottaMD

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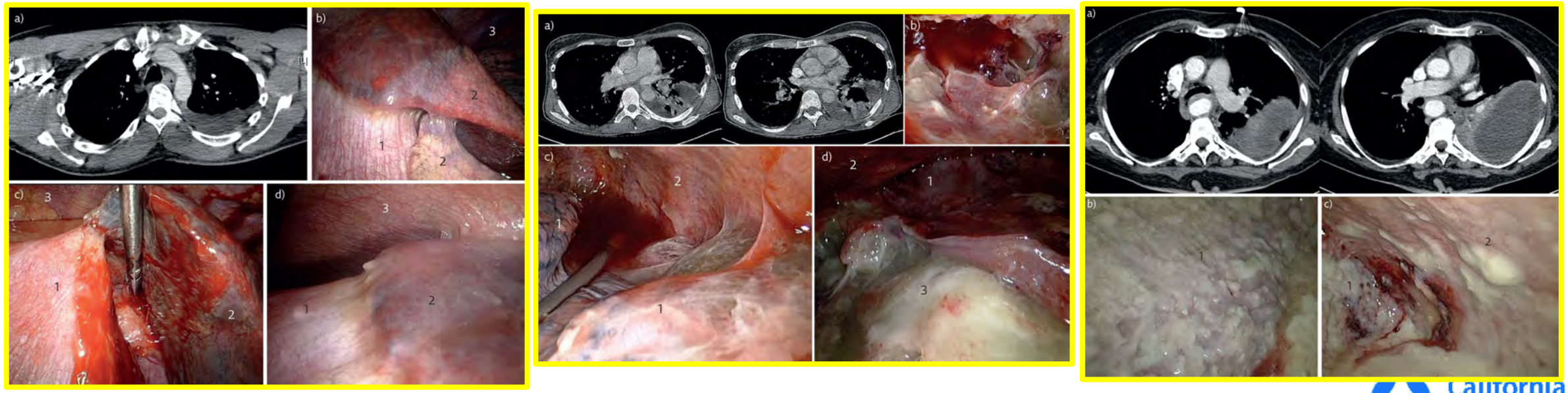
EXTRA SLIDES

EMPYEMA STAGES

1. Exudative – sterile exudate low in cell count
2. Fibrinopurulent – Frank pus and increase in WBC
3. Organizing – In growth of fibroblasts into the fibrinous peel

1 **2** **3**



Subotic et al. *Breathe* 2018

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NOTES ON SOCIETY GUIDELINES

- No ATS guidelines for empyema however, Light et al ATS proceedings 2005 – stepwise approach from least aggressive to most aggressive
- BTS guidelines:Roberts ME, Rahman NM, Maskell NA, Bibby AC, Blyth KG, Corcoran JP, et al.BTS Pleural Guideline Development Group British Thoracic Society Guideline for pleural disease. *Thorax* . 2023;78:s1–s42.
- Chest guidelines: Colice GL, Curtis A, Deslauriers J, Heffner J, Light R, Littenberg B, Sahn S, Weinstein RA, Yusen RD. Medical and surgical treatment of parapneumonic effusions : an evidence-based guideline. Chest. 2000 Oct;118(4):1158-71. [PubMed] [Reference list]
- STS guidelines: Ann Thorac Surg 2018 – earlier VATS beneficial. See PPT from Davitt.
- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10765400/#bib10 – 2023 Dec good synopsis of MIST-3 – will await final results on which treatment may be better, was more of a feasibility study that we can do a RCT in these groups

STS "GUIDELINES"

Current State of Empyema Management

Tara R. Semenkovich, MD, MPHS, Margaret A. Olsen, PhD, MPH, Varun Puri, MD, MSCI, Bryan F. Meyers, MD, MPH, and Benjamin D. Kozower, MD, MPH

Division of Cardiothoracic Surgery, Department of Surgery, and Divisions of Infectious Diseases and Public Health Sciences, Departments of Medicine and Surgery, Washington University in St. Louis, St. Louis, Missouri

Background. Empyema affects up to 65,000 patients annually in the United States. Recent consensus guidelines demonstrate ambiguity about optimal treatment. We examined current treatment practices and outcomes for inpatient treatment of empyema using a comprehensive, longitudinal data set that encompasses an entire state cohort of hospitalized patients.

Methods. We queried the Healthcare Cost and Utilization Project New York State Inpatient Database (2009 to 2014) for patients with primary empyema and subsequent readmissions. Patients were categorized into three groups by definitive treatment during their initial hospitalization: chest tube drainage, video-assisted thoracoscopic surgery (VATS) decortication and drainage, or open decortication and drainage. Treatment outcomes, including success rates, readmission, reintervention, and mortality, were compared between groups.

Results. The cohort included 4,095 patients undergoing intervention for primary empyema discharged during this period with chest tube, VATS, or open drainage and decortication. Most patients received definitive operative management (chest tube: 38.2%, VATS: 32.1%, open: 29.8%; $p < 0.001$). Patients had a high mortality rate during their initial hospitalization (chest tube: 15.4%, VATS: 4.7%, open: 6.0%; $p < 0.001$) and a substantial 30-day readmission rate for empyema (chest tube: 7.3%, VATS: 3.8%, open: 4.1%; $p < 0.001$), with reintervention at readmission significantly higher for chest tube (6.1%) vs surgical patients (VATS: 1.9%, open 2.1%; $p < 0.001$).

Conclusions. This study characterizes recent treatment practices of patients with empyema. Higher readmission and reintervention rates were observed in patients managed with chest tubes, suggesting some of these patients may benefit from earlier definitive surgical intervention.

(Ann Thorac Surg 2018;105:1589–96)

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SURGICAL VS. NON-SURGICAL MANAGEMENT OF EMPYEMA: THE STUDIES

- **Many limitations**
  - Few RCTs (all small sample sizes)
  - low to moderate quality of evidence
- **No difference in mortality** (only one 1 reported mortality as an endpoint)
- Significant decrease of 2.52 days in VATS vs. thoracostomy
  - Not significant for pediatrics
- Significant increased cost in VATS vs. thoracostomy (3 studies)
- Significant decrease in chest tube duration with VATS or OPEN (outpatient longer in surgical arms?)
- Fibrinolytics should not be used routinely due to lack of data (prior to MIST3)
- Flushing w/ saline TID for 3 days beneficial

Redden et al *Cochrane Database 2017*

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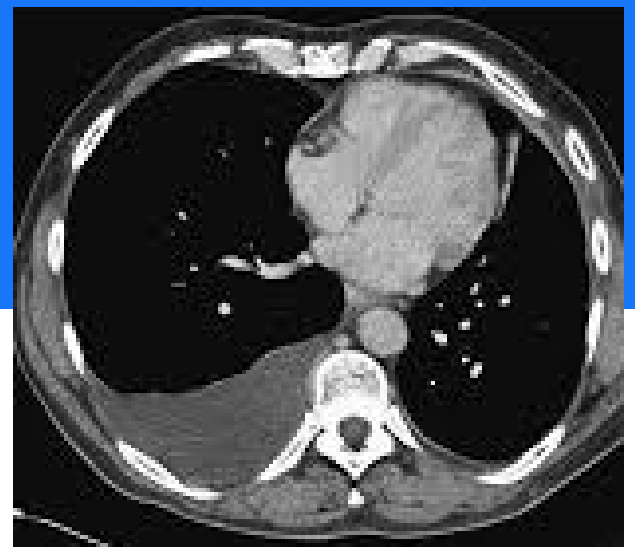
BENEFITS OF SURGERY FIRST

- Direct visualization of the infected space AND treatment
- Standard of care if chest tube/fibrinolytics/antibiotic treatment fail – so why not just do it right off the bat, if can't get into the OR right away, give fibrinolytics or saline (PIT trial)
- Fibrinolytics are contraindicated: anticoagulation, hemothorax, BPF, that's a lot of issues with fibrinolytics

- t-PA/DNase (10mg/5mg) – relative contraindication in BPF, hemothorax/pleural bleeding (jTD 2015)
  - Anitocoagulation? What types?

CHEST TUBE AND INTRAPLEURAL FIBRINOLYTICS



- MIST 1 RCT (Multicenter Intrapleural Sepsis Trial1)
  - Intrapleural streptokinase: NO difference in mortality, rate of surgery, LOS
- MIST 2 RCT (Multicenter Intrapleural Sepsis Trial2) **N=52 t-PA/DNase**
  - t-PA and DNase combined resulted in decreased LOS (-6.7 days) and 77% reduction in need for surgical intervention compared to placebo
  - **NO mortality diff: 8% vs. 4% (P=0.46) t-PA/DNase vs. placebo**
  - **DNase ALONE NOT recommended**
    - **3x increased risk for surgery referral, no fluid-drainage benefit, trend towards higher mortality**

Table 2. Primary and Major Secondary Outcomes, According to Study Group.*

Outcome	t-PA	DNase	t-PA–DNase	Placebo
Change from baseline in hemithorax area occupied by effusion (primary outcome) — %	−17.2±24.3	−14.7±16.3	−29.5±23.3	−17.2±19.6
Percent difference vs. placebo (95% CI)	2.0 (−4.6 to 8.6)	4.5 (−1.5 to 10.5)	−7.9 (−13.4 to −2.4)	NA
P value	0.55	0.14	0.005	NA
Surgical referral — no. referred/total no. (%)	3/48 (6)	18/46 (39)	2/48 (4)	8/51 (16)
Odds ratio vs. placebo (95% CI)	0.29 (0.07 to 1.25)	3.56 (1.30 to 9.75)	0.17 (0.03 to 0.87)	NA
P value	0.10	0.01	0.03	NA
Hospital stay — no. of days	16.5±22.8	28.2±61.4	11.8±9.4	24.8±56.1
Percent difference vs. placebo (95% CI)	−8.6 (−40.8 to 3.3)	3.6 (−19.0 to 30.8)	−14.8 (−53.7 to −4.6)	NA
P value	0.21	0.73	<0.001	NA

* Plus–minus values are means ±SD. The mean values for the primary analysis are unadjusted, whereas the treatment effects have been adjusted for minimization criteria and opacification of the chest radiograph at baseline, according to the statistical analysis plan. Data on hospital stay are for all patients in the primary analysis (i.e., including two patients with outlying results). NA denotes not applicable.

Maskell NEJM 2005
Rahman NEJM 2011

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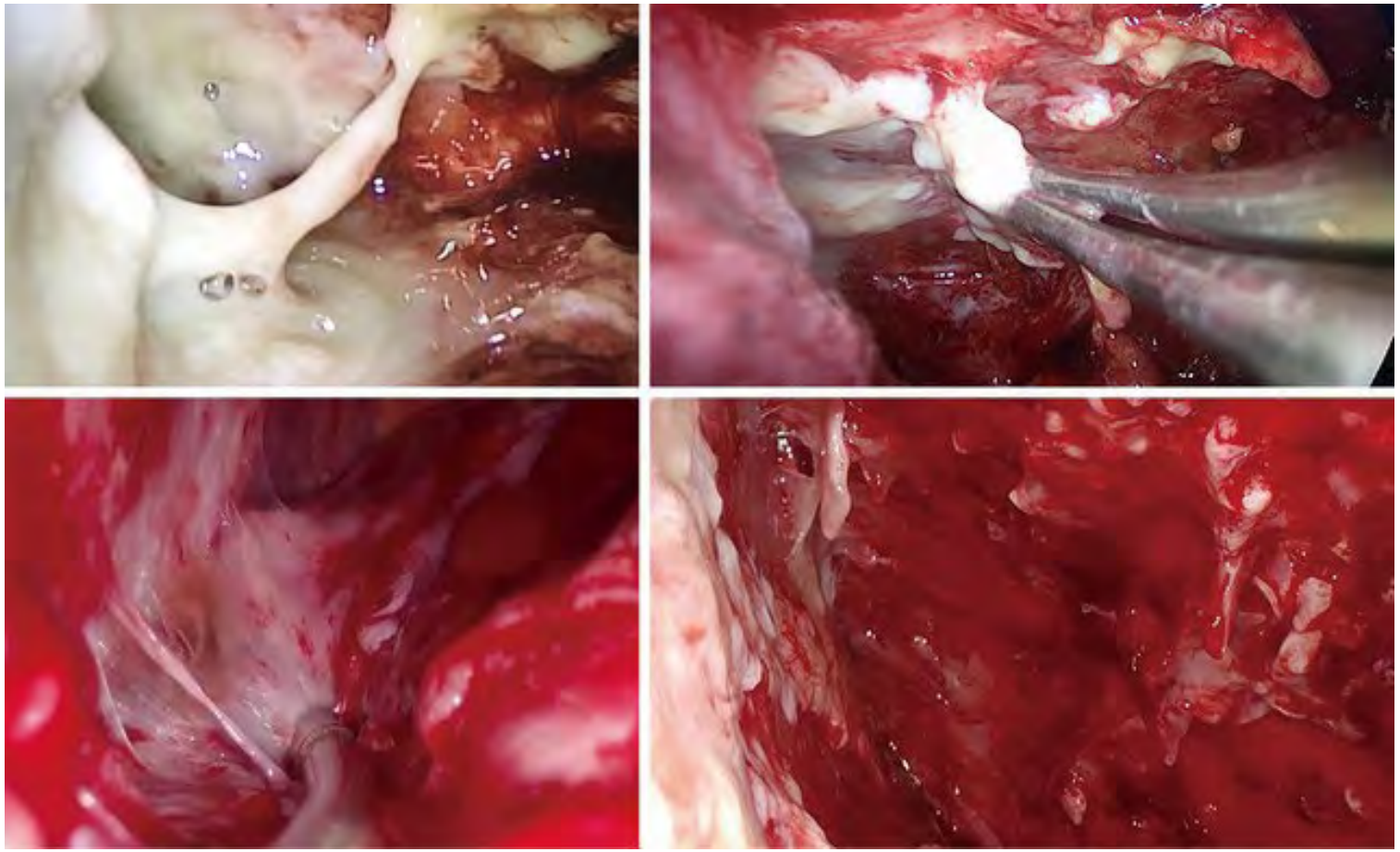
INTRAOPERATIVELY VATS DECORTICATION



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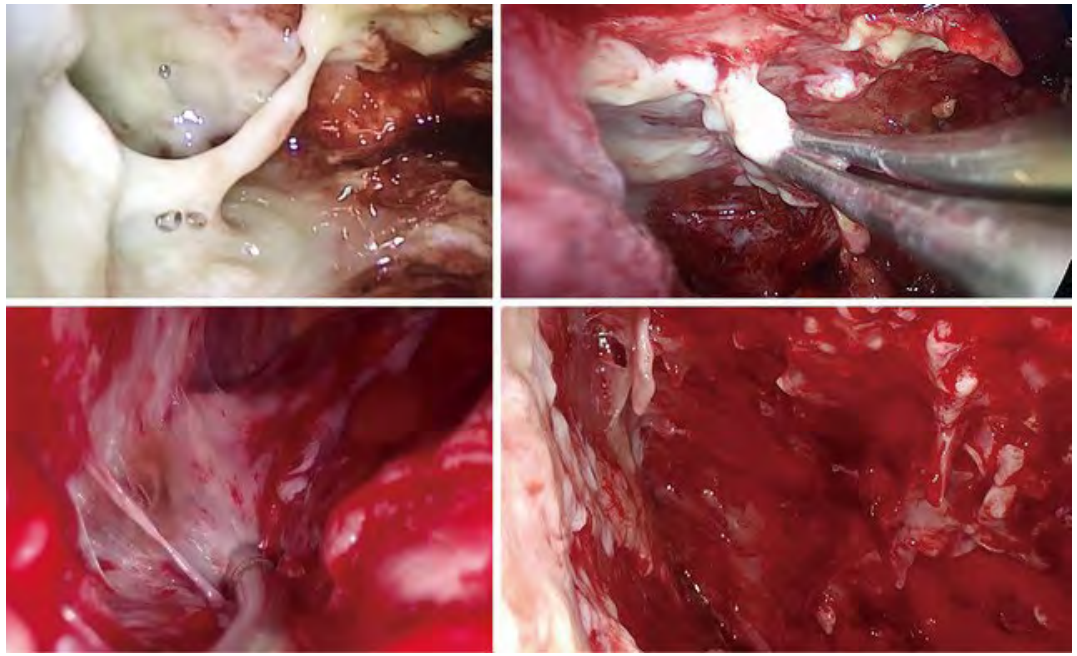
TAKE HOME POINTS

- Ultimate decision is with the treatment team (HBS/PULM/ID/Surgeon) and the patient
- Yes --------- **Case by case situation**
- If surgery – VATS is the preferred first line method for Stage 1 and 2 empyema
  - Conversion rates to thoracotomy range from 5.6% to 61%
  - Thoracotomy for extreme chronic rind (Stage 3)
- Multidisciplinary approach EARLY on is the key (HBS, Pulm, Surg, ID, Radiology, IR)



[Thoracic Oncology Original Research]

CHEST

Bleeding Risk With Combination Intrapleural Fibrinolytic and Enzyme Therapy in Pleural Infection

An International, Multicenter, Retrospective Cohort Study

- 4.1% pleural bleeding
  - 25% required operation
- 30.6% Total adverse events other than bleeding
- 39.9% Pain requiring escalation of analgesics

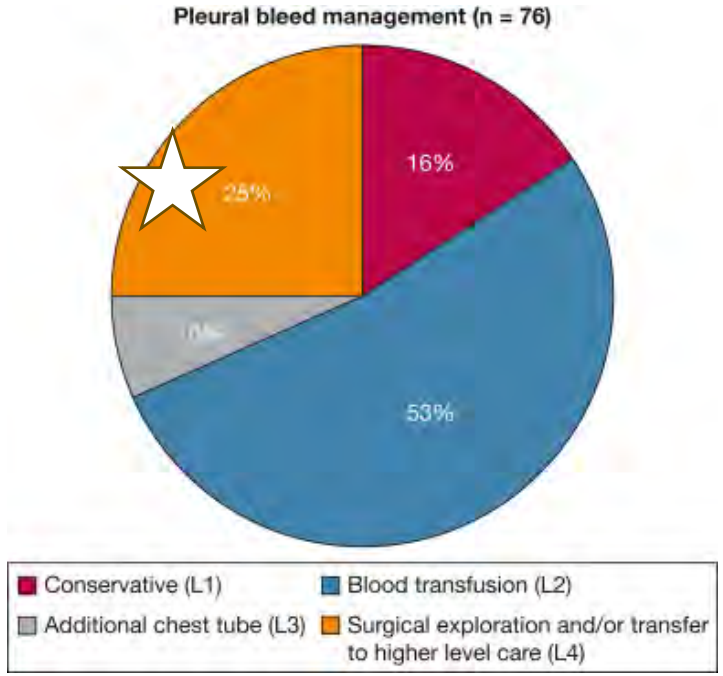


Table4. Main Categories of Adverse Events Reported After IET Administration

Adverse Event	No.	%of All Adverse Events (n= 561)	%of Study Population (n= 1,833)	95%CI
Pain requiring escalation of analgesics	224	39.9	12.2	11%-14%
Increased oxygen requirement	71	12.6	3.9	3%-5%
Increased level of care	44	7.8	2.4	2%-3%
Death	16	2.8	0.9	0%-1%
Hemoptysis	7	1.2	0.4	0%-1%
Other[a]	55	9.8	6.9	5%-8%

Chest 2022

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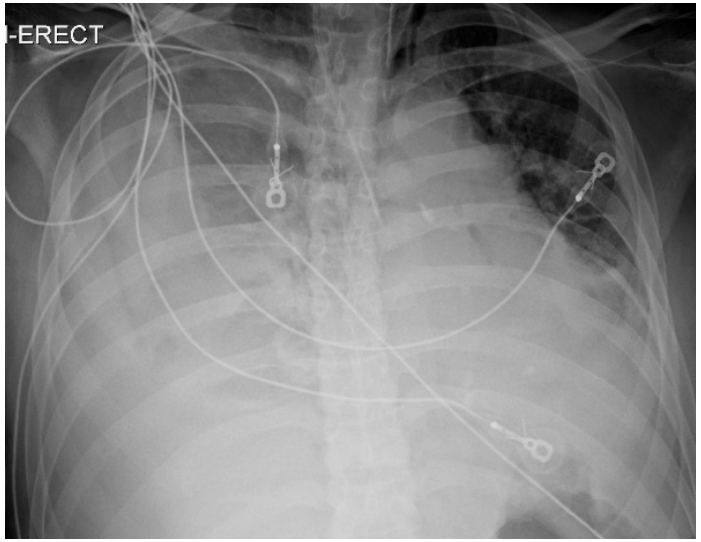
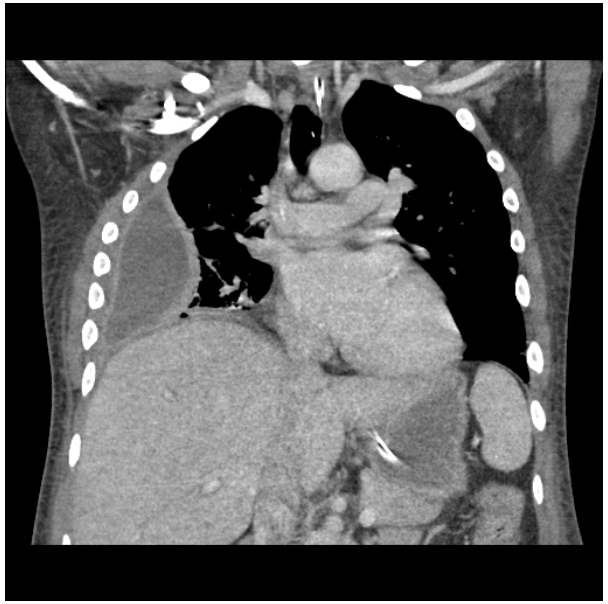
55 MALE +ETOH, SCHIZOPHRENIA: R EMPYEMA S/P R VATS DECORTICATION

PRE-OP





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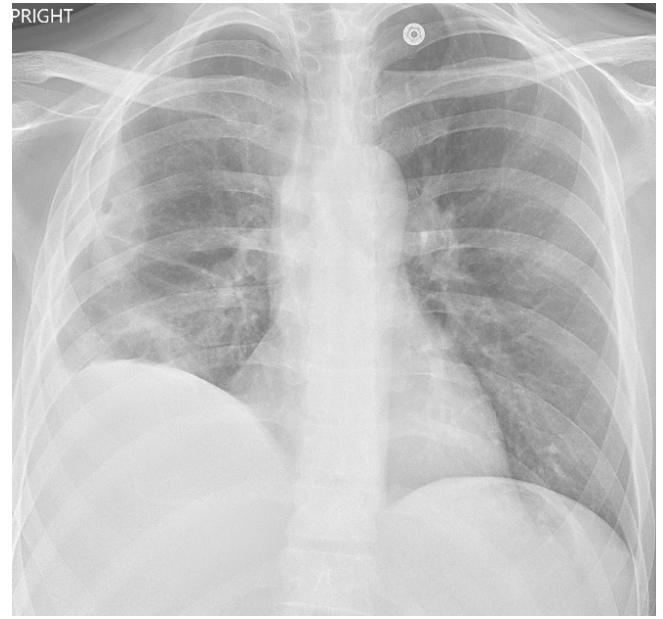

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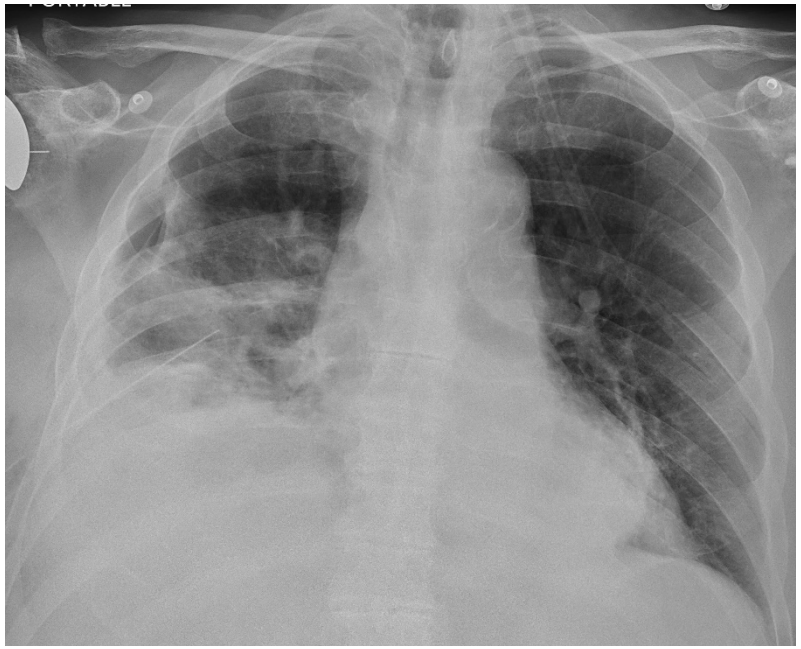
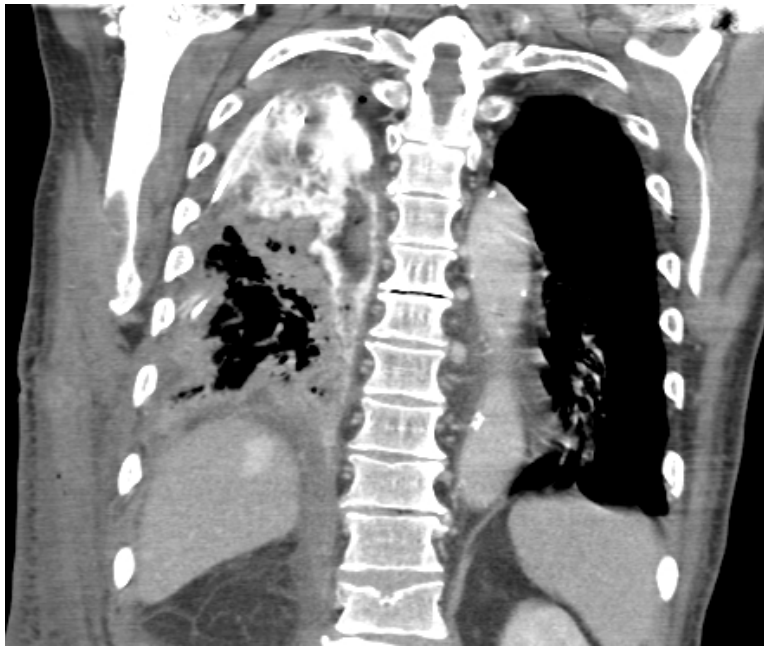
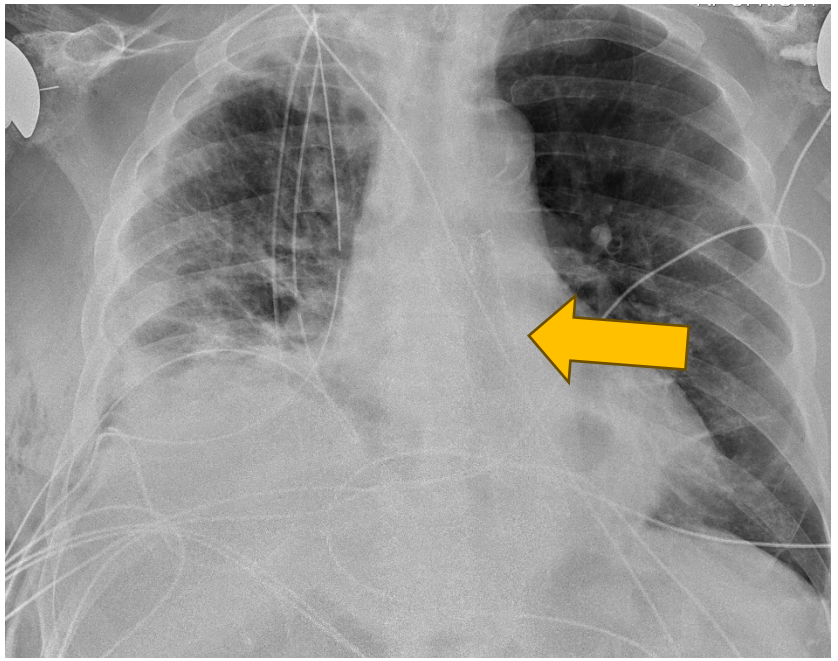
84M +ETOH +TOB COUGH/SOB: R EMPYEMA S/P R VATS DECORTICATION

PRE-OP





D/c Home POD 7



Con: A Surgical Approach is NOT Fiirst Line for Lung Entrapment?

Ai-Yui M. Tan, MD
Staff Physician
Cedars-Sinai Medical Center

Dr. Tan received her medical degree from the Ruhr University School of Medicine in Bochum, Germany. She completed internal medicine residency at Icahn School of Medicine Elmhurst Hospital Center in New York followed by fellowships in pulmonary and critical care at the University of Illinois at Chicago and interventional pulmonary medicine at the Chicago Chest Center. Dr. Tan is now faculty in the Division of Pulmonary and Critical Care at the Cedars-Sinai Medical Center in Los Angeles.

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Hands-on Session: CPR Devices

William Auyeung, MD
Assistant Professor
VA Palo Alto/Stanford University

Dr. William Auyeung received his medical degree from UC San Diego School of Medicine. He completed his fellowships in Pulmonary, Critical Care, and Sleep Medicine at Stanford University. He is currently a Staff Physician at the Palo Alto VA Medical Center and serves as a Clinical Assistant Professor (Affiliated) within the Stanford University Department of Medicine, Division of Pulmonary, Allergy, and Critical Care Medicine.

Hands-on Session: Monitors and Defibrillators

Crystal IvesTallman, MD
Assistant Professor
UC San Francisco-Fresno

Crystal Ives Tallman is an emergency medicine and critical care physician at UCSF Fresno. She completed her emergency medicine residency at UCSF Fresno and her critical care fellowship at the University of Michigan. She works 50% of her clinical time in the medical ICU and 50% in the emergency department. She is an ECMO cannulator and a founding member of the UCSF Fresno ECMO faculty group. She is the education director for Emergency Medicine and Critical Care at UCSF Fresno.

Hands-on Session:
Percutaneous Ventricular Assist Devices

Daniel Gerber, MD
Assistant Professor
Stanford University/VA Palo Alto

Dr. Gerber received his medical degree from George Washington University and completed his residency and fellowship training at Stanford University. He is board certified in internal medicine, cardiovascular medicine, critical care medicine, and adult echocardiography. Currently, Dr. Gerber is a Clinical Assistant Professor at Stanford University where he serves as Director of the Cardiac ICU and Director of Critical Care Ultrasound.

Hands-0n Session: Pleural Catheters

Jeffrey B. Velotta, MD, FACS
Physician
Kaiser Oakland

Dr. Jeffrey Velotta, MD, FACS is a thoracic surgeon at Kaiser Permanente Oakland Medical Center, an adjunct Clinical Assistant Professor in the Department of Surgery at the University of California, San Francisco (UCSF) School of Medicine, and Clinical Professor in the Department of Clinical Science at the Kaiser Permanente Bernard J. Tyson School of Medicine. Dr. Velotta's clinical and research interests involve innovative techniques and regionalization pathways for lung cancer, esophageal cancer, and mesothelioma.

Ai-Yui M. Tan, MD
Staff Physician
Cedars-Sinai Medical Center

Dr. Tan received her medical degree from the Ruhr University School of Medicine in Bochum, Germany. She completed internal medicine residency at Icahn School of Medicine Elmhurst Hospital Center in New York followed by fellowships in pulmonary and critical care at the University of Illinois at Chicago and interventional pulmonary medicine at the Chicago Chest Center. Dr. Tan is now faculty in the Division of Pulmonary and Critical Care at the Cedars-Sinai Medical Center in Los Angeles.

Ilana Krumm, MD
Fellow
UC San Francisco

Ilana Roberts Krumm is a Chief Clinical Fellow in the Division of Pulmonary/Critical Care Medicine at the University of California – San Francisco, and is simultaneously obtaining a Master's in Education from UC Berkeley. She is pursuing additional subspecialty training next year at the Harvard-combined Interventional Pulmonology fellowship program. Her passion for medical education is evident in her body of work, and she aims to use her expertise in her pursuit of a career in advancing medical education.

Hands-on session cheat sheet—monitors and defibrillators

Lifepak 15 V4(+) Stryker

1. Rhythm detection

- Manual mode
    - Default is to power on in manual mode.
    - Standard ECG with 4 wire cable
- Attach to green port, turn on monitor.
- Attach leads.
- Press lead button to automatically select lead II.
- Therapy leads
    - Attach therapy leads, anterior-lateral.
- Press lead button.
    - Acquiring a 12-lead EKG
        - Requires placing limb and pre-cordial leads.
        - Press 12-lead button to acquire ECG.
        - Use speed dial to enter patient age and sex (default is 50y and male).
        - Prints out EKG and algorithm interpretive statement.

2. Defibrillation

- 200J is pre-selected.
- If you want different energy level, press energy select and use speed dial.
- Press charge, when fully charged shock becomes available. Press the shock button to shock.
- To cancel the charge, press the speed dial. If you don't press the shock button within 60 seconds, the charge will automatically be cancelled.
- To activate CPR metronome, press CPR.
- If you press the shock button and nothing happens, sync may be on. Push sync to turn this off, then press the shock button again.

3. Synchronized cardioversion

- Pt is connected with 4 wire ECG leads. Lead II is displayed.
- Therapy electrodes are applied.
- Press synch, note sense mark triangles above the QRS.
- Press energy select, and select appropriate energy .
- Press charge, shock when fully charged.
- Release shock button when you see energy delivered.
- Always re-synchronize between cardioversion attempts!

4. Pacing

- 4 lead ECG leads are applied to the patient, lead II is displayed.
- Place therapy electrodes—electrode with heart goes lateral. Other electrode is posterior (incorrect configurations may require more energy to pace).
- Press Pacer. Sense markers will appear over the QRS complexes.
- Press rate to adjust rate.

- Press current to adjust current.
- Increase current until you see electrical capture (usually between 50 and 100 mA).
- Confirm mechanical capture.

5. Additional

- Can monitor spO2, some with capability to monitor carboxy and met hemoglobin (requires rainbow sensor).
    - Place on non-dominant ring finger, cable to back of hand.
    - Shield sensor from light .
    - If carboxyHb > 10% or metHb > 3%, an advisory occurs.
        - Acknowledge advisory by pressing alarm.
    - Can check values by printing out vital signs, or by using speed dial to select SpO2, and select carboxy or met values, will display for 10 sec and revert back to SpO2.
    - Details available by highlighting and clicking SpO2 area with speed dial.
- Pleth can be displayed by selecting channel 2 or 3 with speed dial, and selecting SpO2.
- NIV BP
    - Default cuff pressure is 160 mmHg, can be changed with speed dial.
    - Recurring BP can be set to various intervals.
- EtCO2 monitoring
    - Open connector door, attach filter line. Turn clockwise until tight.
    - Display waveform in channel 2 or 3.
- Invasive BP monitoring
    - Can measure 2 invasive pressures simultaneously.
    - Set up transducer system as usual.
    - Default label is P1, can select P1 and choose the desired label with the speed dial.
    - To see waveform, select channel 2 or 3, select waveform, and choose desired waveform.
    - Zero the transducer, open the stopcock to air, select zero from the menu. Close stopcock when zeroing is complete (message appears at bottom of the screen).
    - Scale is auto-selected.
- Trend graphs
    - Can display vital sign and ST segment trends (J point elevation trend) over time in channel 2 or 3.
    - 12 lead EKG must remain connected for ST segment trending. If ST segment deviation is noted, repeat EKG is automatically printed.
    - Can print trends over time, if desired.

R series Zoll

1. Rhythm detection

- Turn dial from off to monitor.
    - Second waveform FIL is filtered – filters artifact.
        - Can see rhythm underlying compressions.
        - Aids in decision to pre-charge.
- Hit lead button to change view from leads I-III, or pads.

- Size increases waveform size on monitor and print .
- Alarms off is default.
- Strip is printed default surrounding shocks, can print strip at other times with strip button.

2. Defibrillation

- Connect energy cable.
- Place pads (has CPR quality monitoring pad). Default is anterior/posterior. Pad number one can also be placed laterally on the upper right portion of the chest for anterior/lateral pad positioning. Triangular pad can then be placed more laterally, and CPR sensor can be detached and separately applied.
    - Separate pads for pediatrics with age and weight guidelines. Can put adult pads on large pediatric patients. CPR quality monitor does not detach from pediatric pads.
    - Pro padz are radiolucent adult pads for cath lab.
        - Pro padz connect slightly differently and don't provide CPR feedback.
- Turn to defibrillation mode.
- Default is 120J and will increase after each shock automatically by default.
    - Adult 120-150-200 J
    - Pediatric 50-70-85 J (2-4 J/kg)
- Energy select to select desired energy.
    - Default is overridden when you energy select.
    - Maximum is 200J – zoll energy delivery compensates for the patient impedance. Can see delivered energy displayed below selected energy. Customizes shock for every pt.
- Charge, when lit up press to shock. Hold until shock delivers.

3. Synchronized cardioversion

- 3 lead is built into triangular shaped pad for pacing (doesn't need additional 3 leads if properly placed).
- Turn to defib mode.
- Synchronize with synch button.
    - White markers on top of QRS
- Energy select and charge.
- Press and hold shock button until energy releases—waits for the next R wave and delivers shock at appropriate time.
- Synch prior to every shock!
- Press down on energy arrows disarms energy safely without shocking.

4. Pacing

- Turn to pacing mode.
- May need to add additional 3 lead EKG if you modified your pad placement.
- Select rate (clockwise to increase).
- Select energy (mA) to electrical capture .
- Confirm mechanical capture.
- Default is demand pacing, can select asynchronous pacing if desired.
- 4:1 button slows pacer to see underlying rhythm (push and hold down).

5. Other monitoring

- CPR monitoring capabilities
    - Idle timer, time off chest
    - While compressions ongoing, first number is depth, second is rate.
    - If compressing too fast or too slow, metronome starts to get you back to ideal rate.
    - Release measures recoil.
    - Diamond fills up to show you are within AHA guidelines for depth and rate.
- EtCO2
    - Reusable piece connects to single use plastic piece (zoll logo in front, “ribs toward your patient’s ribs”), no need to zero.

Per AHA, routine use of mechanical chest compression devices is not recommended. It may be useful where reliable, high-quality manual compressions are not possible or may cause risk to personnel:

- moving ambulance
- angiography suite
- prolonged resuscitation
- concerns for infectious disease exposure

	Stryker LUCAS 3	ZOLL AutoPulse
Weight with battery and carry case	22 lbs	39 lbs
Nominal battery runtime	45 min	30 min
Time to fully charge	<2 hrs using Power Supply @72°F <4 hrs using Battery Charger @72°F	<4.25 hrs @77°F
Compression modes	30:2, 50:2, continuous	30:2, 15:2, continuous
Compression rate	Adjustable: 102, 111, 120 ±2 compressions/min	80 ±5 compressions/min
Depth	Adjustable: 1.8-2.1 inches	20% of chest depth
Patient sizing	Chest height 6.7-11.9 in	Chest circumference 29.9-51.2 in
	Max chest width 17.7 in	Chest width 9.8-15 in
Patient weight	No limit, but need to fit inside	Maximum 300 lbs

- Minimize interruptions to manual chest compressions when applying device
- Pause device compressions when analyzing heart rhythm
- If there is any malfunction or poor fit, resume manual CPR

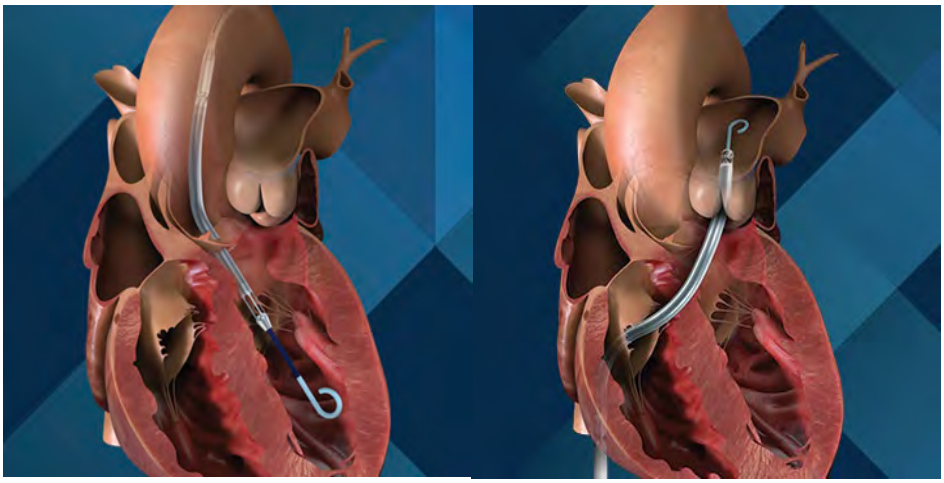
- Devices are defibrillator-proof and water-resistant
- Compared to manual CPR:
  - No significant difference in rates of ROSC on systematic review
  - Expect similar chest wall bruising, broken ribs, and soreness/pain
  - No increase in risk of pneumothorax, hemothorax, or abdominal organ injury

Pleural Disease Hands on Course: Thoracic Surgeon Point of View

1. R/o Malignancy – Thoracentesis x 1, if inconclusive consider VATS or Pleuroscopy and biopsy +/- pleurodesis
2. Talc may not be best agent – asbestos recalls for majority of talc
3. Consider doxycycline – similar efficacy
4. Before considering PleurX – should really discuss with patient and med onc on indications, pros/cons, infection risks, feasibility at home, response to systemic treatment, what type of cancer is it for
5. Should not do PleurX for infection ever or if concern for infection is high
6. When placing chest tube or pigtail, always remember right on the TOP of the rib, intercostal branch bleeders are much more common than reported, and often don't present immediately
7. Smaller tubes are more adequate than previously reported in older literature
8. No 36 Fr tubes should be used anymore
9. Consider "Softer" chest tubes (i.e. Atrium), much less pain than classic "rigid" chest tubes
10. I prefer NOT tunnelling for pigtails and chest tubes, only for PleurX catheters, residents and fellows often get into the wrong area in the pleura when tunnelling
11. Routine post-pull CXRs are NOT required
12. Must always get post-procedure CXR after any implementation into the pleural space – confirmation of placement and potential complications
13. Do you do QDAY or BID TPA/Dornase for empyemas?
14. Pigtails any size are most likely fine when TPA/Dornase is utilized
15. For pleuroscopy, when in doubt the more tissue the better ESPECIALLY for potential mesothelioma diagnosis
16. For IP fellows, recommend spending time with your thoracic surgeon colleagues for tips and tricks and vice versa

Abiomed Impella Platform

Catheter-mounted, microaxial ventricular assist devices provide acute/temporary left, right, or biventricular mechanical circulatory support



Impella CP

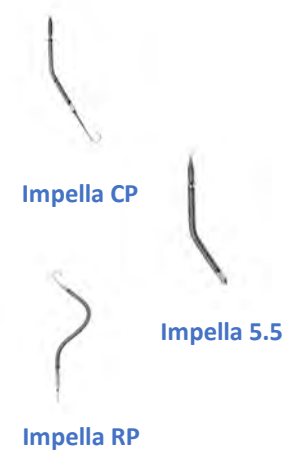
14F percutaneous femoral or axillary artery access. Flows ≤4.3 L/min.

Impella 5.5

23F surgical axillary artery graft. Flows 5.5+ L/min.

Impella RP

23F percutaneous jugular or femoral vein access. Flows 4+ L/min.



Indications

High risk PCI

Cardiogenic shock

VA ECMO unloading/weaning ("ECpella")

Contraindications

LV thrombus, mechanical AVR, severe AS (AVA ≤0.6 cm2), moderate AR or greater (≥2+), severe PAD precluding placement, significant RV failure (biventricular support indicated), ASD/VSD, LV rupture, cardiac tamponade

Complications

Access: neurovascular injury, limb ischemia, bleeding, infection, device migration

Hemodynamic: RV dysfunction, ventricular arrhythmias, valve injury

Hematologic: hemolysis, thrombocytopenia, thromboembolism

Cardiac Critical Care

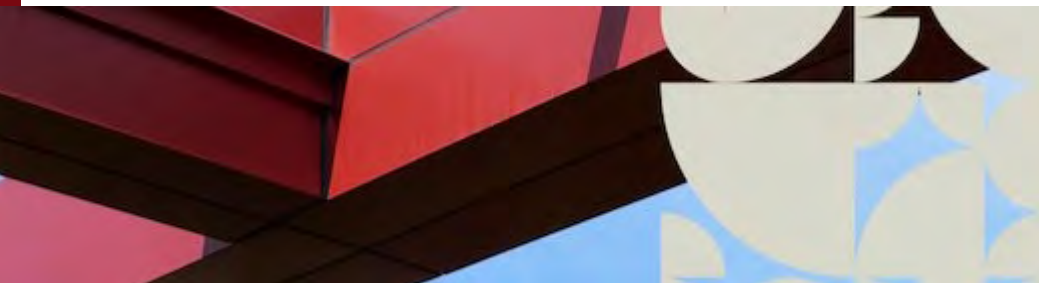
Daniel Gerber, MD
Assistant Professor
Stanford University/VA Palo Alto

Dr. Gerber received his medical degree from George Washington University and completed his residency and fellowship training at Stanford University. He is board certified in internal medicine, cardiovascular medicine, critical care medicine, and adult echocardiography. Currently, Dr. Gerber is a Clinical Assistant Professor at Stanford University where he serves as Director of the Cardiac ICU and Director of Critical Care Ultrasound.

California Thoracic Society
ATS Chapter

Critical Care Cardiology: Contemporary Approach to Cardiogenic Shock

Daniel A. Gerber, MD
Director, Stanford Cardiac Intensive Care
Director, Stanford Cardiogenic Shock Initiative
Clinical Assistant Professor, Division of Cardiovascular Medicine
Stanford University School of Medicine
3/9/2024



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I have no disclosures or conflicts of interest.

TL;DR

Early identification & tailored intervention improve outcomes in cardiogenic shock

Goals

1. Recognize cardiogenic shock earlier
2. Review management strategies for various cardiogenic shock phenotypes
3. Discuss mechanical circulatory support

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Case

60M late-presenting anterior STEMI s/p successful LAD PCI

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- VS: HR 100, BP 85/70, O2 94% 2L NC
- Labs: lactate 3, Cr 1.5
- POCUS: severe anteroseptal/apical hypokinesis, EF ~30%, B-lines

Early recognition

Diagnostic Criteria

Hypotension
SBP <90 mmHg >30 min or vasoactive support

Hypoperfusion
AMS, AKI/oliguria, ALI, lactate >2 mmol/l

Cardiac Impairment

Low output: CI <2.2 L/kg/min

+

Congestion: PCWP >15 mmHg

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LV/RV, systolic/diastolic, valvular, pericardial, arrhythmic

+

Low output: CI <2.2 L/kg/min, SvO2 <60%, LVOT VTI <15 cm, pulse pressure <25 mmHg, cool extremities

+

Congestion: PCWP >15/RA >12 mmHg, POCUS (IVC, E/E', B-lines), JVD, pulmonary/peripheral edema

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***Don't be fooled by normal MAP or EF!**

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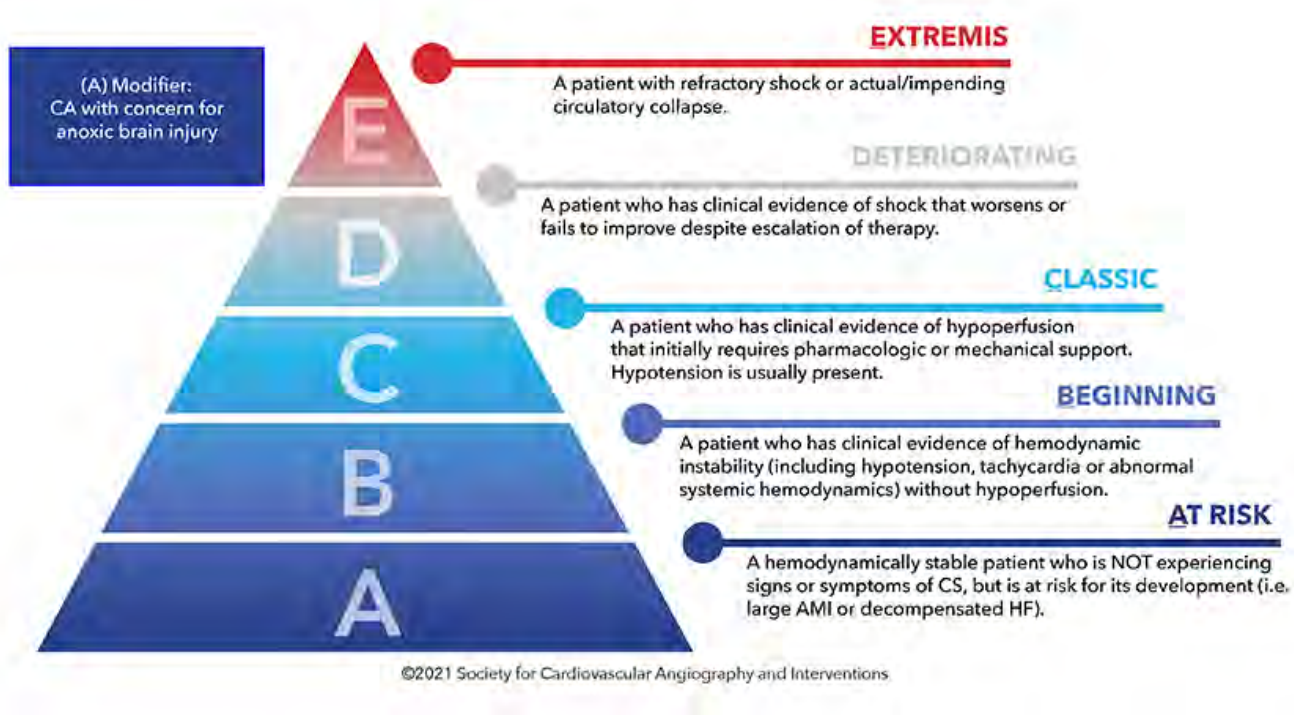
Prognostic Classification SCAI Stages



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- VS: HR 100, BP 85/70, O2 94% 2L NC
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=**SCAI C AMI-CS**

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Case

60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

- BP 85/70 = MAP 75
- If EF 60→30...SV 70→35, CO 5→2.5, MAP 80→40

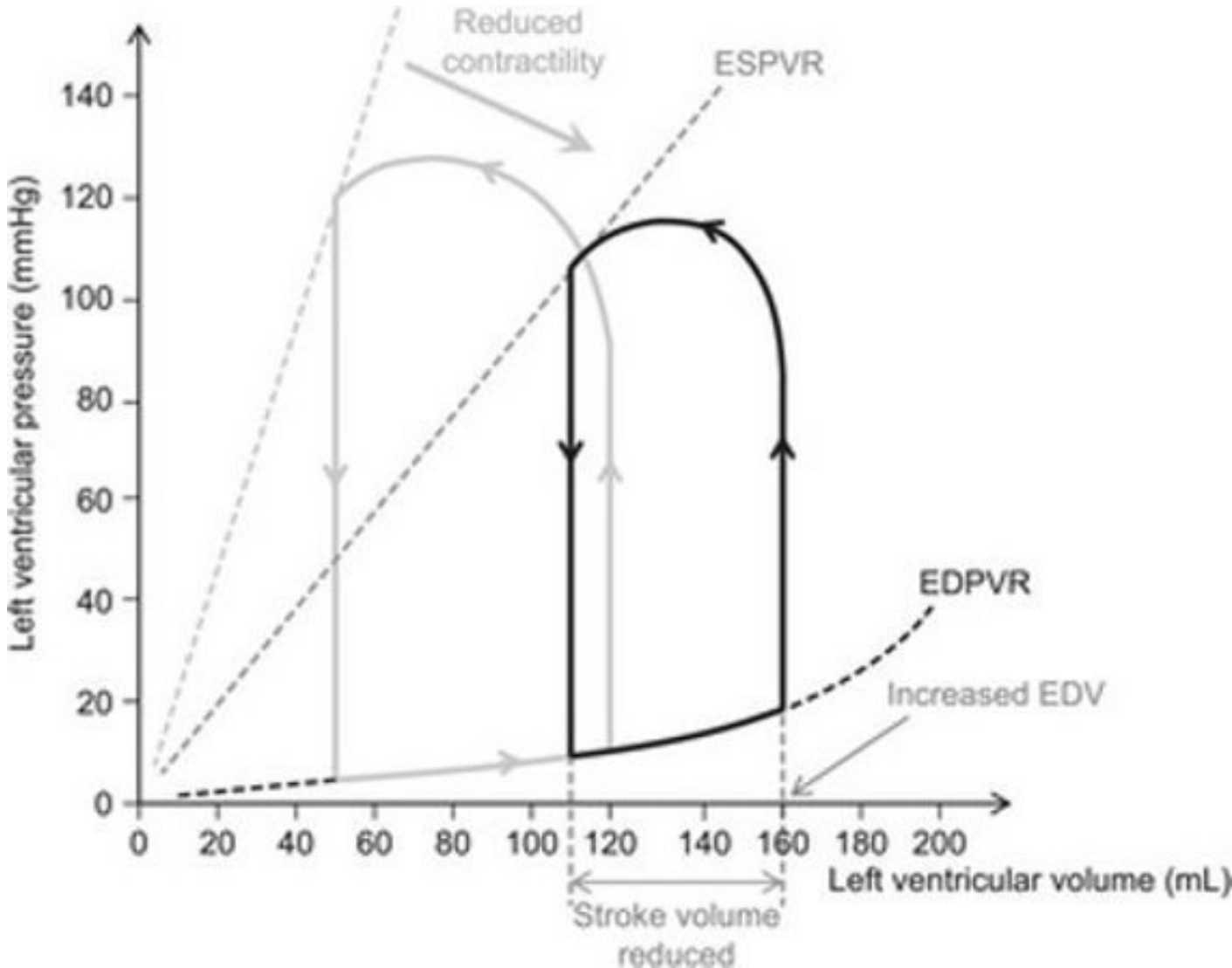
Why is MAP 75, not 40?

Compensatory mechanisms

1. Pathological remodeling
2. Neurohormonal activation

Compensatory mechanisms

1. Pathological remodeling
2. Neurohormonal activation



Compensatory mechanisms

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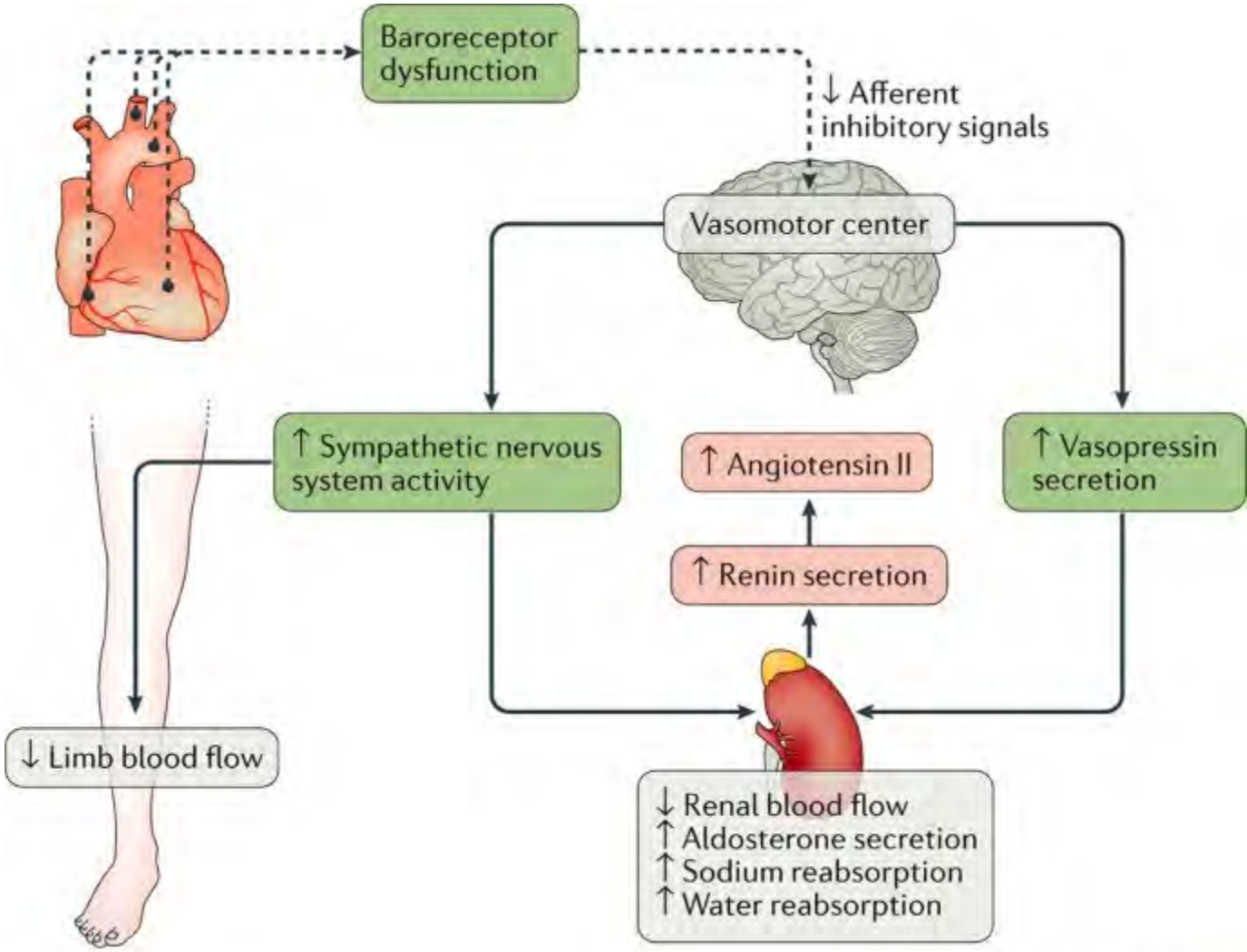

Case

60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

1. ***Too soon for structural remodeling!*
   - (eventually LVEDV 120→160 ml increases SV 35→45)

2. Neurohormonal compensation
   - HR 70→100 increases CO 2.5→3.5
   - SVR 1200→1700 restores MAP→75

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   - HR 70→100 increases CO 2.5→3.5
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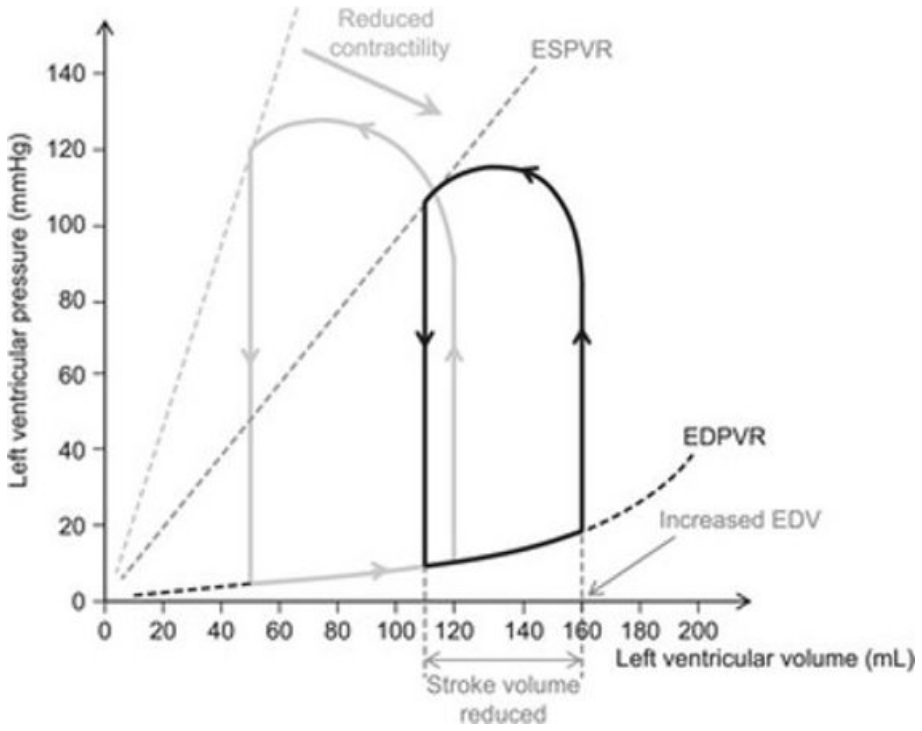
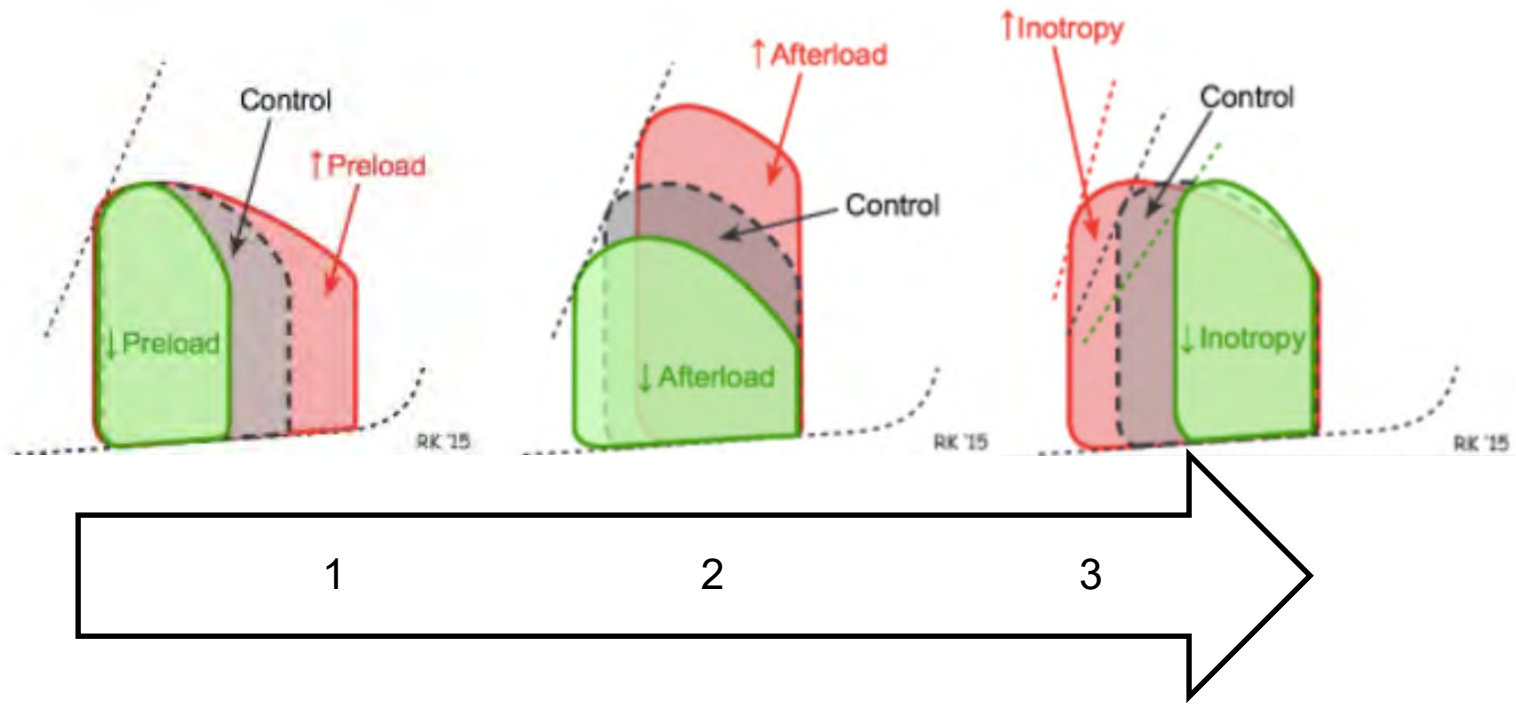
<u>At the cost of myocardial O2 demand & tissue perfusion</u>

Lecture Aims

1. Recognize cardiogenic shock earlier
2. Review management strategies for various cardiogenic shock phenotypes
3. Discuss mechanical circulatory support

Targets for vasoactive support





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Case

60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

- HR 100, BP 85/70 (MAP 75), CO/CI 3.5/1.8
- Lactate 3→4, Cr 1.5→2, no response to diuretics

What do you do?

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Evidence-based vasoactive support

1. SOAP II 2010
   - NE vs DA in 1,679 shock pts (62% septic, 17% CS, 16% hypovolemic)
   - No difference in 28-day mortality
   - Prespecified CS subgroup: DA ↑mortality (P=0.03) & arrhythmias (24 vs 12%)
2. Levy et al 2011
   - NE+Dob vs Epi in 30 refractory HF-CS pts targeting MAP 65-70
   - Similar ↑MAP & CI, but NE+Dob ↓HR, ↓arrhythmias, ↑UOP, ↓lactate
3. Levy et al 2014
   - NE vs Epi in 57 AMI-CS pts s/p PCI targeting MAP 65-70
   - Dob in 67% of both groups, much longer duration with NE
   - No difference in primary endpoint CO/CI (p=0.4)
   - Terminated early for ↑"refractory shock" in Epi arm driven by lactate & HR
   - No difference in other outcomes or perfusion markers (Cr, UOP, LFTs, Tn)
4. DOREMI 2021
   - Dob vs Mil in 192 CS pts
   - No difference in primary or any secondary endpoints
   - No difference in HR, arrhythmias, or hypotension

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(Not-so-)unique shock phenotypes

1. Classic CS
2. Normotensive CS
3. Vasodilatory/Mixed CS
4. RV shock
5. AS/LVOTO
6. MS

(Not-so-)unique shock phenotypes

Classic CS: SBP<90, CI <2.2, PCWP >15, SVR >1200

1. Restore perfusion pressure (MAP 65-75): NE reasonable 1st-line vasopressor
2. +Inotrope if ongoing hypoperfusion/CI <2.2: inodilator (Dob/Mil > Epi) often preferable

(Not-so-)unique shock phenotypes

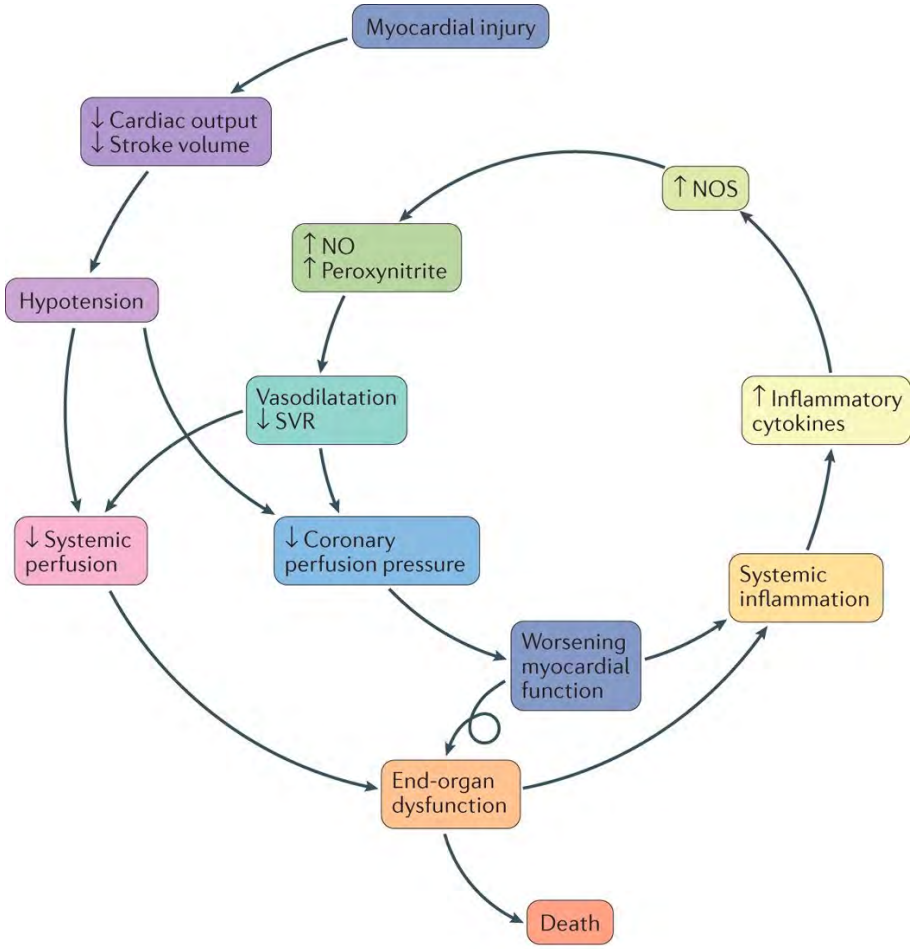
Normotensive CS: SBP >90, CI <2.2, PCWP >15, SVR >>1200

1. Reduce afterload: pure vasodilator reasonable if MAP >65-75
2. +Inodilator (Dob/Mil) if MAP 65-75 + ongoing hypoperfusion/CI <2.2

(Not-so-)unique shock phenotypes

Vasodilatory/Mixed CS: SBP <90, CI <2.2, PCWP >15, SVR <800

1. Restore perfusion pressure (MAP 65-75): NE reasonable 1st-line vasopressor
2. +Inotrope if ongoing hypoperfusion/CI <2.2: inodilator may not be tolerated, inoconstrictor often preferred (Epi > Dob/Mil) until vasodilatory component resolves



Reyentovich et al. 2016

(Not-so-)unique shock phenotypes

RV shock: CI <2.2, RAP >12-14, PCWP variable

1. Maintain preload: consider fluid boluses, dynamically assess responsiveness
2. Maintain systemic perfusion pressure (Epi, NE, Vaso)
3. Reduce RV afterload: consider inhaled pulmonary vasodilators (caution in group II PH), treat hypoxemia/acidemia
    - *"What's good for the lung tends to be good for the RV!"*

(Not-so-)unique shock phenotypes

RV shock: CI <2.2, RAP >12-14, PCWP variable

Positive Pressure Ventilation (BIPAP or MV): Only when unavoidable and proceed cautiously, especially in obstructive shock! Decreases RV preload (increases RAP but decreases VR) + increases RV afterload.

Consider the following:

1. Pre-induction
   - Start inoconstrictor (Epi, NE) or have available. Err on oversupporting the RV.
   - Preoxygenate with HFNC + inhaled pulmonary vasodilator
   - Consider small calcium boluses (~250-500 mg)
   - Consider preemptive femoral access for VA-ECMO, discuss with ECMO team
2. Induction/Intubation
   - Consider awake intubation while spontaneously breathing (especially in obstructive shock!)
   - Very gentle, often multimodal
   - Minimize BMV
3. Mechanical ventilation
   - PVR lowest @ FRC! Both atelectasis and overdistention increase PVR.
   - Primary predictors of acute cor pulmonale: PCO_2, Pplat, ΔP
   - Initiate PEEP ~5-8 & Vt ~6 ml/kg
   - Titrate to PCO_2 <60, Pplat <27, ΔP <17 if tolerated

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(Not-so-)unique shock phenotypes

AS/LVOTO: afterload-dependent and preload sensitive state

1. *Phenylephrine* is ideal to achieve our hemodynamic goals
    - Maintain coronary/peripheral perfusion (maintain BP)
    - Increase filling time/LVEDV/LVOT area to reduce dynamic LVOT gradient ("fixed" in AS)
    - Reduce myocardial work (slow HR, minimize dynamic LVOTO)
2. Maintain AV synchrony to optimize preload
3. Heart team evaluation for valve intervention! AS/LVOTO in HF or shock is a slippery slope. ("A mechanical problem needs a mechanical solution!")

AS/LVOTO + LV dysfunction: highly recommend PAC-guided inotropic support

(Not-so-)unique shock phenotypes

MS: preload-dependent state.

Hemodynamic goals:

1. Reduce HR to maximize filling time (metoprolol, esmolol, digoxin, ivabradine (off-label), avoid chronotropy)
2. Maintain sinus rhythm and AV synchrony to maximize LV filling (amiodarone if needed)
3. Heart team evaluation for valve intervention ("A mechanical problem needs a mechanical solution!")

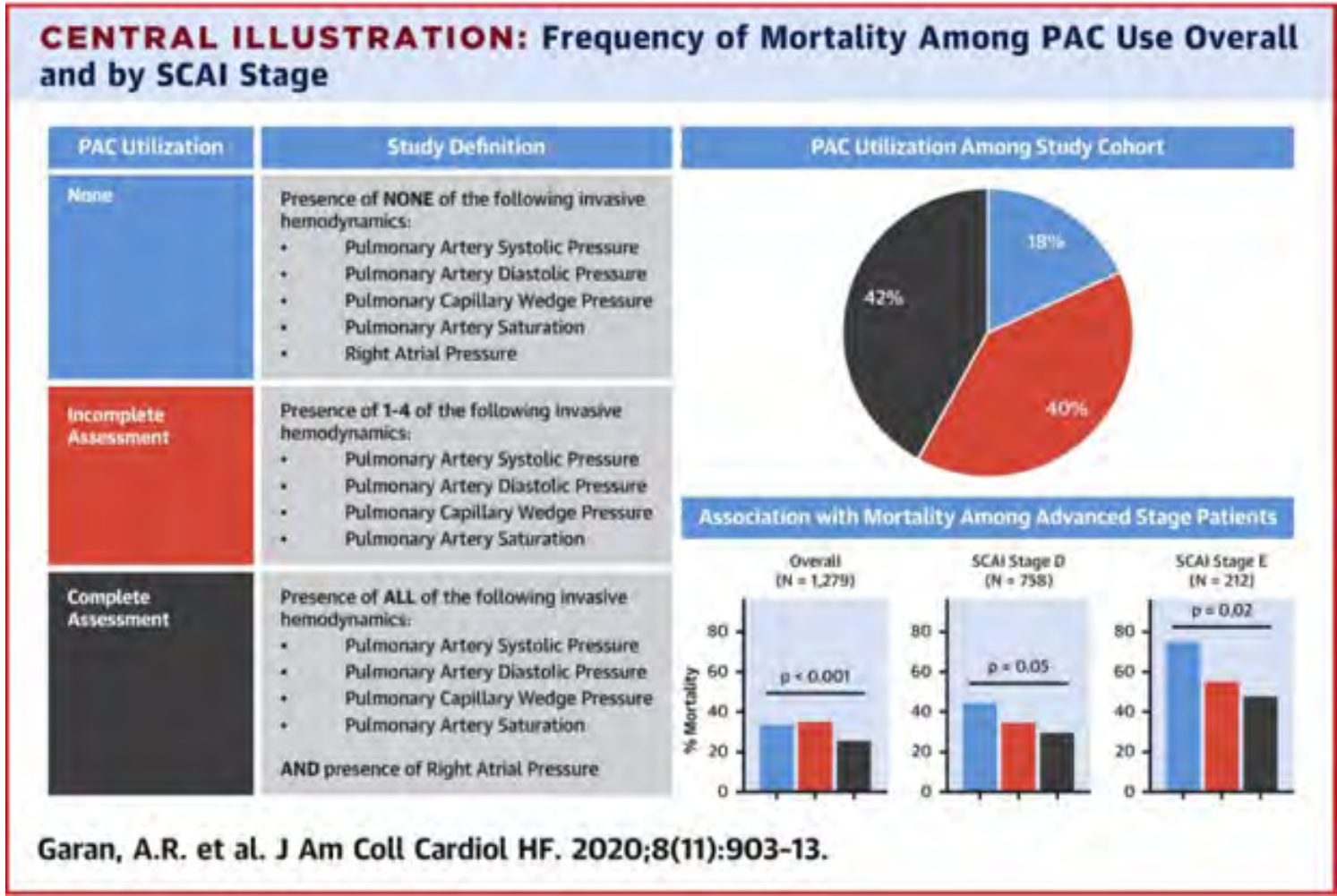
MS + RV shock: highly recommend PAC-guided inotropic support. Often requires a combination of agents to slow HR and support RV without negative inotropes.

A plug for invasive hemodynamics

Complete Hemodynamic Profiling With Pulmonary Artery Catheters in Cardiogenic Shock Is Associated With Lower In-Hospital Mortality: CSWG Registry

1,414 CS pts across 8 tertiary CICUs

Complete hemodynamic assessment (RAP, PAs, PAd, PCWP, MvO2) associated with lower mortality across all SCAI stages of CS severity



Case

60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

- HR 100, BP 85/70 (MAP 75), CO/CI 3.5/1.8
- Lactate 3→4, Cr 1.5→2, no response to diuretics

What do you do?

- Escalate diuretics? Ultrafiltrate?
- Add norepinephrine? Add clevidipine? MAP goal?
- Add an inotrope?

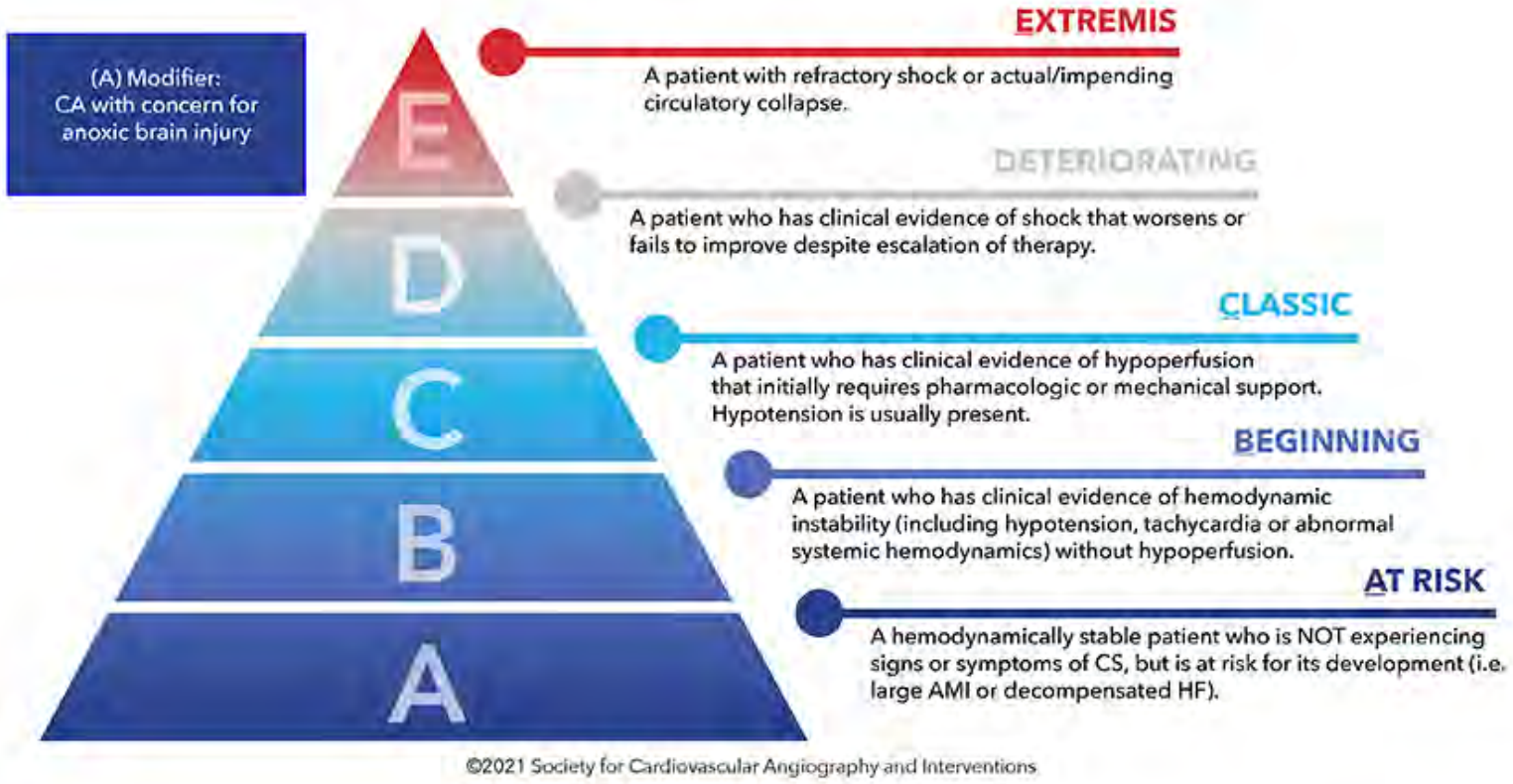
Case

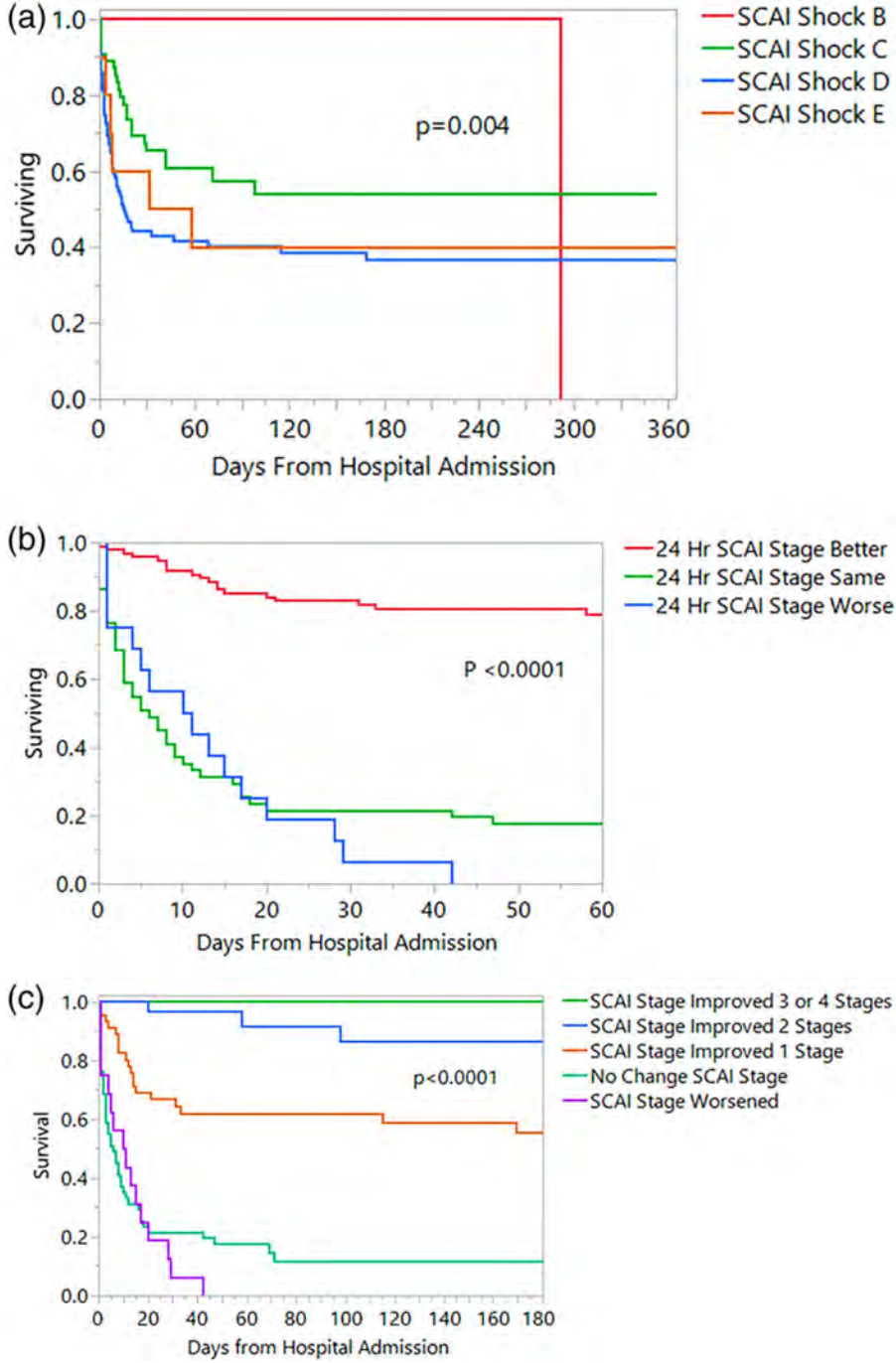
60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

- HR 100, BP 85/70 (MAP 75), CO/CI 3.5/1.8
- Lactate 3→4, Cr 1.5→2, no response to diuretics

What do you do?

- Dobutamine 2.5→5 mcg/kg/min
- Lactate and Cr stabilize at 3 and 2, UOP ~20 ml/h





Baran et al. 2020

Lecture Aims

1. Recognize cardiogenic shock earlier
2. Review management strategies for various cardiogenic shock phenotypes
3. Discuss mechanical circulatory support

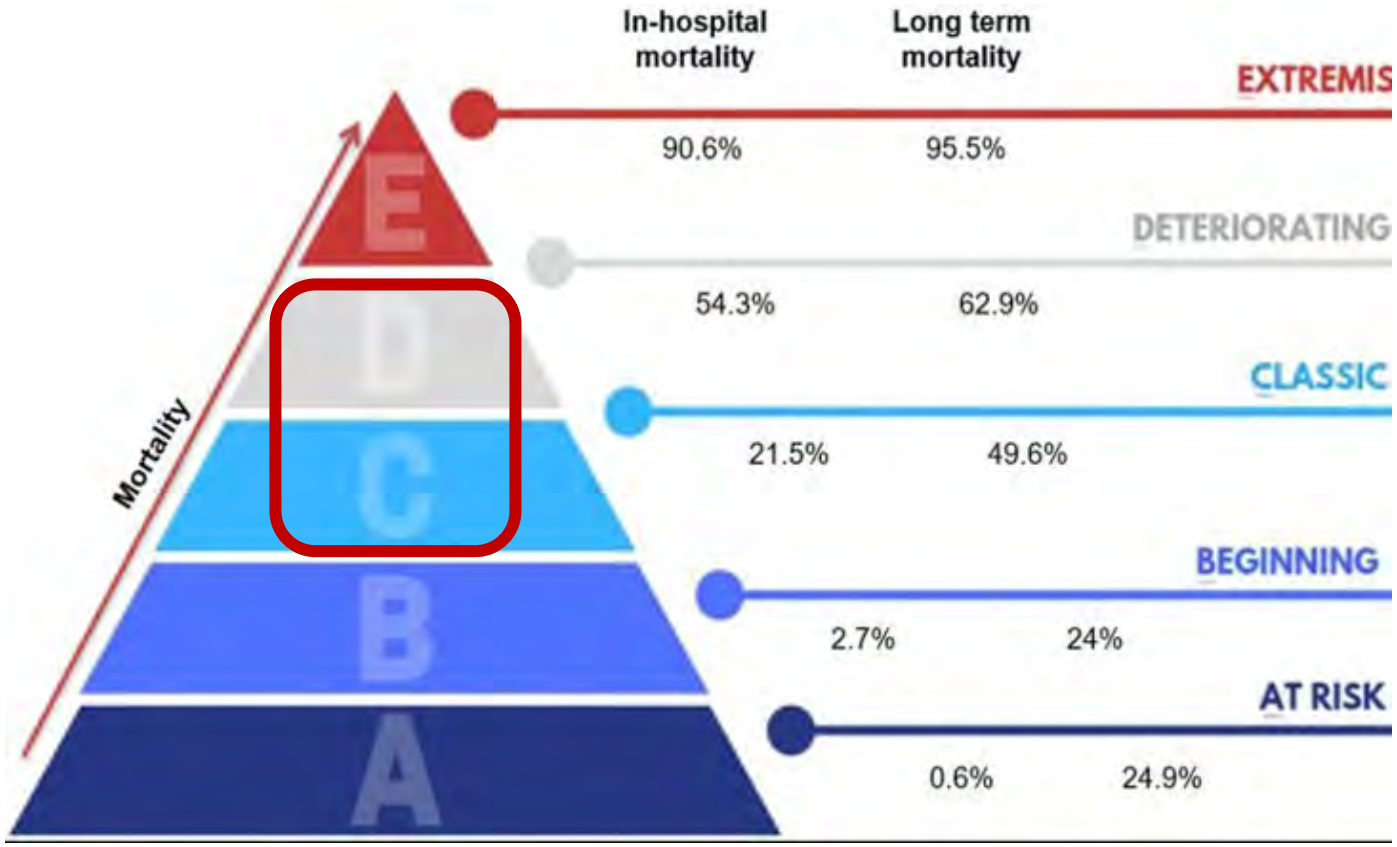
Mechanical circulatory support

Acute/temporary MCS goals:

1. Improve organ perfusion
2. Reduce congestion
3. Reduce myocardial demand
4. Facilitate recovery or bridge to transplant/VAD

Mechanical circulatory support

Timing is critical!



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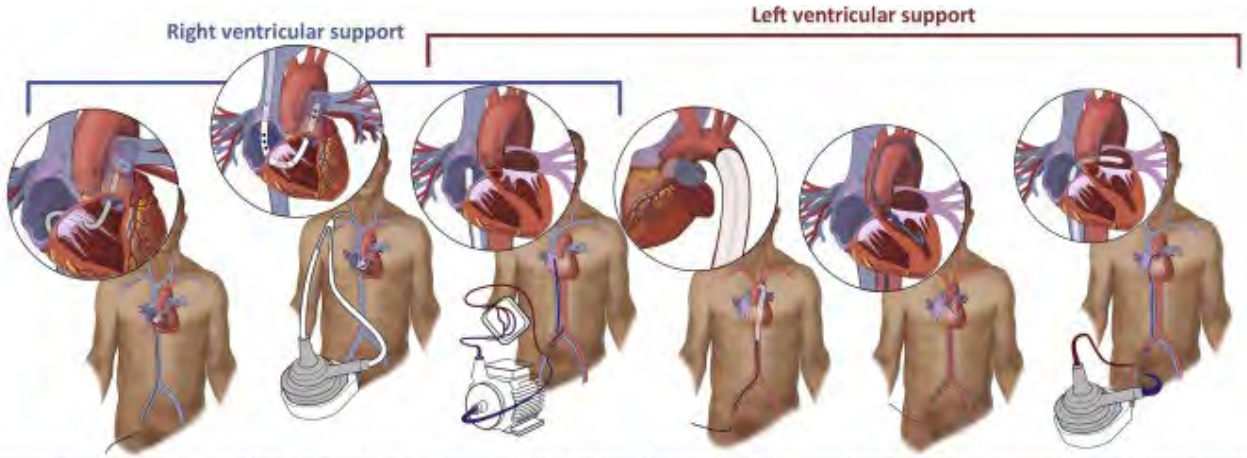
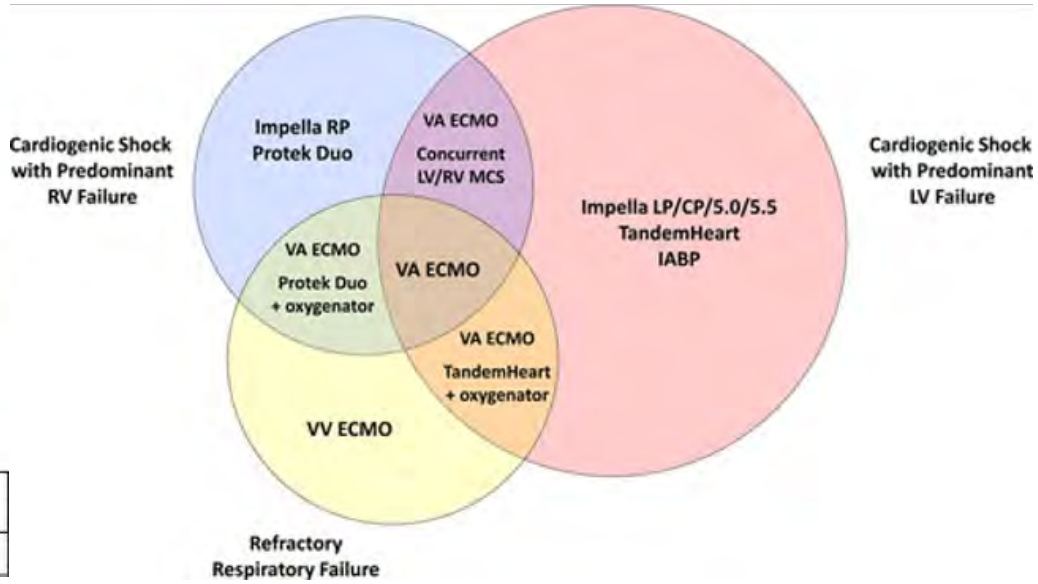
Mechanical circulatory support

Support strategy?



	Impella RP	TandemHeart RA-PA	VA-ECMO	IABP	Impella (2.5, CP, 5.0, 5.5)	TandemHeart LA-FA
Flow	max 4.0 l/min	max 4.0 l/min	max 7.0 l/min	0.5 l/min	2.5 - 5.5 l/min	max 4.0 l/min



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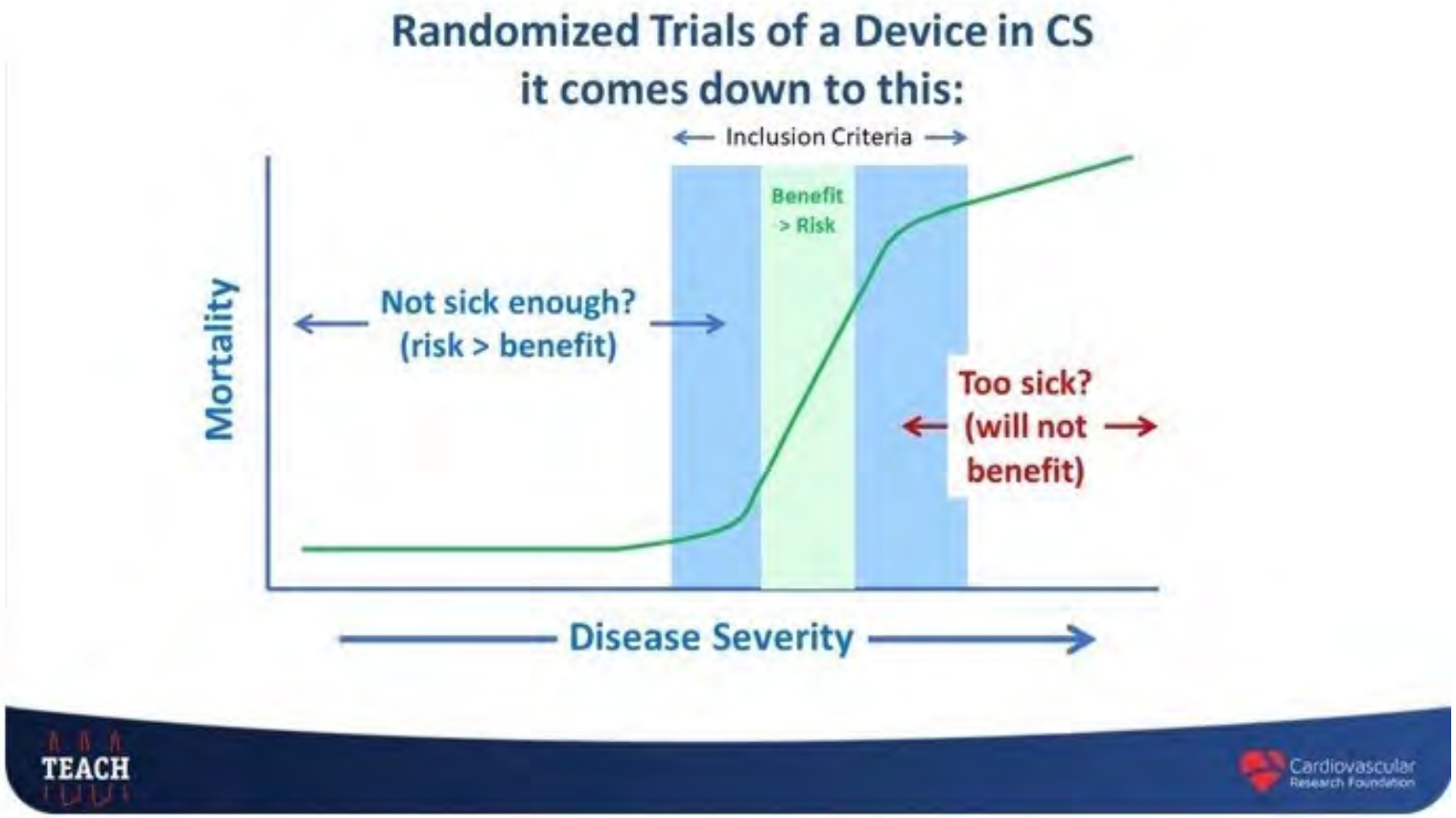
Mechanical circulatory support



Shashank Sinha, Jason Katz, Devesh Rai

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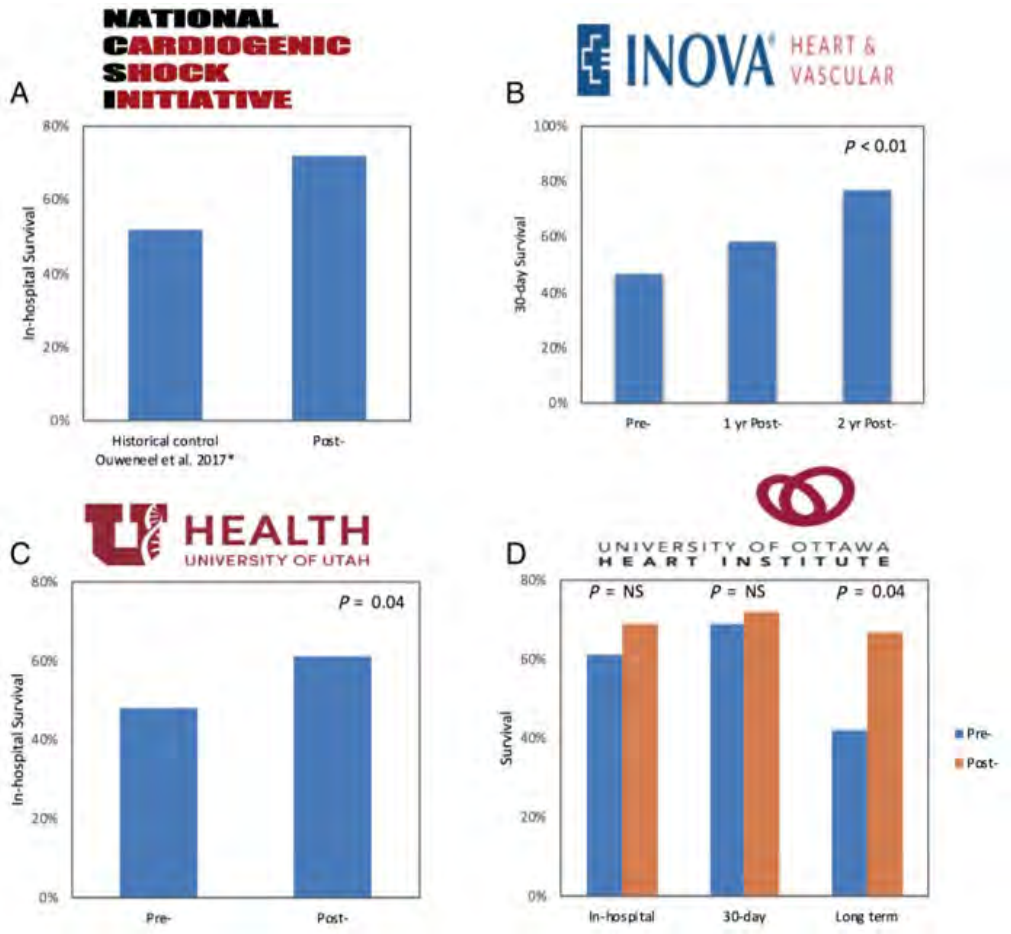
Multidisciplinary shock teams

Cardiogenic shock teams and centres:
A contemporary review of multidisciplinary care for cardiogenic shock
Moghaddam et al, ESC Heart Failure2021;8: 988–998



4 single-center before-and-after studies

Shock Teams associated with reduced mortality

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Case

60M late-presenting anterior STEMI s/p successful LAD PCI in AMI-CS

- SCAI C AMI-CS refractory to inotropic support

What do you do?

- Impella 5.5 via axillary graft, dobutamine weaned off to rest the LV
- Lactate cleared, UOP increased, Cr returned to baseline
- LV function improved over several days
- Impella removed with stable hemodynamics and perfusion markers
- Successful bridge to recovery

Summary

1. Recognize cardiogenic shock earlier
2. Optimize loading conditions (preload, afterload/perfusion pressure)
3. Add inotropic support for persistent hypoperfusion
4. In refractory shock or with complex hemodynamics:
    - Engage multidisciplinary heart team
    - Expedite invasive hemodynamics to guide pharmacologic therapy and consideration of mechanical support

Thank you!

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Cardiac Arrest and Resuscitation

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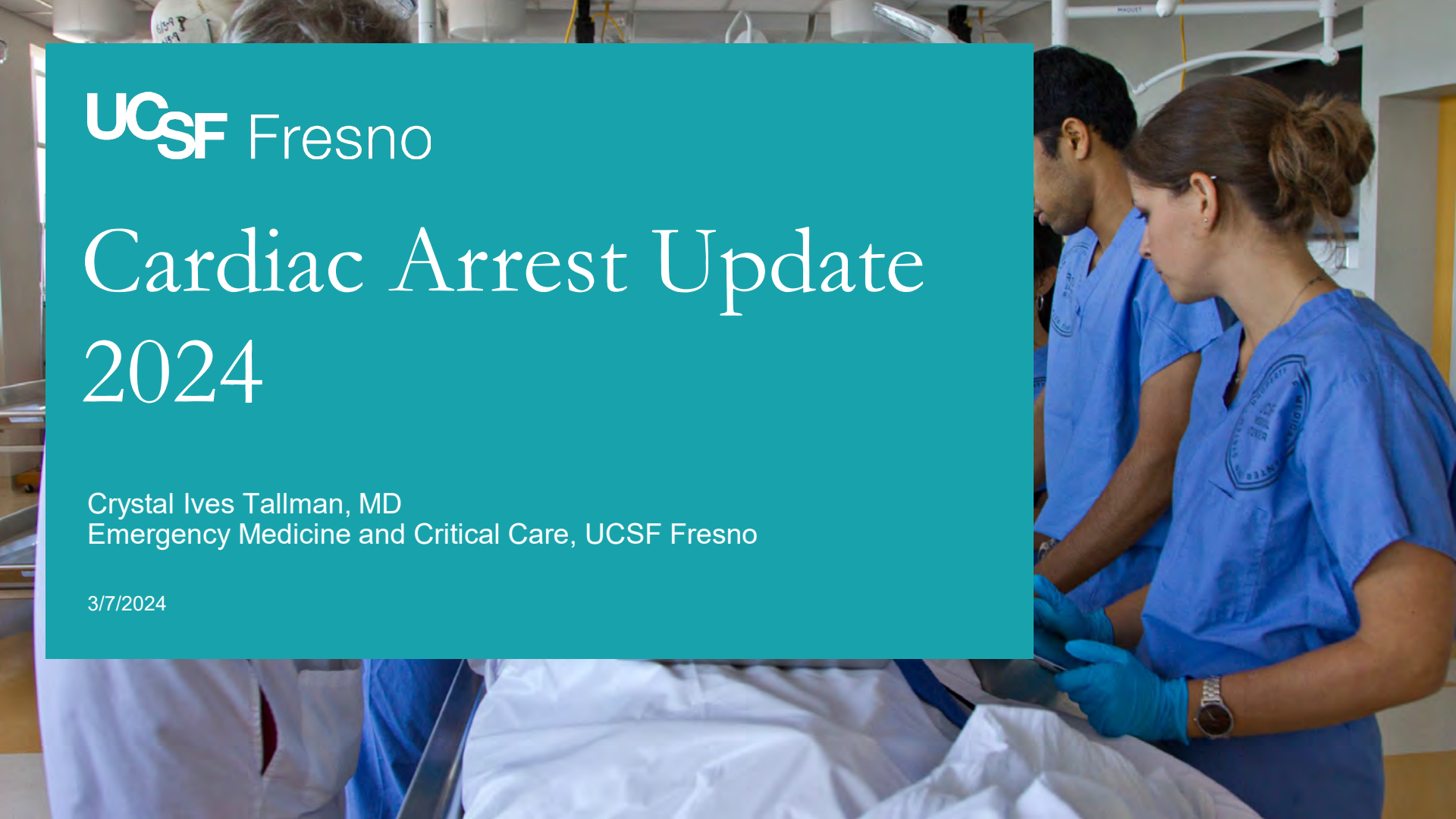


UCSF Fresno

Cardiac Arrest Update 2024

Crystal Ives Tallman, MD
Emergency Medicine and Critical Care, UCSF Fresno

3/7/2024

Financial Disclosures

- No relevant disclosures
- I will discuss off-label and investigational use of drugs or devices

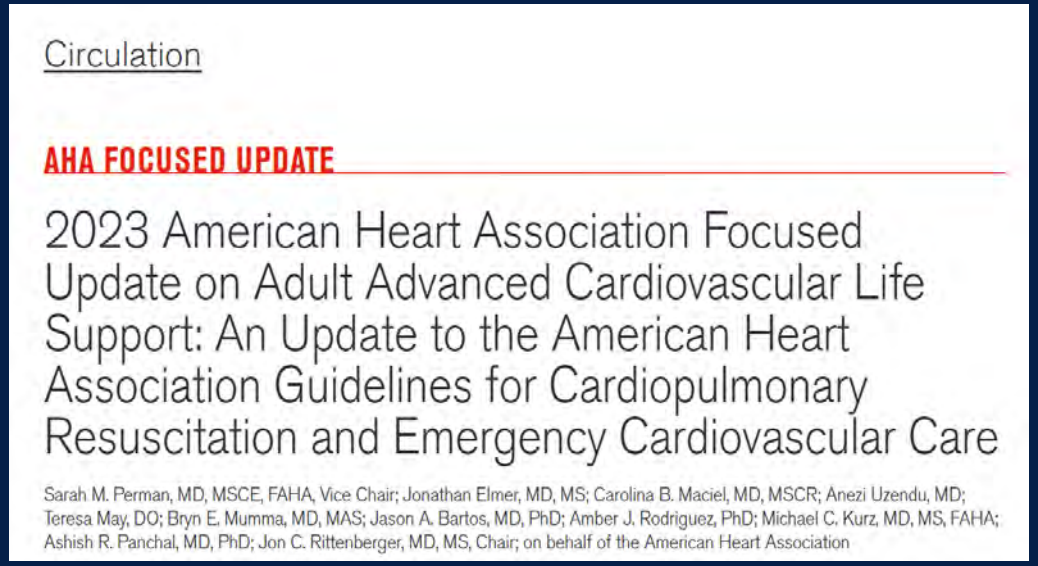
Overview

- Tools to optimize cardiopulmonary resuscitation → “cardiocerebral resuscitation”
- Goal directed use of intra-arrest medications – epinephrine and calcium
- Evidentiary basis behind consideration of ECPR

Cardiocerebral Resuscitation

What leads to neurologically intact survival?

- Early recognition of cardiac arrest and bystander CPR
- High-quality, minimally interrupted chest compressions
- Early defibrillation



Circulation

AHA FOCUSED UPDATE

2023 American Heart Association Focused Update on Adult Advanced Cardiovascular Life Support: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Sarah M. Perman, MD, MSCE, FAHA, Vice Chair; Jonathan Elmer, MD, MS; Carolina B. Maciel, MD, MSCR; Anezi Uzendu, MD; Teresa May, DO; Bryn E. Mumma, MD, MAS; Jason A. Bartos, MD, PhD; Amber J. Rodriguez, PhD; Michael C. Kurz, MD, MS, FAHA; Ashish R. Panchal, MD, PhD; Jon C. Rittenberger, MD, MS, Chair; on behalf of the American Heart Association

- No routine calcium (B - R)
- ECPR is reasonable in a system that is equipped to do it (B - R)
- Emergency angiography is recommended only if STEMI, electrical instability, signs of ongoing cardiac ischemia, cardiogenic shock (B – NR)
- Deliberate strategy for temperature control (32˚- 37.5˚C) comatose patients post-ROSC (B – R)
- Trial AED for patients on ictal-interictal continuum (C – EO)

PMID: 38108133

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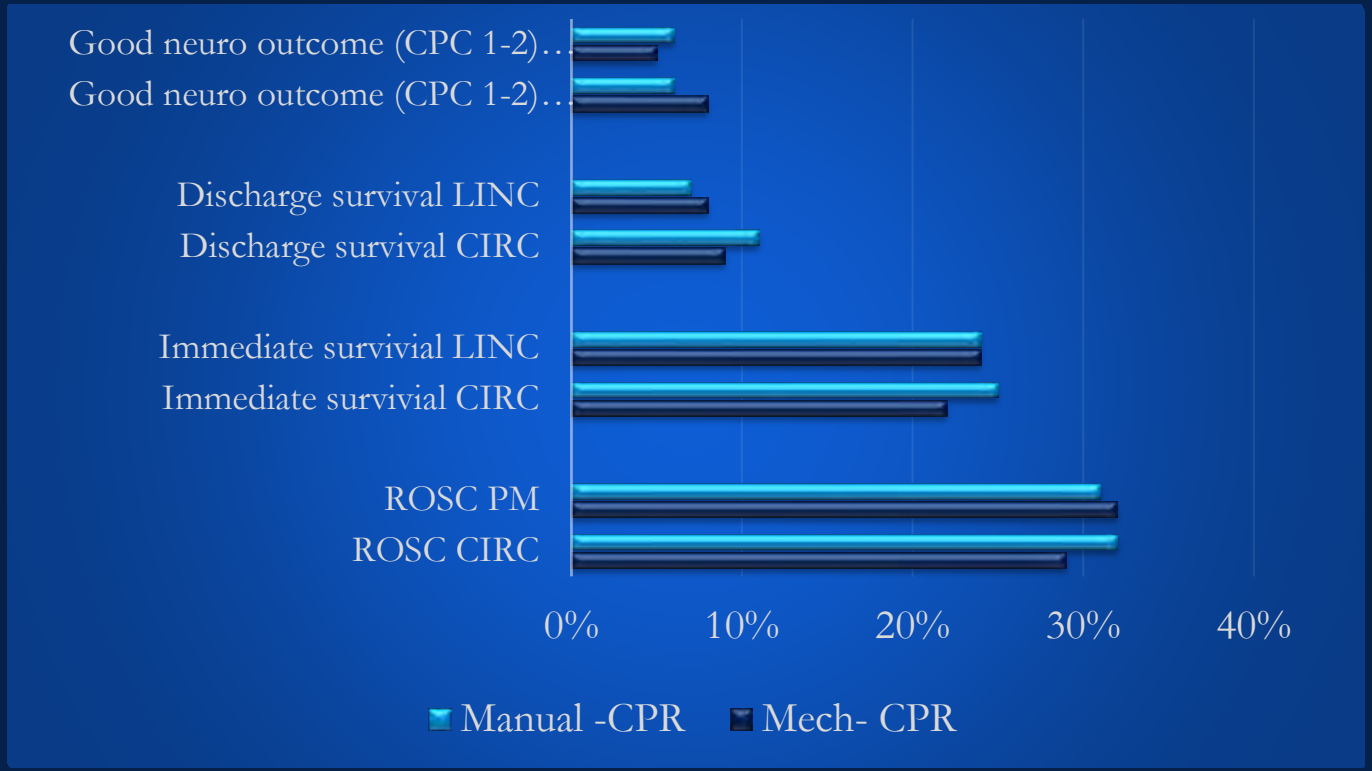
CPR

High-quality, minimally-interrupted chest compressions are critical

- Mechanical vs Manual
- ACE-CPR
- What do we monitor to ensure high-quality CPR?
  - Capnography
  - POCUS
  - Intra-arrest TEE

Mechanical vs Manual



- OHCA – ***multiple RCTs showing similar outcomes***

PMID: 24642406; PMID:25467566; PMID: 24240611; PMID: 30125048

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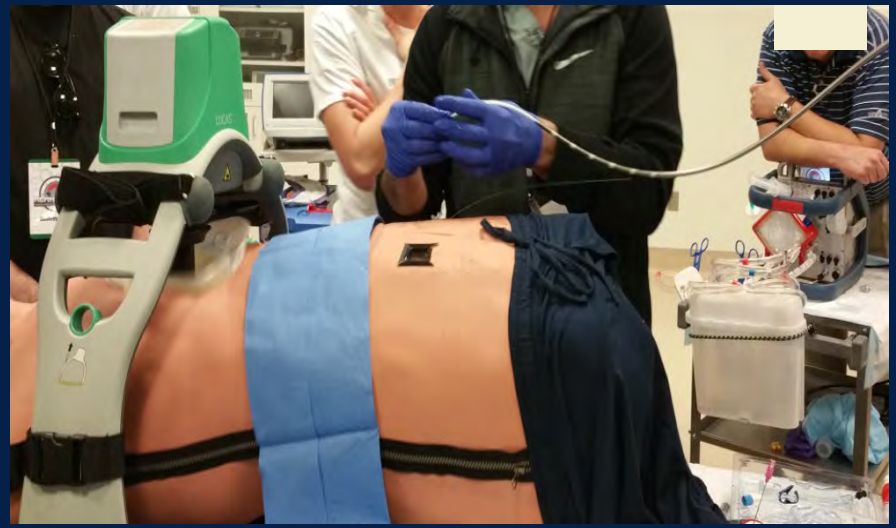
Mechanical vs Manual



- Negatives
  - Delay in CPR initiation/no-flow time
  - Delay to defibrillation
  - Poor positioning
- When to use
  - During transport
  - ECPR
  - Longer continuous compressions – lytics for PE or MI, hypothermia

PMID: 29843753

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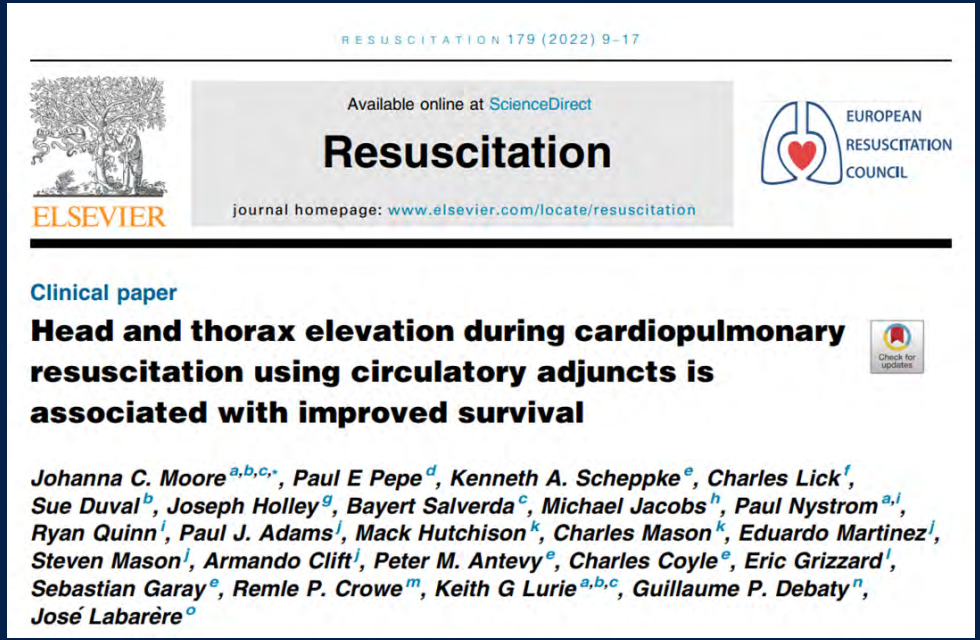
ACE-CPR

- "Automated controlled elevation" CPR – combination of gradual head and thorax elevation, active compressions/decompressions and an impedance threshold device
- "Heads up" CPR



RESUSCITATION 179 (2022) 9–17

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Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

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Clinical paper

Head and thorax elevation during cardiopulmonary resuscitation using circulatory adjuncts is associated with improved survival

Johanna C. Moore[a,b,c,], Paul E Pepe[d], Kenneth A. Scheppke[e], Charles Lick[f], Sue Duval[b], Joseph Holley[g], Bayert Salverda[c], Michael Jacobs[h], Paul Nystrom[a,i], Ryan Quinn[i], Paul J. Adams[j], Mack Hutchison[k], Charles Mason[k], Eduardo Martinez[l], Steven Mason[l], Armando Clift[l], Peter M. Antevy[e], Charles Coyle[e], Eric Grizzard[l], Sebastian Garay[e], Remle P. Crowe[m], Keith G Lurie[a,b,c], Guillaume P. Debaty[n], José Labarère[o]*

Does ACE-CPR improve survival compared to conventional CPR in OHCA?

PMID: 35933057

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ACE-CPR

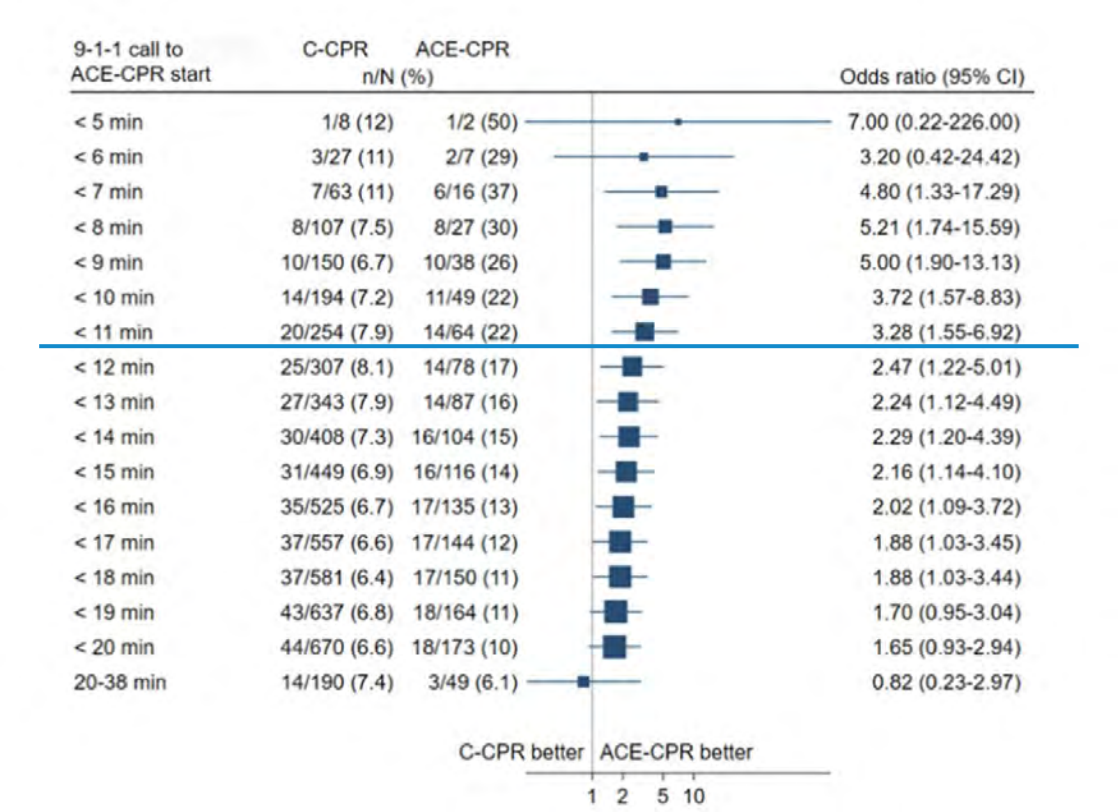
- Raising the head gradually improves venous drainage, lowering intracranial pressure
- Paired with active compression/decompression and/or impedance threshold device to help increase venous return to the heart
- In swine and human cadaver models improves cerebral perfusion pressure

Image from Moore et al. Resuscitation 2022 PMID 35933057

PMID: 35933057; PMID: 29702188; PMID: 30005978

ACE-CPR

- 222 patients received ACE-CPR, matched 4:1 with 860 controls
- 21 **(9.5%)** ACE-CPR patients vs 58 **(6.7%)** controls survived to hospital discharge (OR) 1.44 [0.86 to 2.44]



9-1-1 call to ACE-CPR start	C-CPR n/N (%)	ACE-CPR n/N (%)	Odds ratio (95% CI)
< 5 min	1/8 (12)	1/2 (50)	7.00 (0.22-226.00)
< 6 min	3/27 (11)	2/7 (29)	3.20 (0.42-24.42)
< 7 min	7/63 (11)	6/16 (37)	4.80 (1.33-17.29)
< 8 min	8/107 (7.5)	8/27 (30)	5.21 (1.74-15.59)
< 9 min	10/150 (6.7)	10/38 (26)	5.00 (1.90-13.13)
< 10 min	14/194 (7.2)	11/49 (22)	3.72 (1.57-8.83)
< 11 min	20/254 (7.9)	14/64 (22)	3.28 (1.55-6.92)
< 12 min	25/307 (8.1)	14/78 (17)	2.47 (1.22-5.01)
< 13 min	27/343 (7.9)	14/87 (16)	2.24 (1.12-4.49)
< 14 min	30/408 (7.3)	16/104 (15)	2.29 (1.20-4.39)
< 15 min	31/449 (6.9)	16/116 (14)	2.16 (1.14-4.10)
< 16 min	35/525 (6.7)	17/135 (13)	2.02 (1.09-3.72)
< 17 min	37/557 (6.6)	17/144 (12)	1.88 (1.03-3.45)
< 18 min	37/581 (6.4)	17/150 (11)	1.88 (1.03-3.44)
< 19 min	43/637 (6.8)	18/164 (11)	1.70 (0.95-3.04)
< 20 min	44/670 (6.6)	18/173 (10)	1.65 (0.93-2.94)
20-38 min	14/190 (7.4)	3/49 (6.1)	0.82 (0.23-2.97)

Table from Moore et al. Resuscitation 2022 PMID 35933057

PMID: 35933057

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ACE-CPR

- **No clear benefit** to ACE-CPR compared with C-CPR
- **Exploratory analysis is hypothesis-generating for future randomized controlled trials**
- **Should not affect the current clinical practice**

PMID: 36681427

Ensuring High Quality CPR

Keys to performing High Quality CPR:

- Adequate compression depth
- Compression rate (100-120 cpm)
- Allowing full chest recoil
- Minimizing interruptions from pulse checks, ultrasound, defibrillation

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PMID: 23801105

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Ensuring High Quality CPR – Arterial Doppler

- Arterial doppler US of the femoral artery is **more accurate** than a manual pulse check to detect ROSC
  - 95% vs 54%
  - PSV > 20 cm/s ~ SBP > 60 mmHg

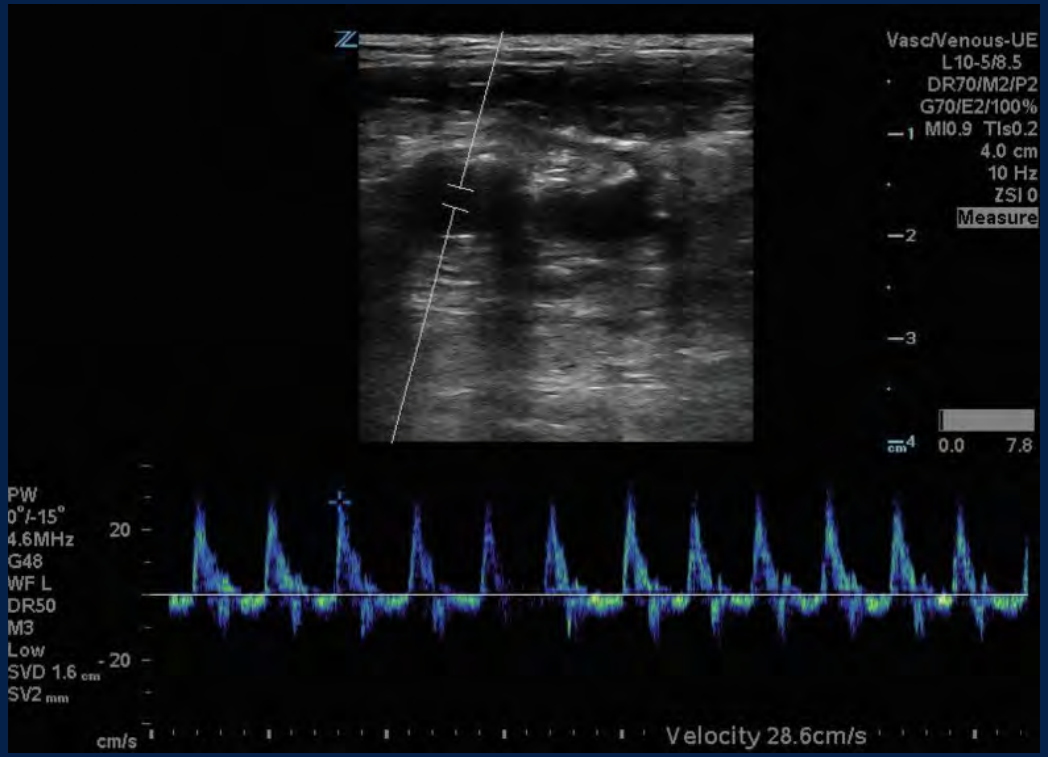

Image from Cohen et al. Resuscitation 2022 PMID: 35131404

PMID: 35131404; PMID: 36646373

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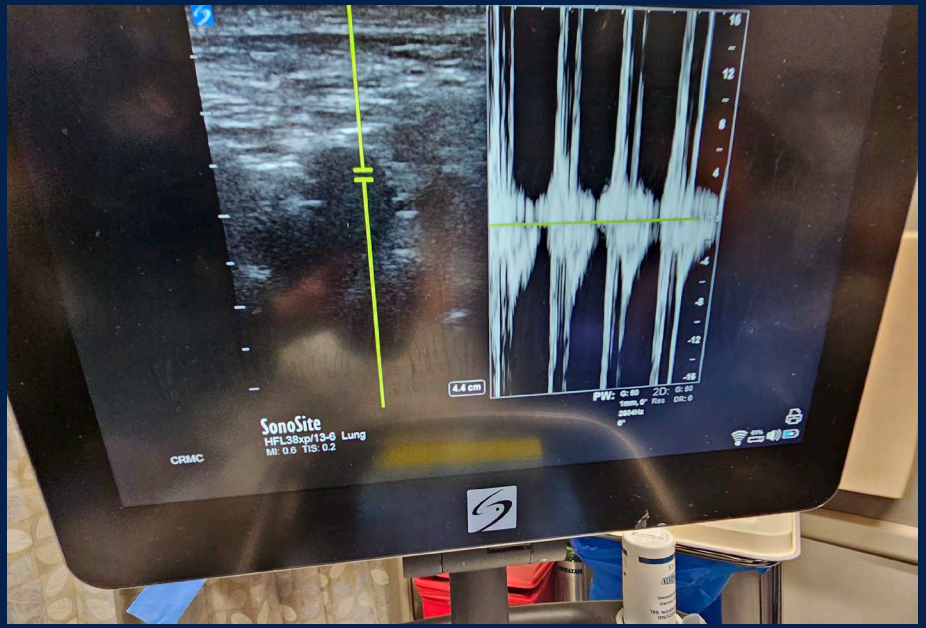
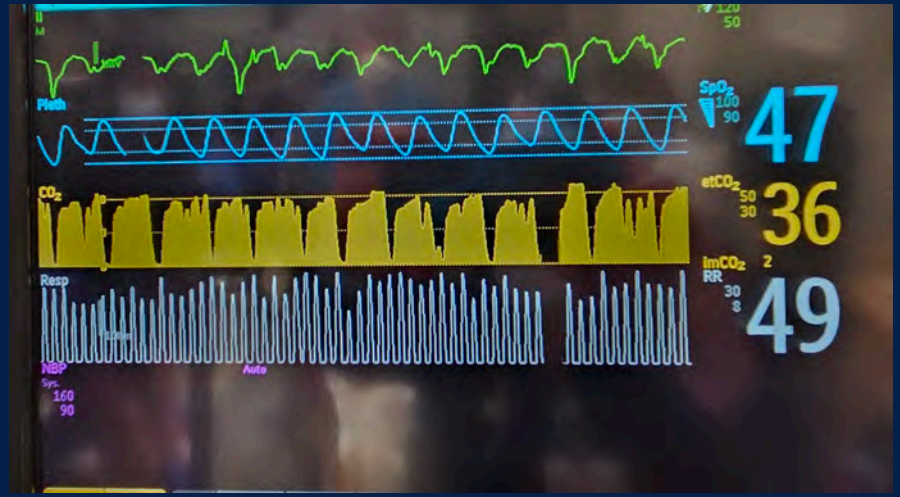

SonoSite
HFL38xp/13-6 Lung
MI: 0.6 TIS: 0.2
CRMC
4.4 cm
PW:


Pleth
CO2
Resp
NBP
Sys
160
90
SpO2
100
90
47
etCO2
50
30
36
imCO2
RR
30
8
49

Ensuring High Quality CPR – Capnography

Capnography during cardiac arrest is an **indicator of CPR quality**

- Capnography is an **indirect measure of pulmonary circulation** and correlates with cardiac index, coronary perfusion pressure and cerebral blood flow
- **Target ETCO2 > 20 mmHg**

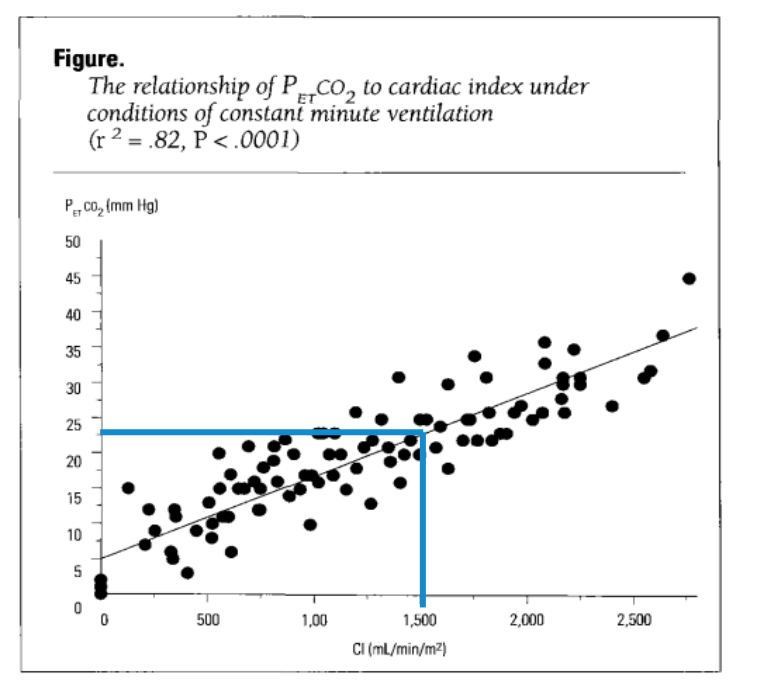


Figure from Idris et al. Ann Emer. Med 1994 PMID: 8135436

PMID: 30142399; PMID: 23801105; PMID: 9233867; PMID: 8135436; PMID: 29217394

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Ensuring High Quality CPR – TEE

Standard hand positioning may compress over the LVOT, impeding forward flow, in over 50% of adults.

- In a retrospective cohort study of patients undergoing ECPR, TEE showing an LVOT that was open during compressions was associated with more successful resuscitation.
- In a swine model, compressing over the LV was associated with better intra-resuscitation hemodynamics than compressions over the LVOT.

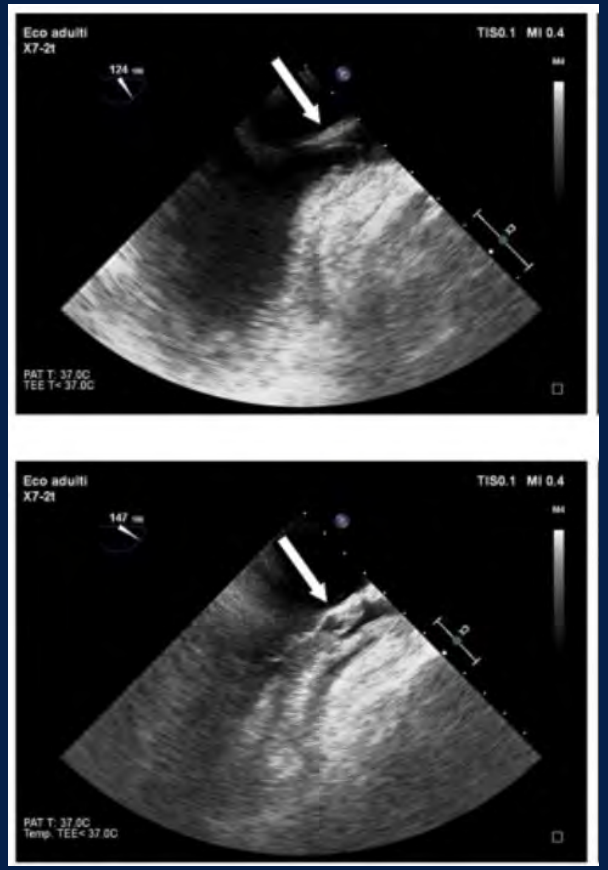

Image from Catena et al. Resuscitation 2019 PMID 30825552

PMID: 37598201; PMID: 30825552; PMID: 37598201

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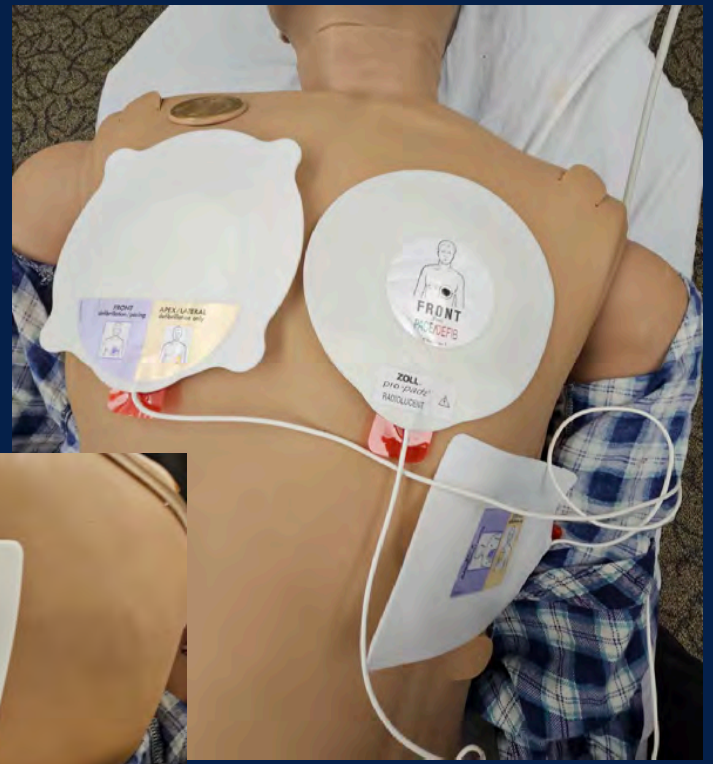
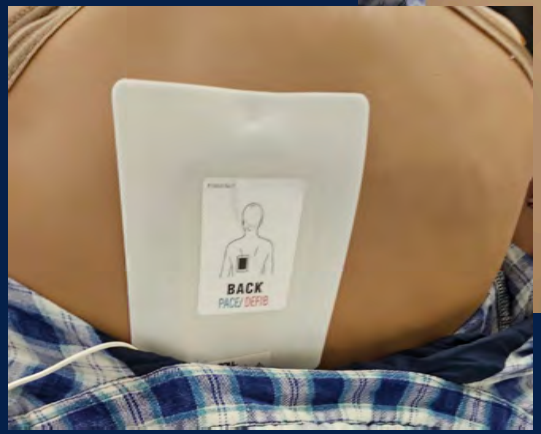
Defibrillation--Refractory VT/VF

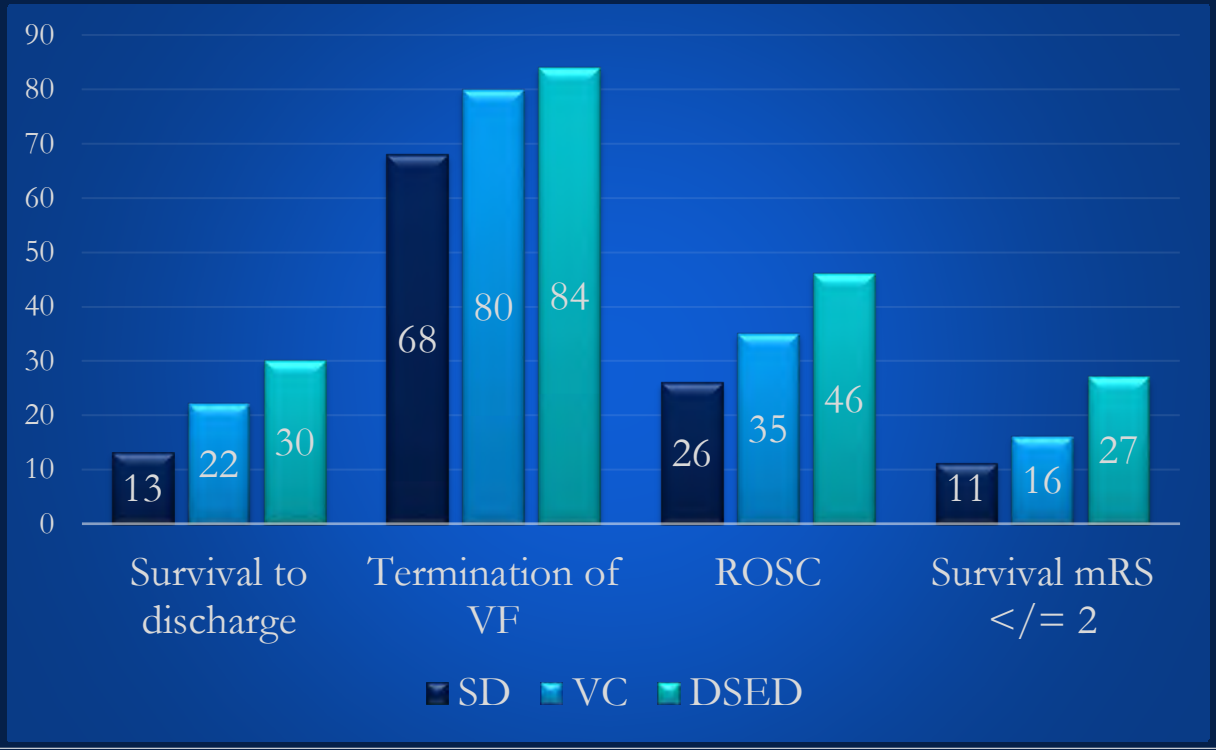
- Dual sequential external defibrillation – DOSE VF





PMID: 36342151; PMID: 32192760; PMID: 31790759

Refractory VT/VF – DOSE VF



PMID: 36342151

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Intra-arrest medications

Goal-directed use of medications intra-arrest

- Epinephrine
- Calcium

Epinephrine

- What is our goal with intra-arrest vasopressors?
- When is epinephrine most effective?

Vasopressor Management in Cardiac Arrest

COR	LOE	Recommendations
1	B-R	1. We recommend that epinephrine be administered for patients in cardiac arrest.
2a	B-R	2. It is reasonable to administer epinephrine 1 mg every 3 to 5 minutes for cardiac arrest.
2a	C-LD	3. With respect to timing, for cardiac arrest with a nonshockable rhythm, it is reasonable to administer epinephrine as soon as feasible.
2b	B-R	4. Vasopressin alone or vasopressin+ methylprednisolone in combination with epinephrine may be considered in cardiac arrest but offers no advantage as a substitute for epinephrine.
2b	C-LD	5. With respect to timing, for cardiac arrest with a shockable rhythm, it may be reasonable to administer epinephrine after initial defibrillation attempts have failed.
3: No Benefit	B-R	6. High-dose epinephrine is not recommended for routine use in cardiac arrest.

Table from Perman et al. Circulation 2023 PMID 38108133

Epinephrine

Epinephrine helps achieve ROSC

- PARAMEDIC2 trial, 5 NHS ambulance services in the UK, epi vs placebo
- Improves ROSC, survival to admission
  - **Overall, very little impact on survival with good neurologic outcome**
- Early recognition of arrest (NNT 11)
- Bystander CPR (NNT 15)
- Early defibrillation (NNT 5)

36% vs 12%
NNT = 4

24% vs 8%
NNT = 6

3.2% vs 2.3%
NNT = 111

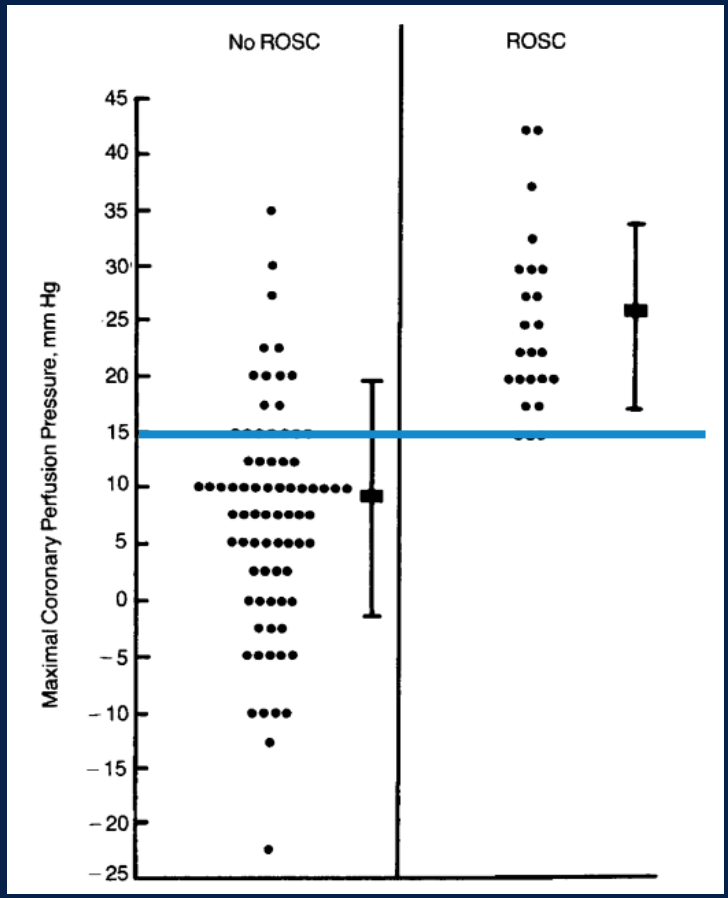
2.2% vs 1.9%

PMID: 30021076; PMID 36864469

Epinephrine

Goal is a coronary perfusion pressure > 15 mmHg

- Only patients with maximal CPP at least 15 mmHg achieved ROSC
- This may be the “critical level” of coronary blood flow necessary to achieve ROSC



Graph from Paradis et al JAMA 1990 PMID 2386557

Epinephrine

How do we ensure a CPP of > 15 mmHg?

- **Target diastolic BP > 25-35 mmHg**
  - Role for intra-arrest arterial line
    - Careful where measuring

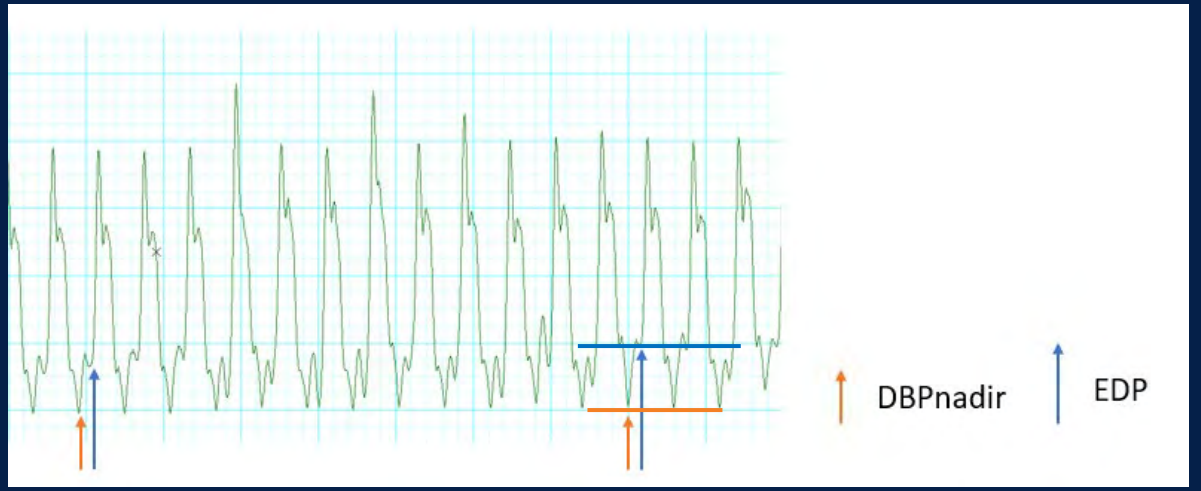


Image from Berve et al Resuscitation 2022 PMID 34710550

PMID: 23801105; PMID: 34710550

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Epinephrine

Epinephrine is **overall best in the first 20 minutes of arrest**

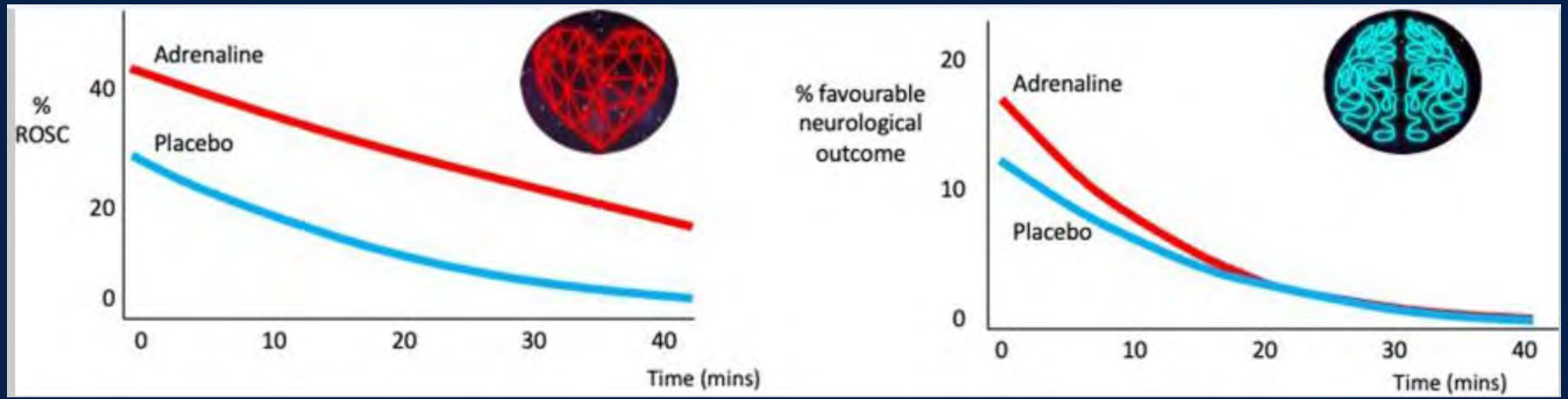

Figure from Perkins et al Crit Care 2023 PMID 36864469

PMID 36864469; PMID: 24846323

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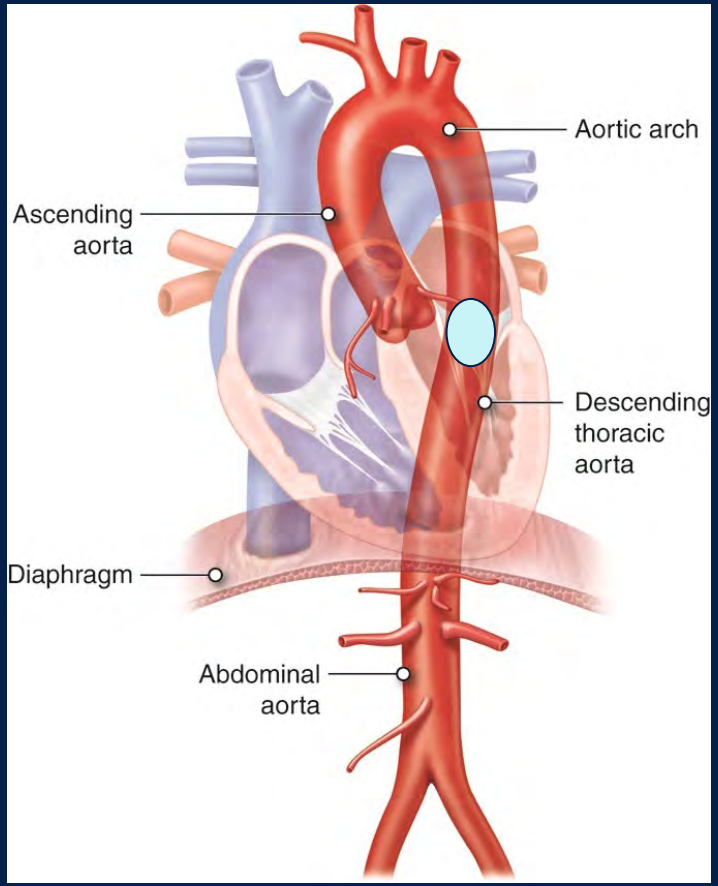
REBOA for Cardiac Arrest

REBOA may improve coronary and cerebral perfusion pressure in cardiac arrest

- In a pilot study (n=15, OHCA), REBOA increased coronary perfusion pressure (median 13.5 to 25.2)
- In pilot study, REBOA placement was feasible in the pre-hospital and ED setting and significantly increased EtCO2
  - Only 4/11 sustained ROSC, no survivors in either study



PMID: 36774277; PMID: 35870557; PMID: 34242736; PMID: 34089774

Calcium

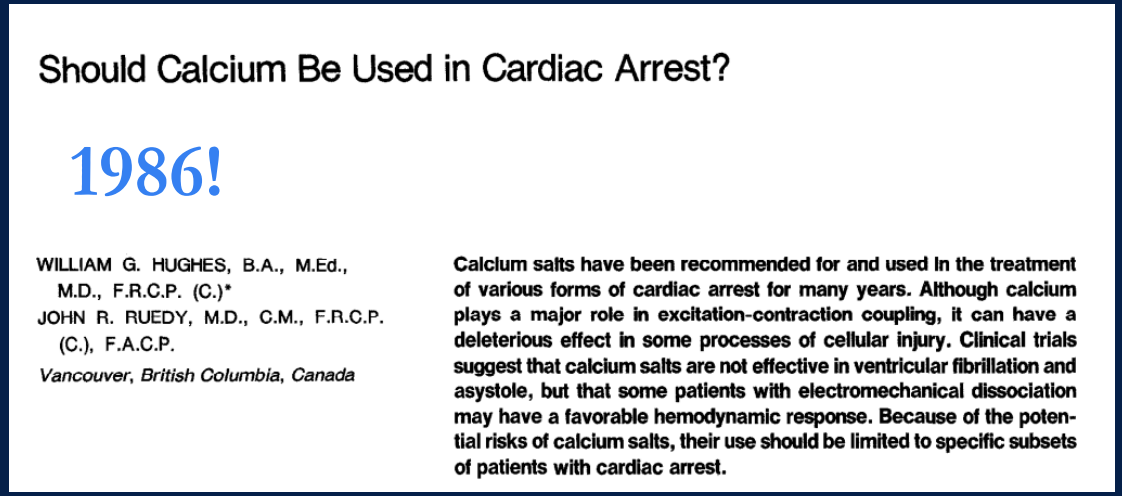
Routine calcium administration in cardiac arrest is not recommended

Nonvasopressor Medications		
COR	**LOE**	**Recommendations**
2b	B-R	1. Amiodarone or lidocaine may be considered for ventricular fibrillation/pulseless ventricular tachycardia that is unresponsive to defibrillation.
2b	C-LD	2. For patients with OHCA, use of steroids during CPR is of uncertain benefit.
3: No Benefit	B-R	3. Routine administration of calcium for treatment of cardiac arrest is not recommended.
3: No Benefit	B-R	4. Routine use of sodium bicarbonate is not recommended for patients in cardiac arrest.
3: No Benefit	B-R	5. Routine use of magnesium for cardiac arrest is not recommended.

Table from Perman et al. Circulation 2023 PMID 38108133

Calcium

- Animal studies in 1930s-1950s
  - Calcium useful in washing out potassium in animal models of induced Vfib
  - Clearly not relevant to human cardiac arrest



Should Calcium Be Used in Cardiac Arrest?

1986!

WILLIAM G. HUGHES, B.A., M.Ed., M.D., F.R.C.P. (C.)*
JOHN R. RUEDY, M.D., C.M., F.R.C.P. (C.), F.A.C.P.
Vancouver, British Columbia, Canada

Calcium salts have been recommended for and used in the treatment of various forms of cardiac arrest for many years. Although calcium plays a major role in excitation-contraction coupling, it can have a deleterious effect in some processes of cellular injury. Clinical trials suggest that calcium salts are not effective in ventricular fibrillation and asystole, but that some patients with electromechanical dissociation may have a favorable hemodynamic response. Because of the potential risks of calcium salts, their use should be limited to specific subsets of patients with cardiac arrest.

PMID: 3526886

Calcium

Essential for cardiac contractility **in normal physiology**

- However, during ischemia and reperfusion, calcium leads to more cellular apoptosis: ***calcium activated entropic doom***
- COCA trial suggests harm with calcium administration

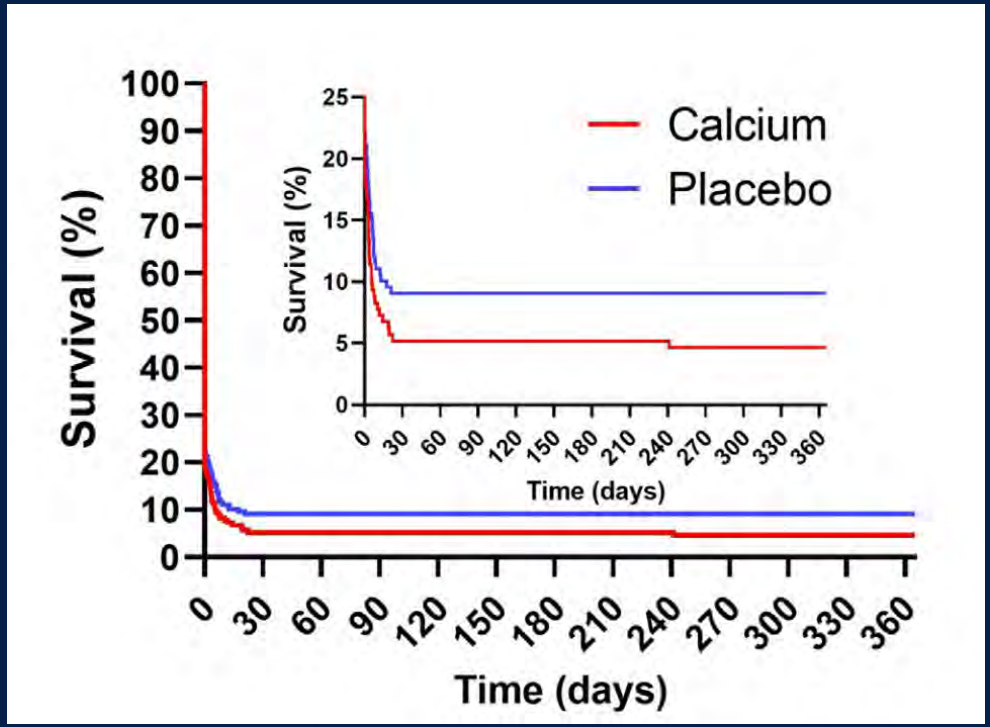


Figure from Vallentin et al. Resuscitation 2022 PMID 35917866

Calcium

No evidence for benefit, possible harm

- Retrospective study of intra-arrest calcium use in the ED over 9 years
  - Calcium administration was associated with ***decreased*** odds to survival to hospital admission (RR 0.74; 95% CI 0.66 – 0.82)
- Meta-analysis by Hsu *et al* found overall low quality evidence (only 3 RCTs) but no evidence of benefit

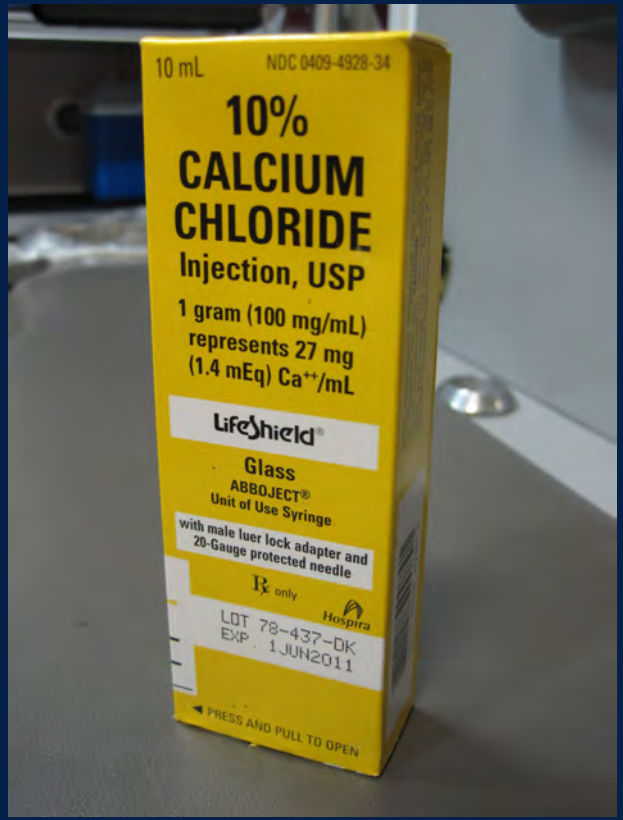


Image from Wikimedia Commons

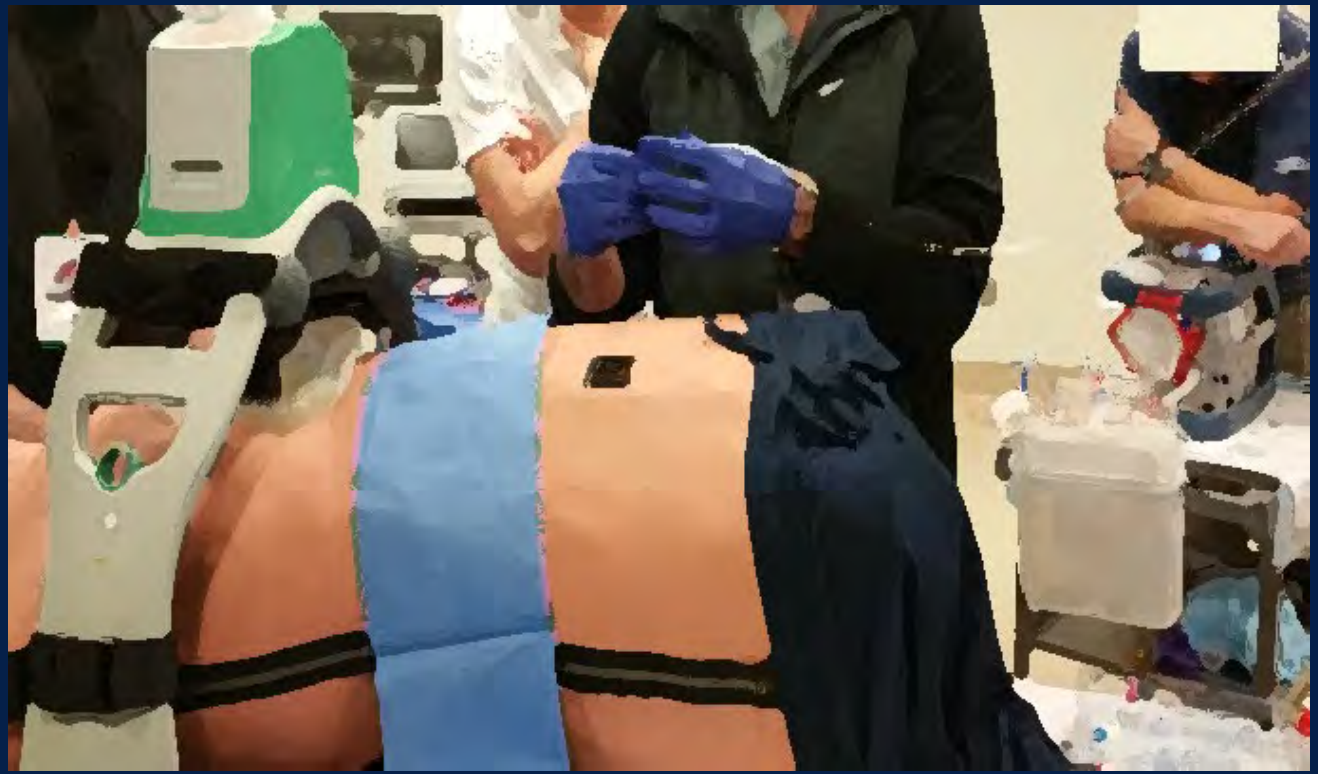
PMID: 37562663; PMID: 37025978

Calcium

When should calcium be considered in cardiac arrest?

- Hyperkalemia
- Calcium channel blocker overdose
- Wide QRS
- Hemorrhage

ECPR



ECPR – ARREST Trial

- Single center, open label RCT
  - Adults (18–75 years old)
  - Witnessed OHCA
  - Initial rhythm VF or pVT
  - no ROSC after three defibrillation attempts
  - LUCAS
  - Transport to the emergency department shorter than 30 min

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PMID: 33197396

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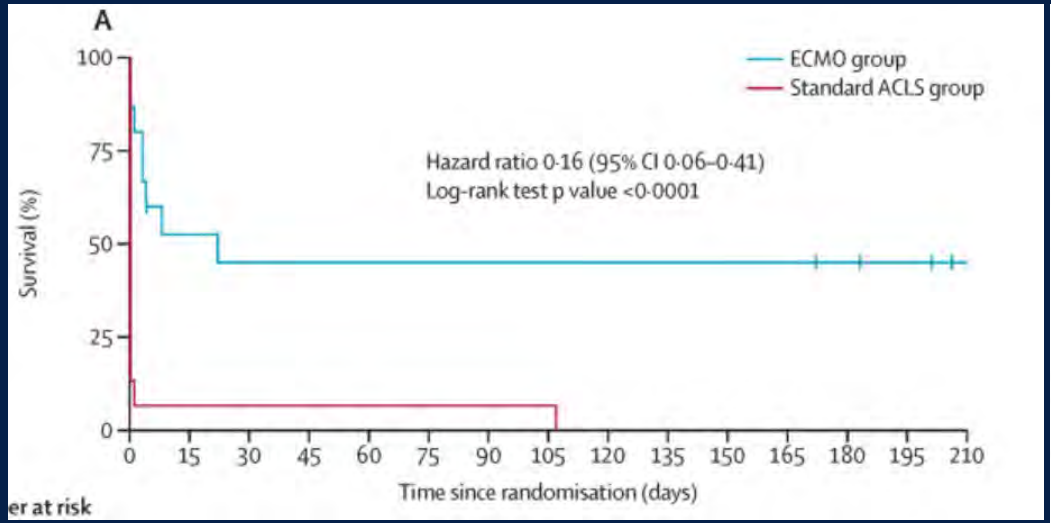
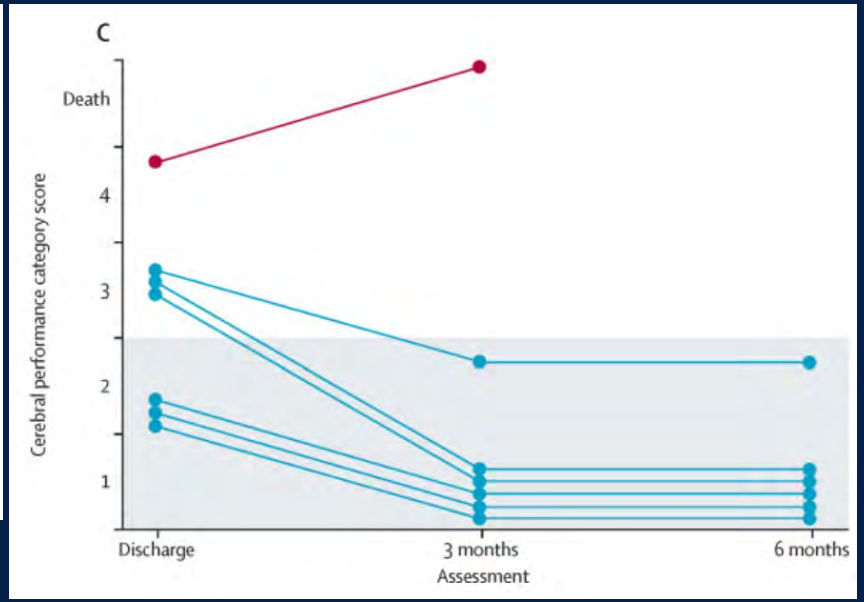
ECPR – ARREST Trial





Figures from Yannopoulos et al. Lancet 2020 PMID 33197396

- 1/15 (7%) survival in standard ACLS arm
- 6/14 (43%) survival in ECMO-facilitated resuscitation arm

PMID: 33197396

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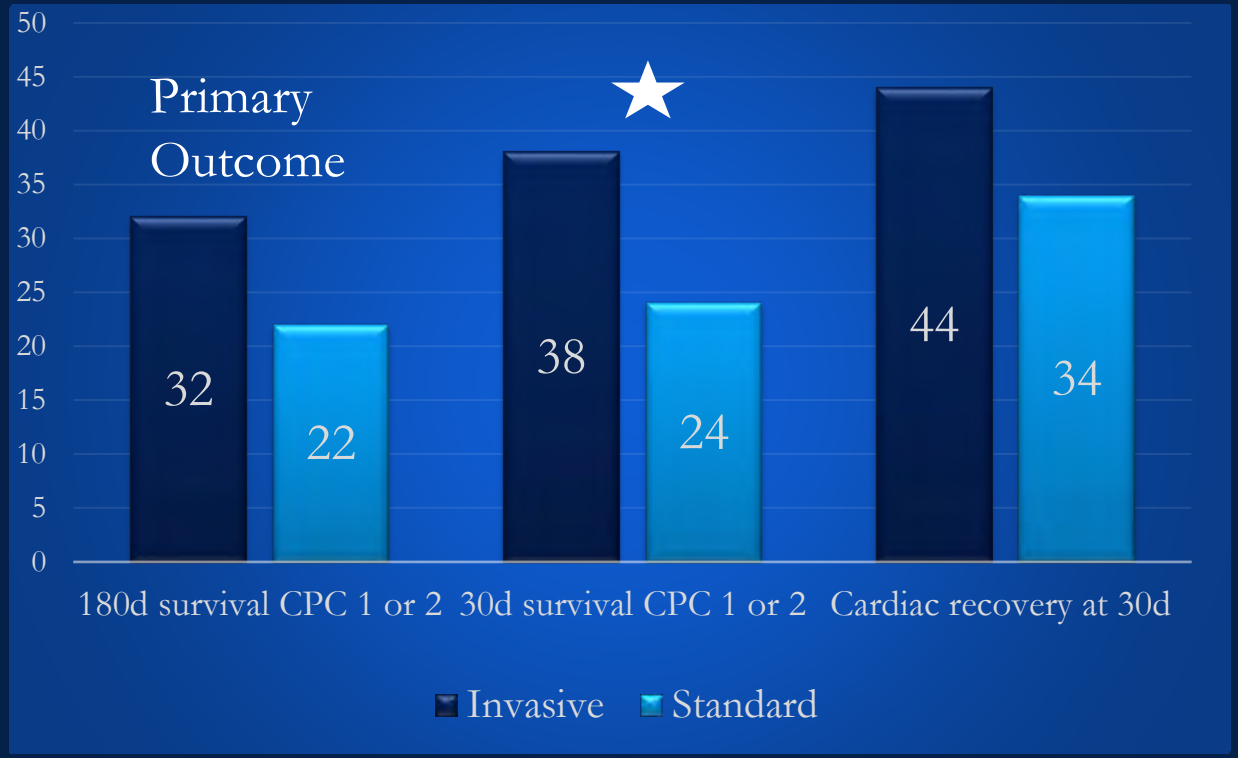
ECPR – Prague OHCA

Adults, witnessed OHCA of presumed cardiac cause, randomized to rapid intra-arrest transport, ECLS and invasive bundle vs standard care

- JAMA 2022
- Single center study in Prague 2013-2020
- Invasive bundle: ECLS, LHC, some intranasal cooling
- 264 randomized: 124 invasive (9 received standard), 132 standard (11 received invasive)
- 58% shockable in invasive group vs 64% shockable in standard group

PMID: 35191923

ECPR – Prague OHCA



PMID: 35191923

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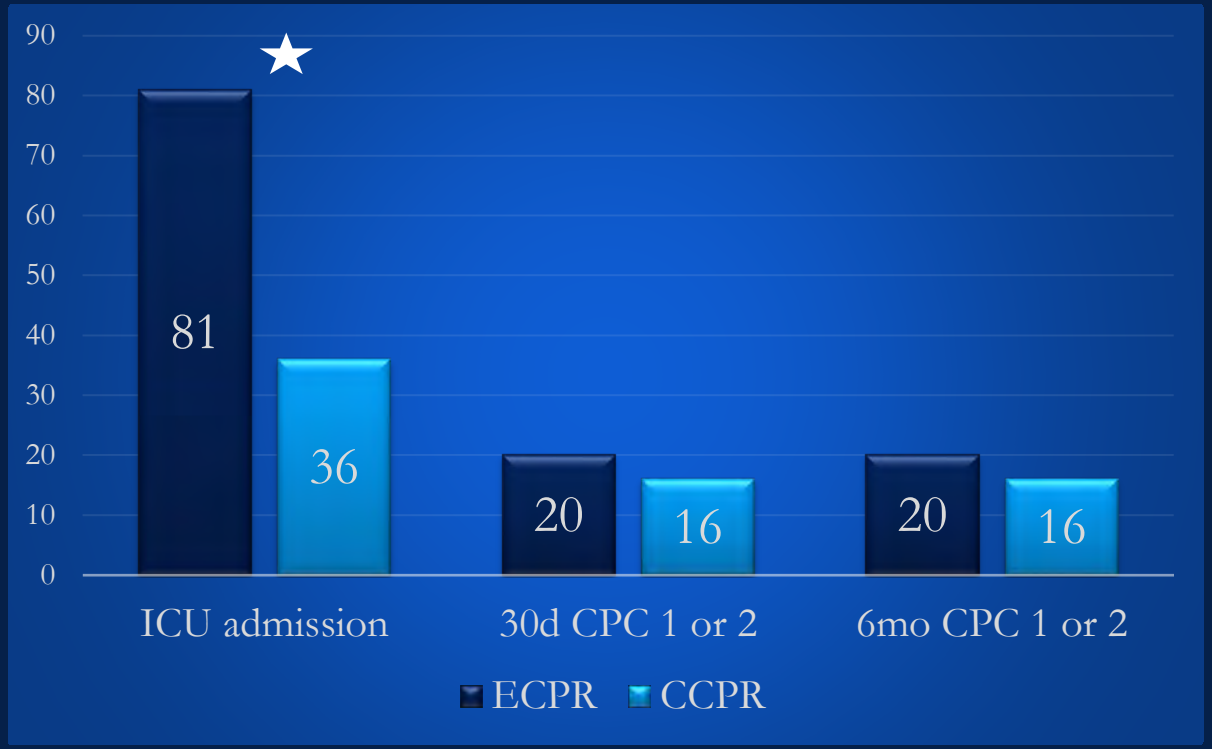
ECPR – INCEPTION

Adults, OHCA refractory VT/VF, randomized to ECPR vs conventional CPR

- NEJM, 2023
- Multicenter RCT in the Netherlands
  - n = 134; 70 ECPR, 64 standard care
  - 18 patients in ECPR group did not receive assigned intervention: 13 ROSC, 3 logistical failures, 2 stopped treatment
  - 12 patients in CCPR did not receive assigned intervention: 9 ROSC, 3 crossed over to ECPR

PMID: 36720132

ECPR – INCEPTION



PMID: 36720132

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ECPR – Low Flow Time

- ARREST: 59 ± 28 min
- Prague OHCA: 62 ± 11 min
- INCEPTION: 75 ±18 min

- In an observational study by Shoji *et al* including 1,524 ECPR patients, survival to discharge and survival with good neurologic outcome strongly associated with low-flow time < 40 min

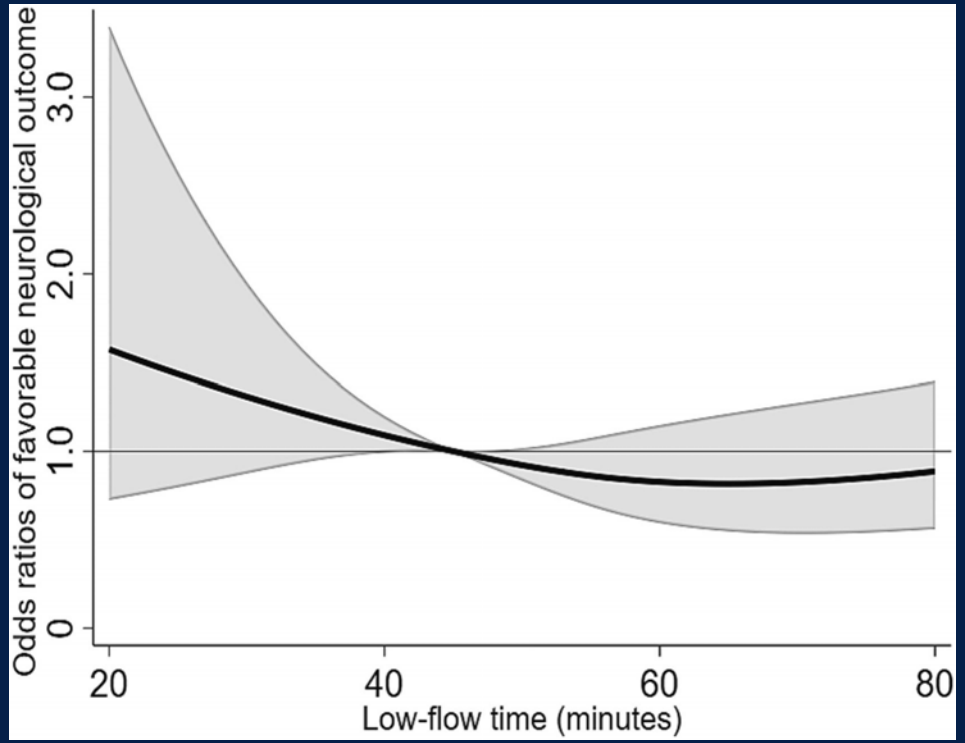

Figure from Shoji et al. Am J Emerg. Med 2023 PMID 37897919

PMID: 37230097; PMID: 37897919

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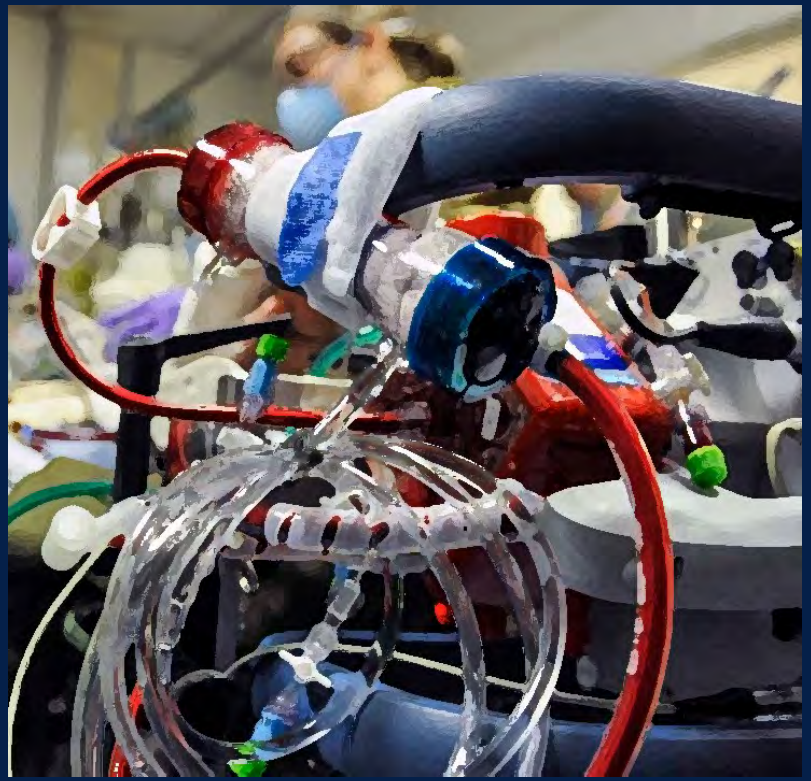
ECPR – Center Volume

- ARREST: 36 cases per year
- Prague OHCA: 27 cases per year
- INCEPTION: 2.8 cases per center per year



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Summary

- Maximize cardiocerebral resuscitation
- Goal directed use of intra-arrest medications
- Evidentiary basis behind consideration of ECPR

UCSF Fresno

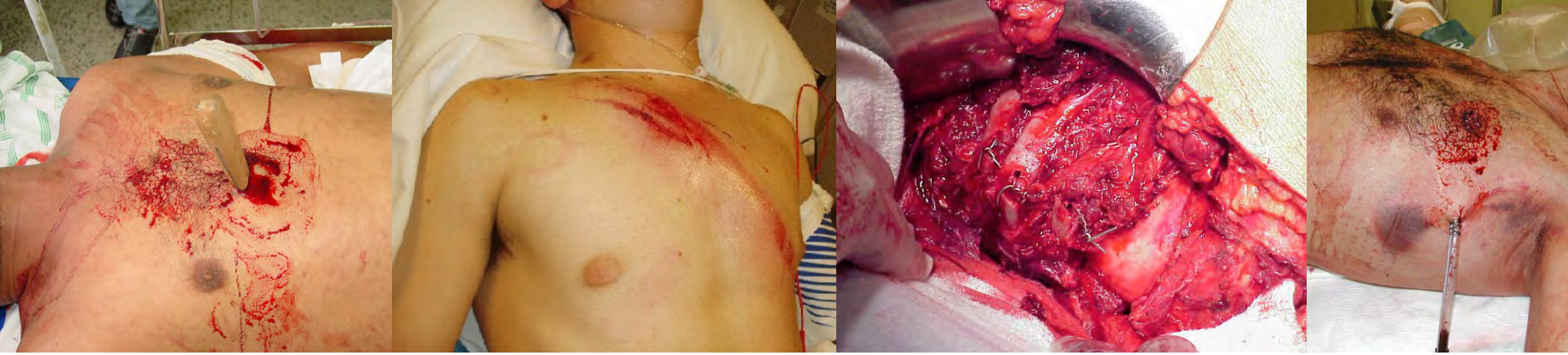
Trauma and Surgical Critical Care

Tasce Bongiovanni, MD, MPP
Assistant Professor
UC San Francisco

Tasce Bongiovanni, MD, MPP serves as an acute care surgeon and surgical critical care intensivist at UCSF Parnassus, and trauma surgeon at Zuckerberg San Francisco General (ZSFG).

Dr. Bongiovanni earned her Master's in Public Policy at the Harvard Kennedy School of Government. Dr. Bongiovanni has deep roots at UCSF spanning more than a decade. She earned her MD at UCSF, and then stayed on to complete her General Surgery Residency, followed by fellowships in Surgical Critical Care and Trauma Surgery at UCSF/ZSFG.

As a research resident, she was a Robert Wood Johnson Foundation (RWJF) Clinical Scholar at the Yale University School of Medicine. She was named as a recipient of a Learning Health Systems NIH K12 Grant and a 2019 John A. Watson Faculty Scholar Award. She continues her research in postoperative care of older adults with a K23 from the NIA and a Harold Amos Faculty Award from RWJF.



Thoracic Trauma

Tasce Bongiovanni, MD, MPP
California Thoracic Society
March 9, 2024
UCSF, Department of Surgery
Trauma, Acute Care Surgery & Critical Care

As adapted from slides by: Rachael Callcut, MD, MSPH

RELEVANT FINANCIAL DISCLOSURES

- I have the following relationships with ACCME defined ineligible companies:

- None

- I WILL NOT discuss off-label use and/or investigational use of any drugs or devices.

Any Trauma

- ABCDE first
- High index of suspicion
- Accurate diagnosis

Initial Assessment

ATLS Principles:

- Airway
- Breathing
- Circulation
- Disability
- Exposure

Remember to be:

- Organized
- Detailed
- Selective
- Rapid
- Team oriented

"Every action must have a life saving purpose."

Main Causes of Thoracic Trauma

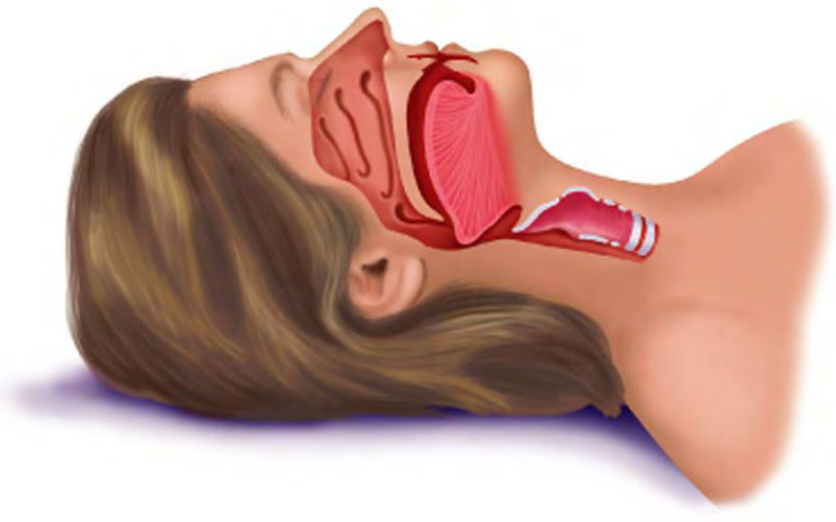
- ***Blunt Trauma****- Blunt force to chest.*

- ***Penetrating Trauma****- Projectile that enters chest causing small or large hole.*

- ***Compression Injury****- Chest is caught between two objects and chest is compressed.*

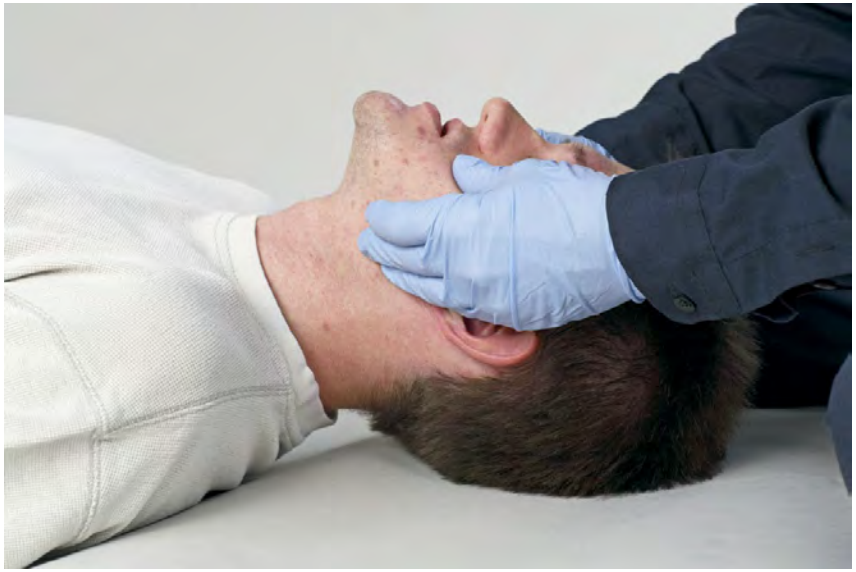
Airway

- Is the patient awake and alert?
- Mouth and breathing passage open?
- Is c-spine immobilization necessary?
  - Injury to the head, neck, or torso = hold head to make sure that they don't move their spine

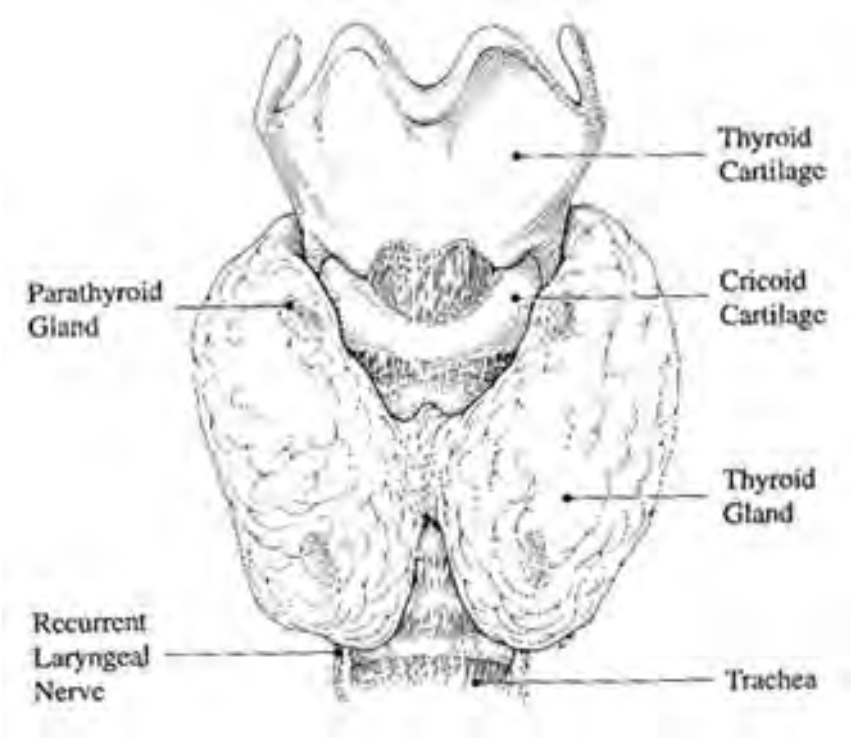


Airway management

- Unconscious without airway obstruction:
  - Chin lift or jaw thrust maneuver
  - Nasopharyngeal/ Oropharyngeal airway
  - Place in recovery position if no concern for spine injury
- Unsuccessful:
  - Intubation/Rescue airway (Combitube, King airway)
  - Cricothyroidotomy



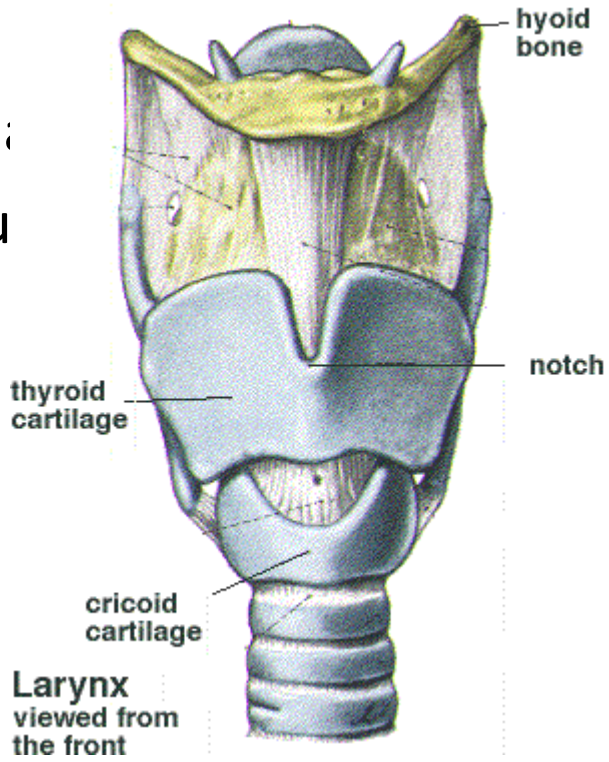
Anticipate Airway Compromise

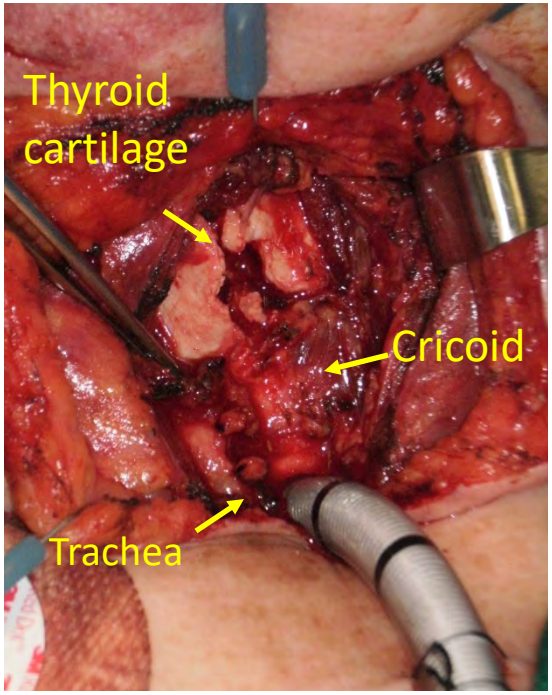


- Cricothyrotomy: true emergency
  - Stabilize cartilagenous framework by holding thyroid cartilage
  - VERTICAL incision at the level of the cricothyroid membrane
  - Incise cricothyroid membrane with #11 blade
  - Dilate with hemostat – leave in hole
  - No. 6 endotracheal tube

Laryngeal Injury

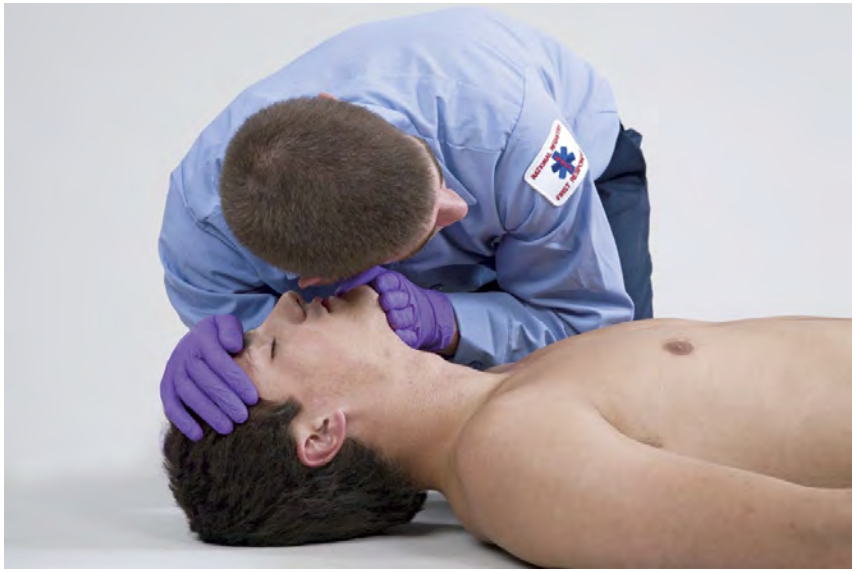
- Triad
  - Hoarse voice
  - Subcutaneous a
  - Palpable fractu





Breathing

- Is the patient breathing?
  - Look for rise and fall of chest
  - Listen for breathing
  - Feel for breath
- Is the chest rising and failing symmetrically?
  - If not breathing, administer “rescue breaths”



Signs of breathing trouble

- Difficult or labored breaths
- Gurgling sounds
- Wheezing
- Bluish lips/nails
- Slow or Fast RR

Breathing

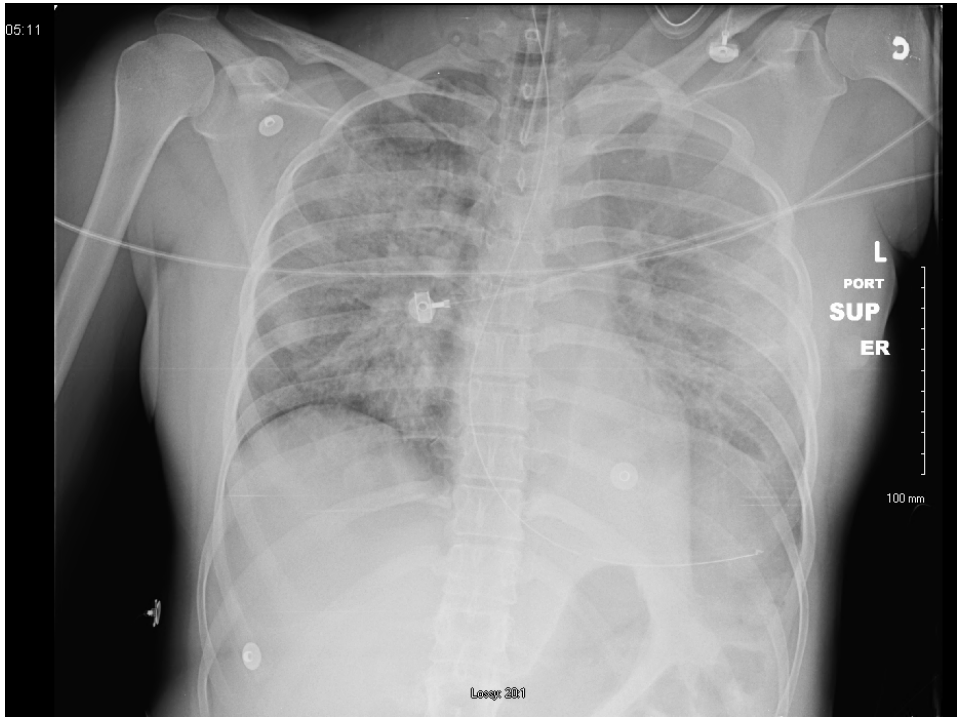
- Is there palpable subcutaneous air?

Subcutaneous Emphysema

- Air collects in subcutaneous fat from pressure of air in pleural cavity
- Feels like rice crispies or bubble wrap
- Can be seen from neck to groin area

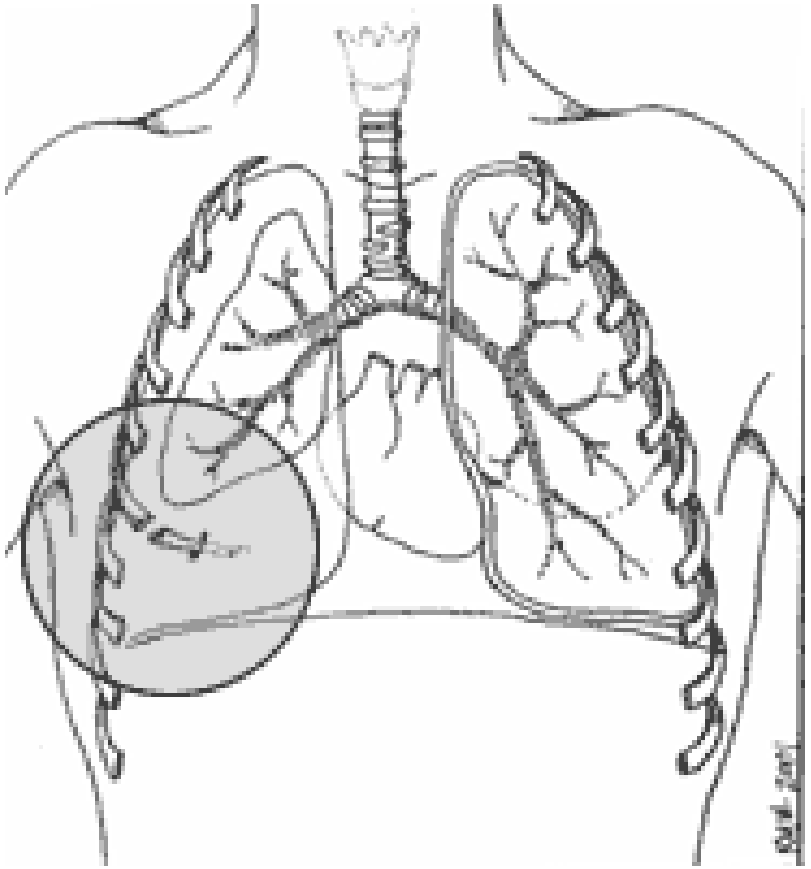
Life Threatening Thoracic Injuries

- Pneumothorax
  - Simple pneumothorax
  - Tension pneumothorax
  - Open pneumothorax
- Flail chest
- Pulmonary contusion
- Massive hemothorax
- Diaphragm Rupture



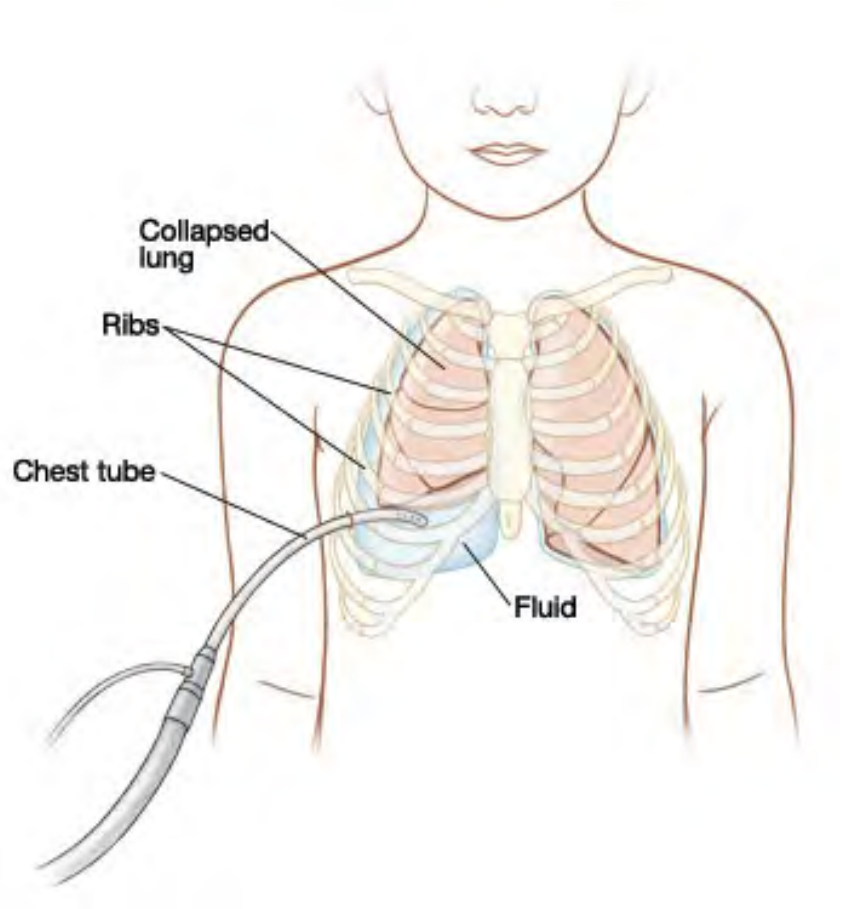
Simple/Closed Pneumothorax

- Opening in lung tissue that leaks air into chest cavity
- Blunt trauma is main cause
- May be spontaneous



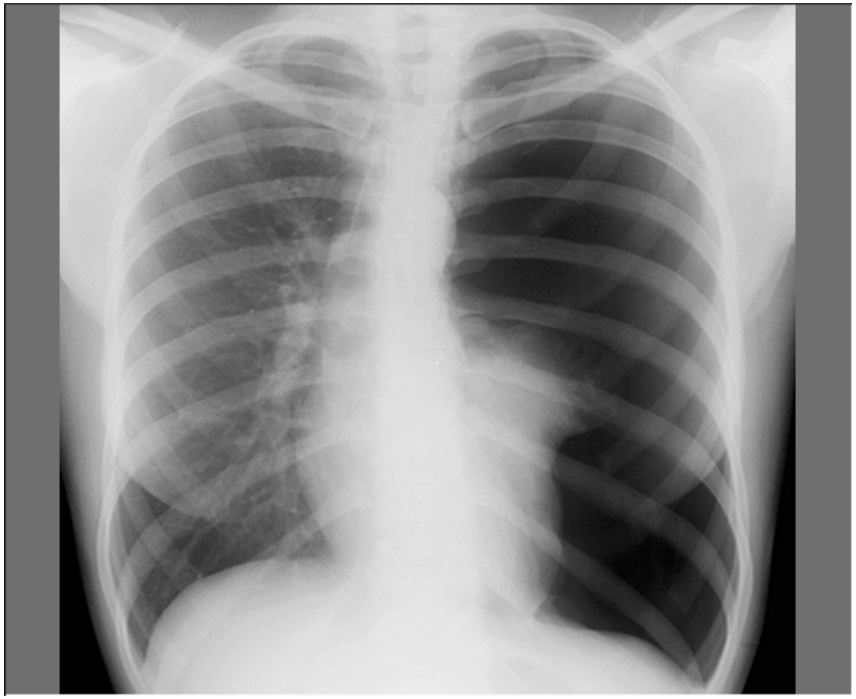
Treatment for Simple/Closed Pneumothorax

- ABC's
- Provide supportive care
- Chest Tube placement



Tension Pneumothorax

- 1 way valve where air forced into thoracic cavity with no escape
- Results in collapse of lung on affected side
- Shifts mediastinum, other lung, great vessels



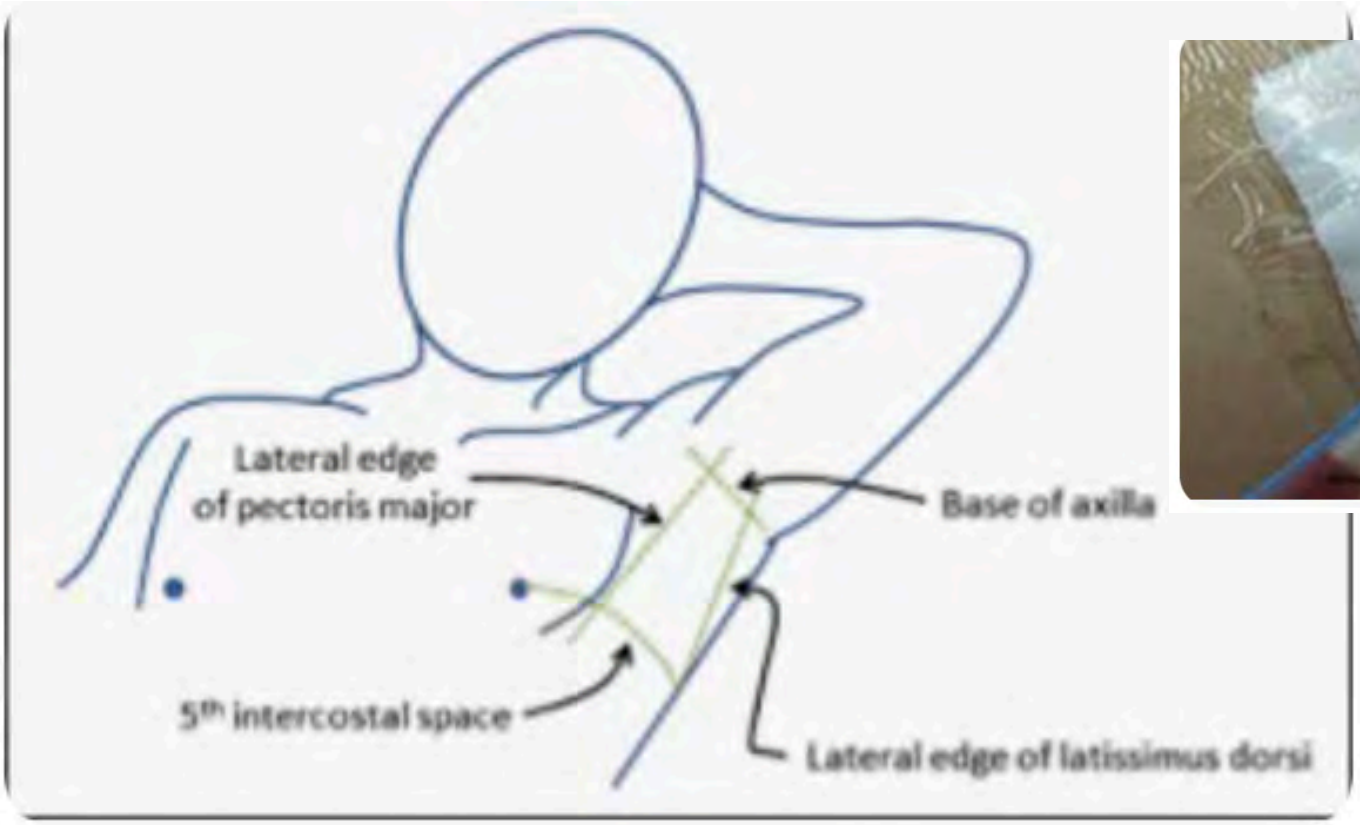
Warning signs of Tension PTX

- Anxiety/Restlessness
- Severe Dyspnea
- Absent Breath sounds on affected side
- Tachypnea
- Tachycardia
- Poor Color
- Accessory Muscle Use
- JVD
- Narrowing Pulse Pressures
- Hypotension
- Tracheal Deviation

Needle Decompression

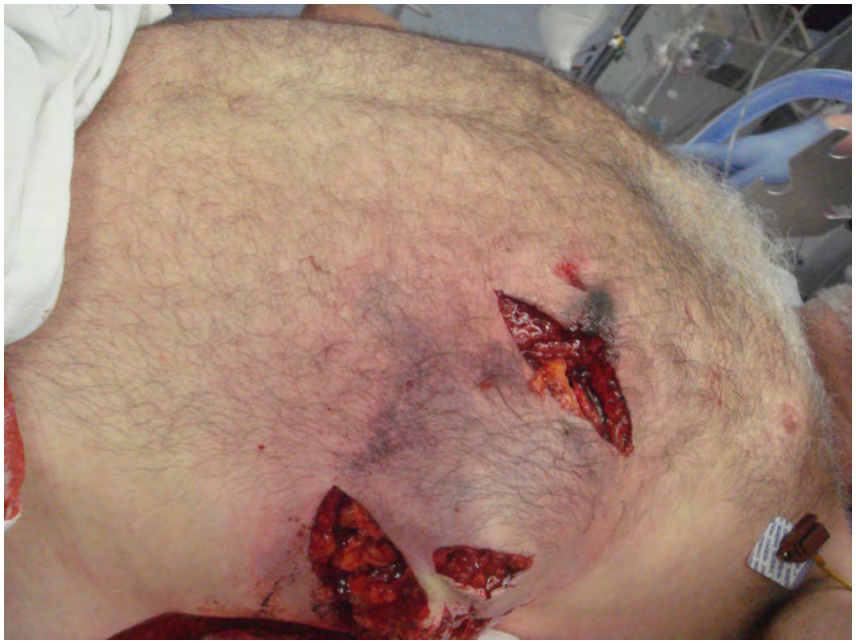
- Used to be: Locate 2-3 Intercostal space midclavicular line
- Now: same place as chest tube
- Cleanse area using aseptic technique
- Insert catheter (14g or larger) at least 3” in length over the top of the 3rd rib(nerve, artery, vein lie along bottom of rib)
- Remove Stylette and listen for rush of air
- Place Flutter valve over catheter
- Reassess for Improvement

Needle Decompression



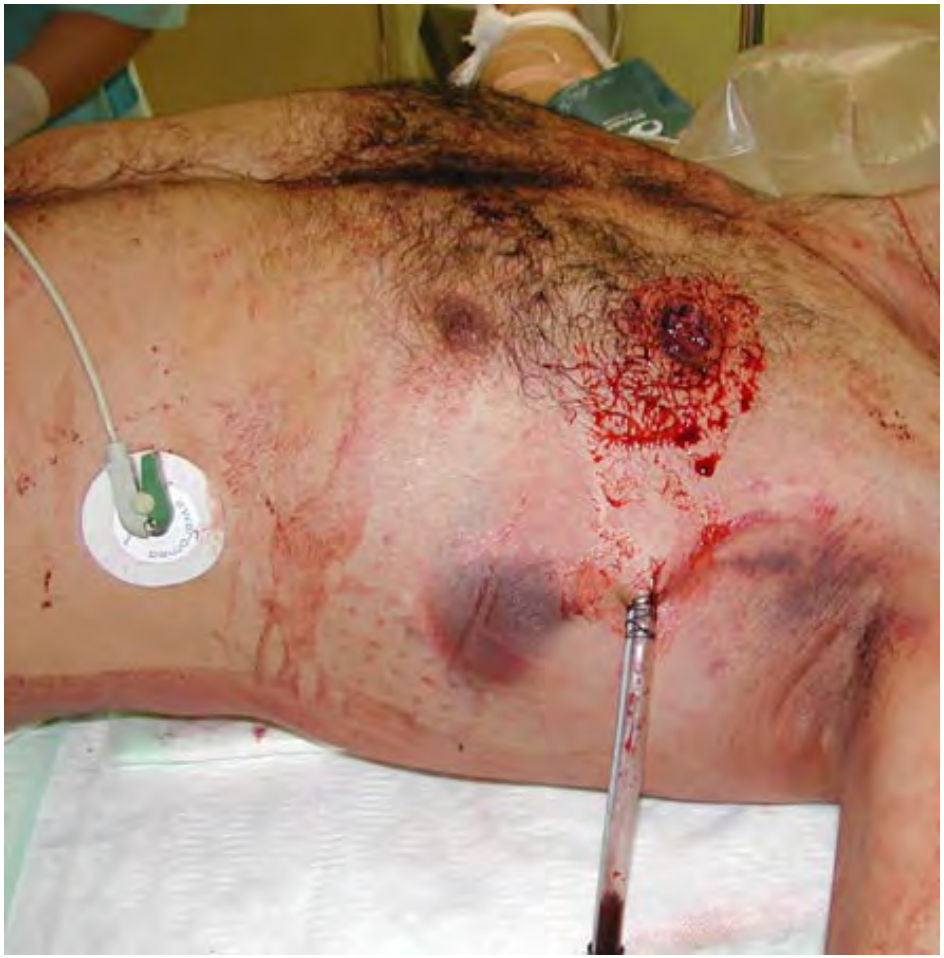
Breathing

- Is there a wound to the chest?
- Is there a wound to the back or flank?



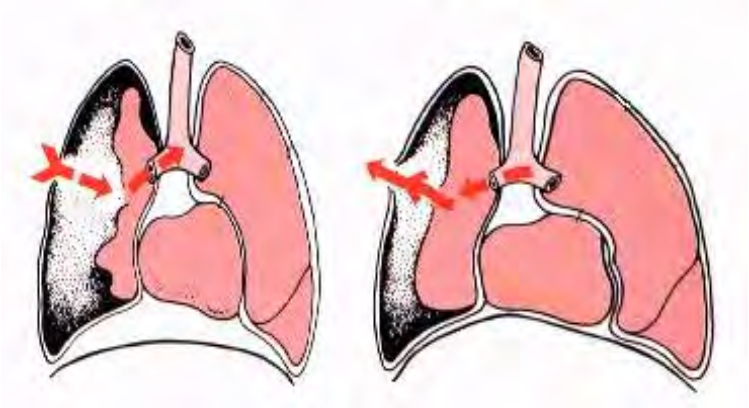
Open Pneumothorax

- Opening in chest cavity that allows air to enter pleural cavity
- Causes the lung to collapse due to increased pressure in pleural cavity
- Life threatening
- Deteriorate rapidly



Warning Signs of Open Pneumothorax

- Dyspnea
- Sudden sharp pain
- Subcutaneous Emphysema
- Decreased lung sounds on affected side
- Bubbles on Exhalation from wound

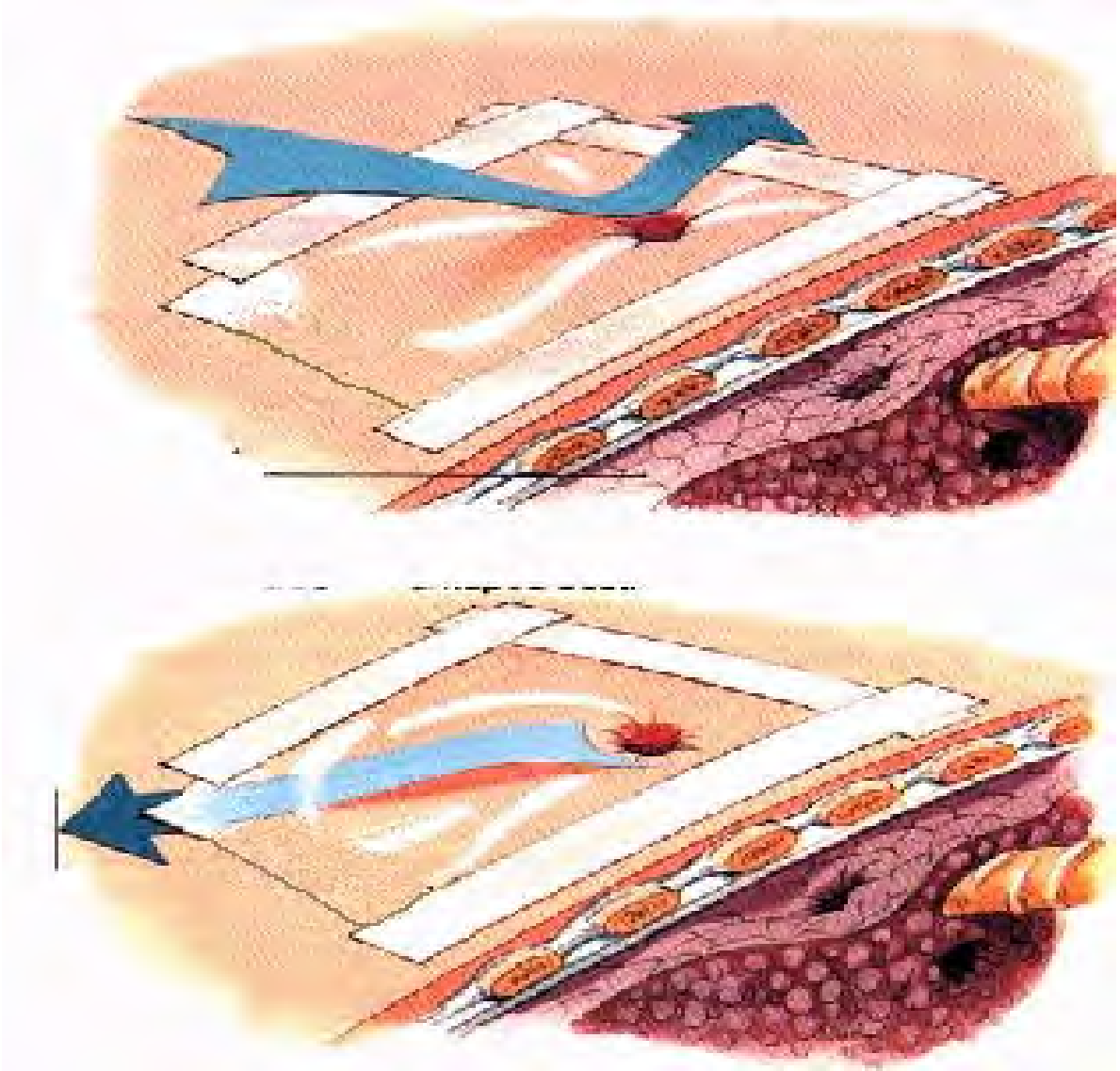


"Sucking Chest Wound"

Open chest wound

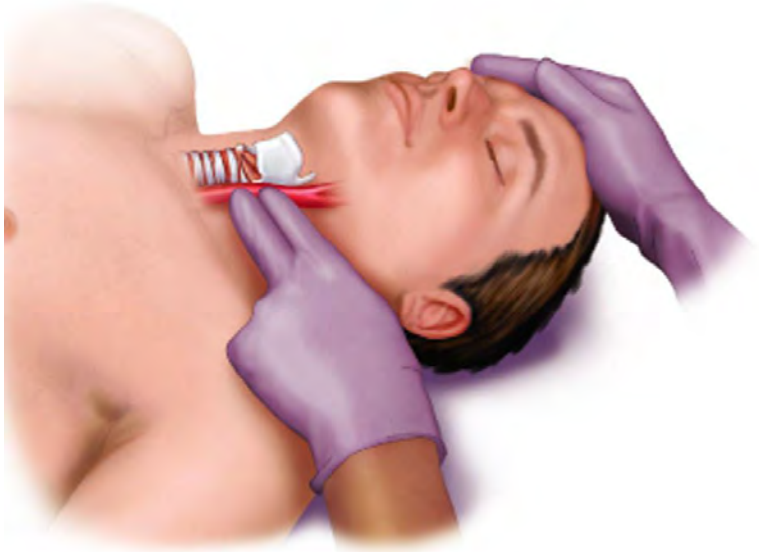
- Re-establish ventilation
- Air movement out airway not chest wall
  - Close open hole
- Relieve tension pneumothorax
  - Needle into pleural cavity
  - 8 cm
  - 14 gauge

Occlusive Dressing

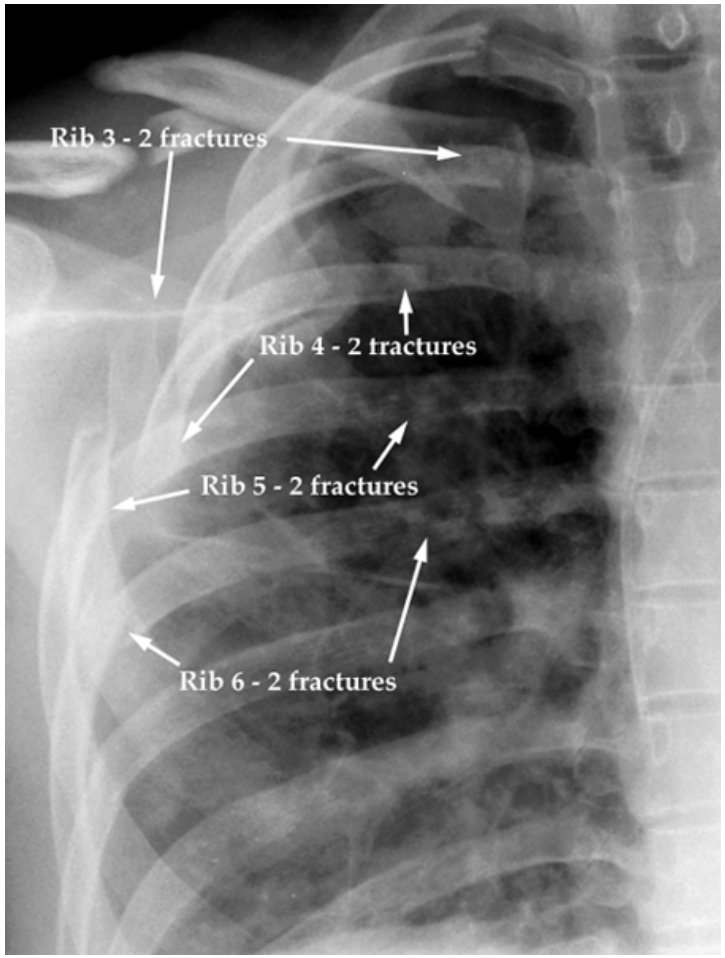


Circulation

- Does the patient have a pulse?
  - Carotid, Femoral, Wrist
- Can you get a BP?
  - Carotid – 60mm Hg
  - Femoral – 70 mm Hg
  - Wrist – 80mm Hg
- Capillary refill?
- Do you have IV supplies?
  - Does the patient need IVFs?
- Should you do CPR?
  - Multiple GSWs – likely already dead



Flail Chest



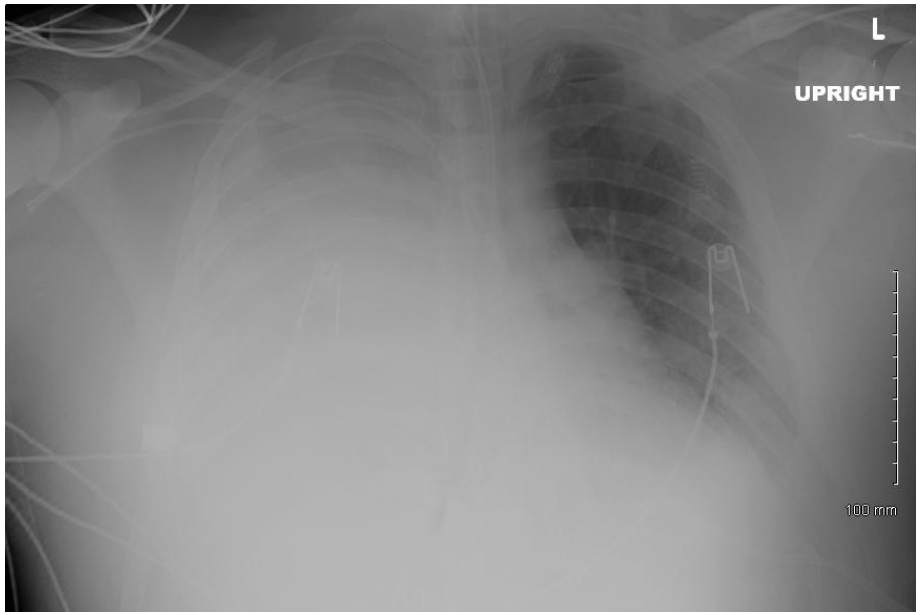
- 2 breaks in a single rib
- More than 2 sequential ribs

Warning Signs of Flail Chest

- Shortness of Breath
- Paradoxical Movement
- Bruising/Swelling
- Crepitus (Grinding of bones)

Massive Hemothorax

- pleural space fills with blood
- Usually occurs due to lacerated blood vessel in thorax
- As blood increases, it puts pressure on heart and other vessels in chest cavity
- Each Lung can hold 1.5 liters of blood

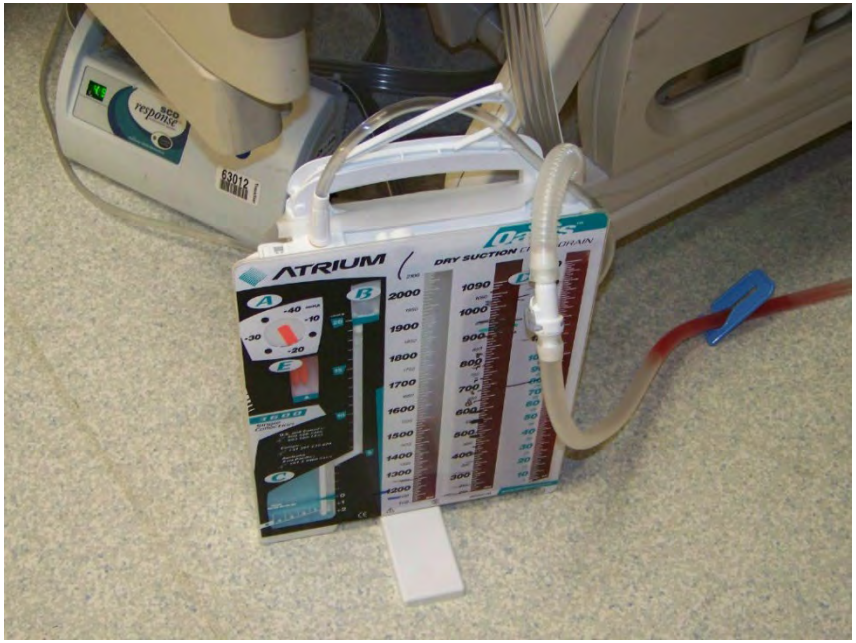


Warning Signs of Hemothorax

- Anxiety/Restlessness
- Tachypnea
- Signs of Shock
- Frothy, Bloody Sputum
- Diminished Breath Sounds on Affected Side
- Tachycardia
- *Flat Neck Veins*

Treatment for Hemothorax

- ABC's
- Secure Airway
- Treat Shock
- CT with auto-transfusion

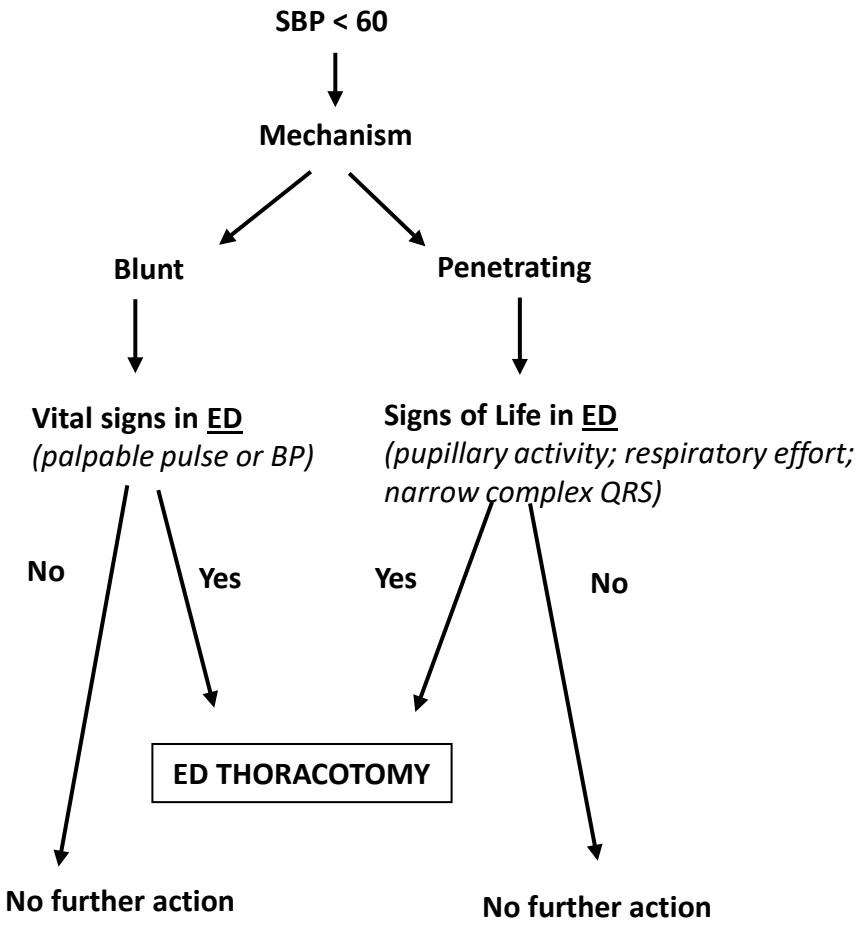


Resuscitative Thoracotomy

Resuscitative Thoracotomy

- Hemorrhage control
- Release of cardiac tamponade
- Open cardiac massage
- Prevention of air embolus
- Cross-clamping of descending thoracic aorta
- Control of intra-abdominal hemorrhage

Example: Protocol



3 Factors to Consider:

1. Mechanism of Injury
2. Location of the Injury
3. Signs of Life

Outcomes:

- Penetrating 8-10%
  - SW 18-24%
  - GSW 4-5%
- Blunt <1%

Controversial Indications

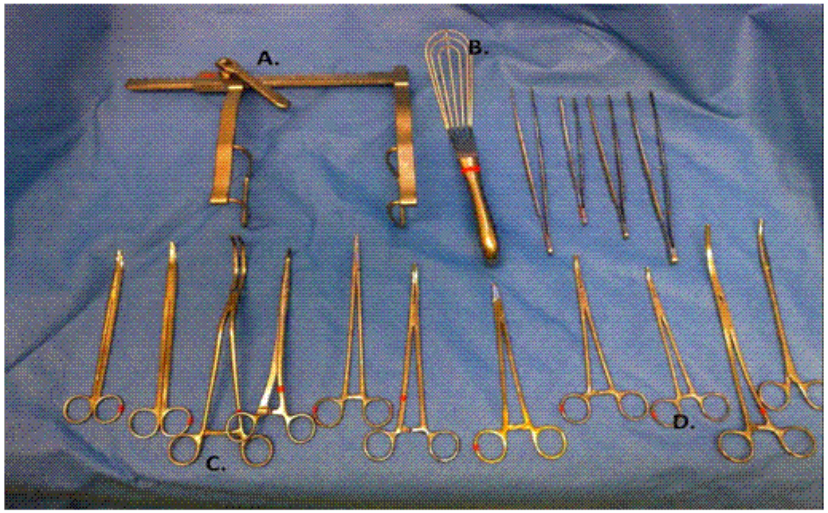
- *Pre-hospital arrest – only in penetrating trauma with loss of VS within 10 (some use 15) minutes prior to arrival
- Penetrating injury with traumatic arrest without previously witnessed cardiac activity
- Penetrating non-thoracic injury with traumatic arrest with previously witnessed cardiac activity
- Blunt thoracic injuries with traumatic arrest with previously witnessed cardiac activity pre-hospital

Contraindications

- Blunt injury without witnessed cardiac activity
- Penetrating abdominal trauma without cardiac activity (ie; use ultrasound to SEE cardiac motion) / no signs of life
- Severe head injury
- Improperly trained team
- Insufficient equipment

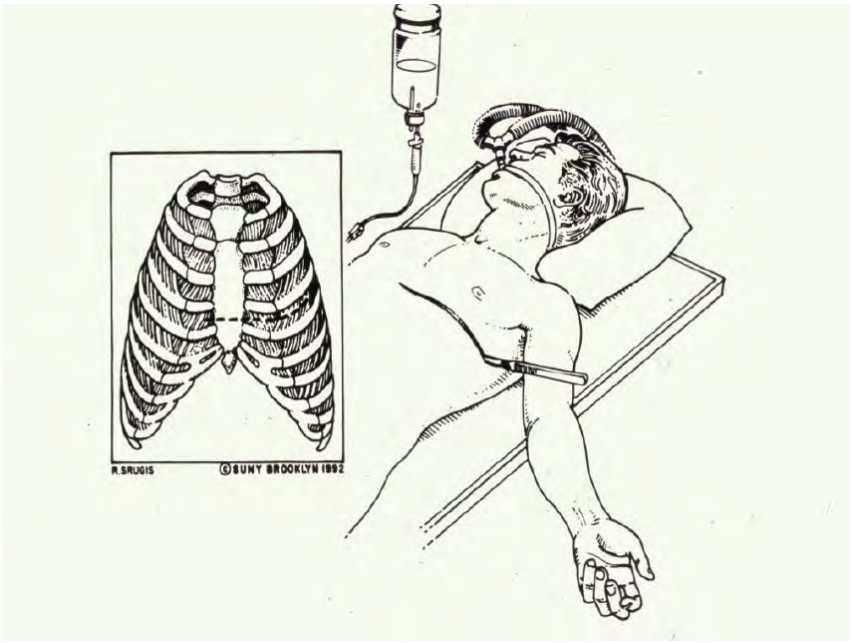
Equipment

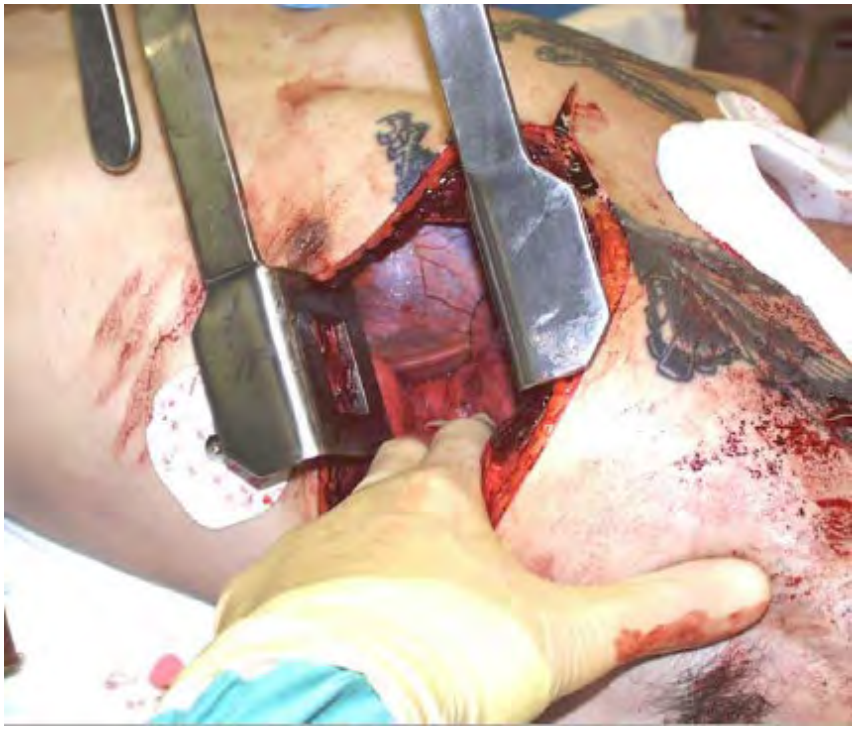
- Retractors, scissors, forceps, scalpels
- Needle holder, curved artery forceps
- Vascular clamps, curved artery forceps, Crawford clamps
- Internal defibrillation paddles
- Skin stapler, sutures, surgical ties



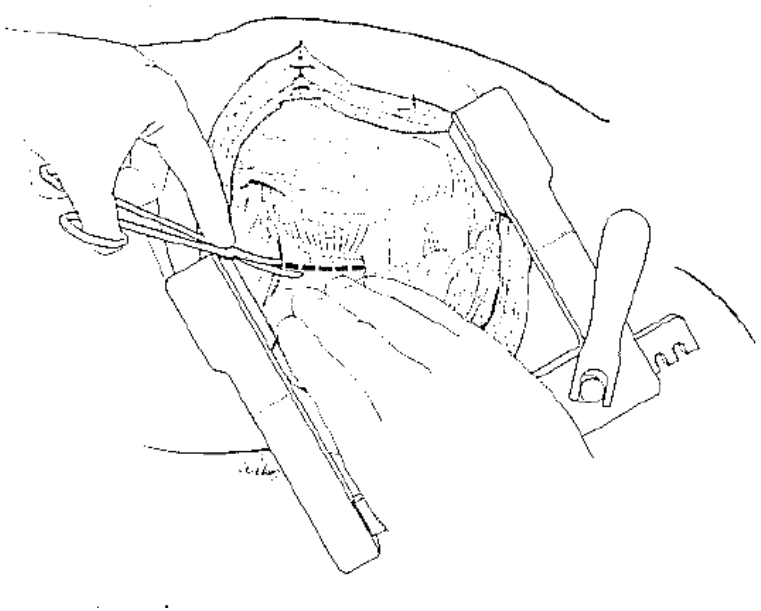
Steps:

- 5th or 6th IC space from sternum to posterior axillary line (below nipple line in men, below inframammary crease in women)
- Initial incision should be through all subcutaneous tissue and down to chest wall.
- Intercostal muscles are incised with scissors
- Insert rib spreader. HANDLE toward the axilla.





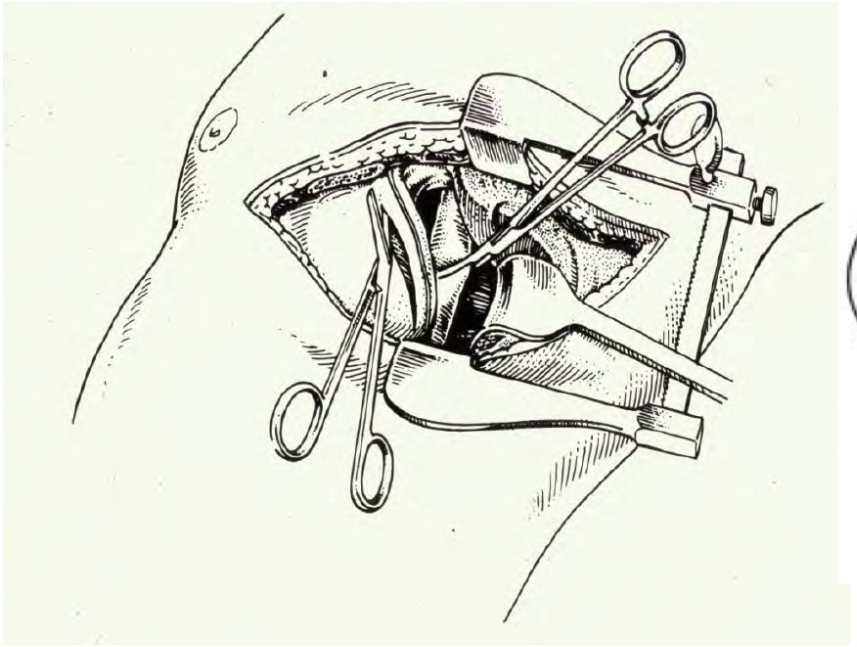
Finochietto Retractor

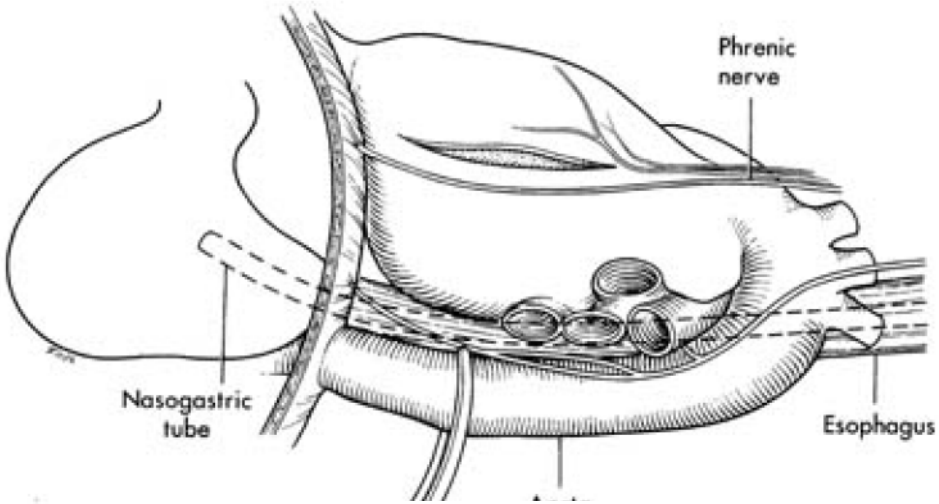


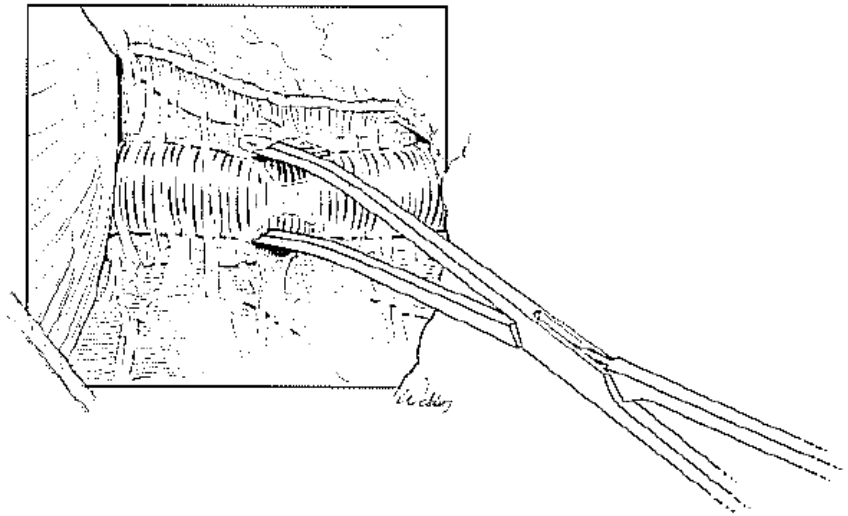
Incise inferior pulmonary ligament

Sweep lung away

L anterolateral thoracotomy





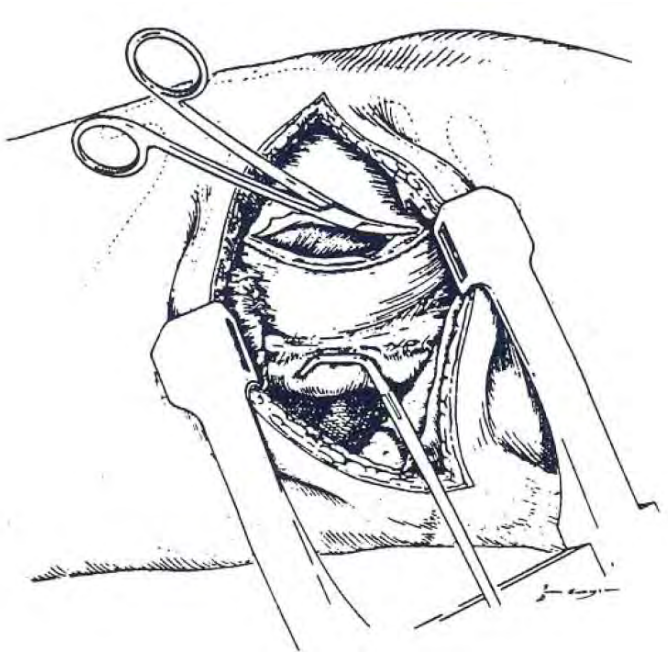


Go lateral to medial along ribs.

First tubular structure = aorta

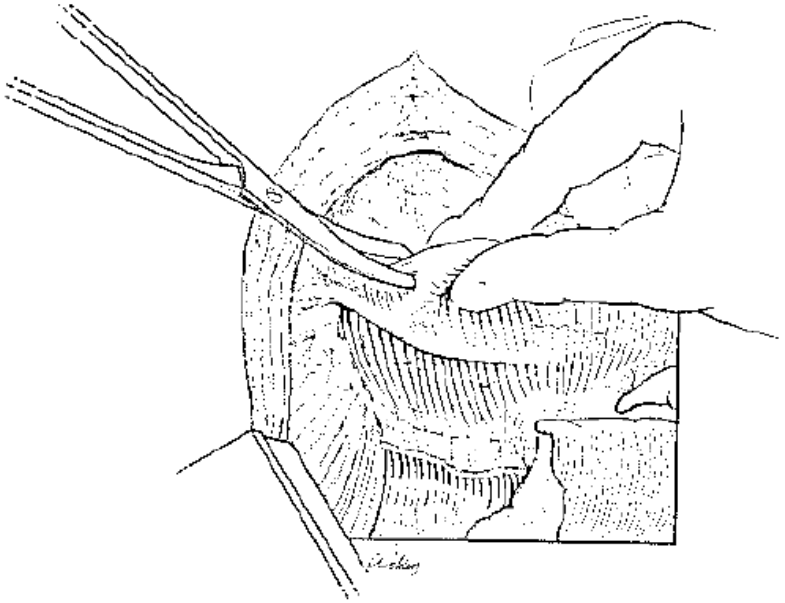
Open Parietal Pleural

Opening Pericardium



Make longitudinal pericardiotomy MEDIAL to phrenic nerve to deliver heart from pericardial cradle

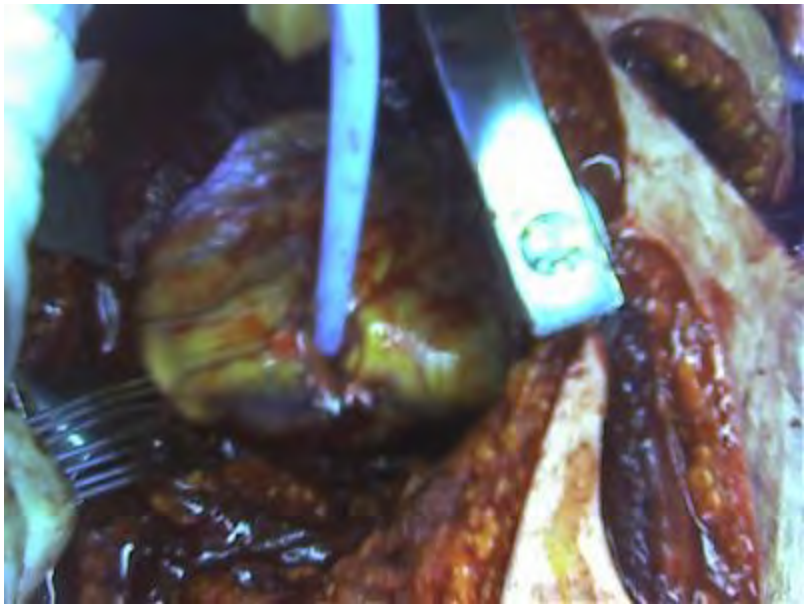
Can also relieve tamponade

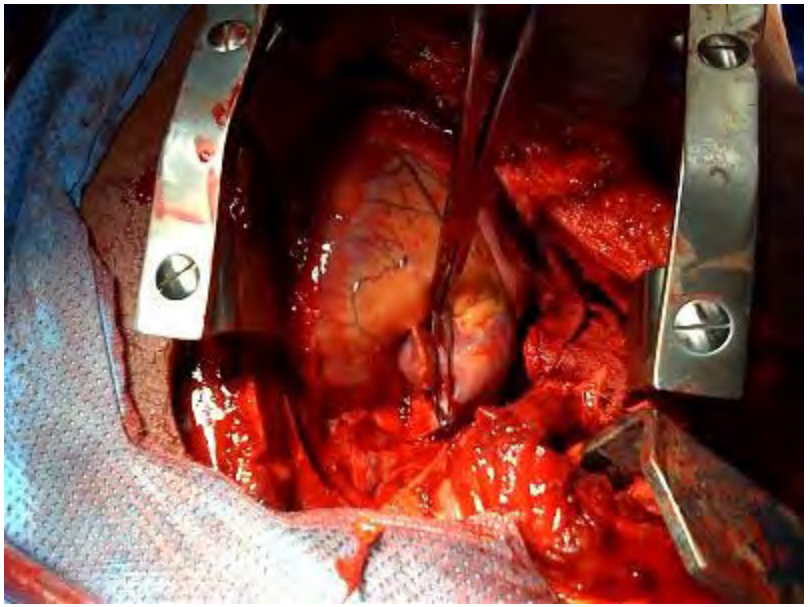


Cardiac Injury

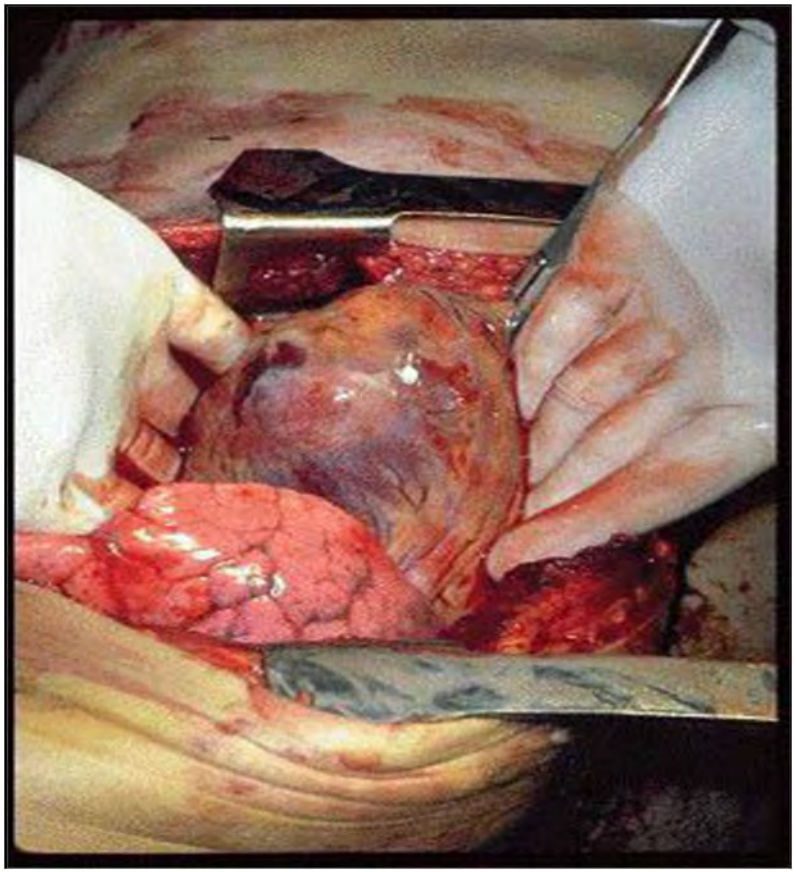
The myocardial defects can be closed with buttressed Vicryl sutures avoiding the coronary arteries.

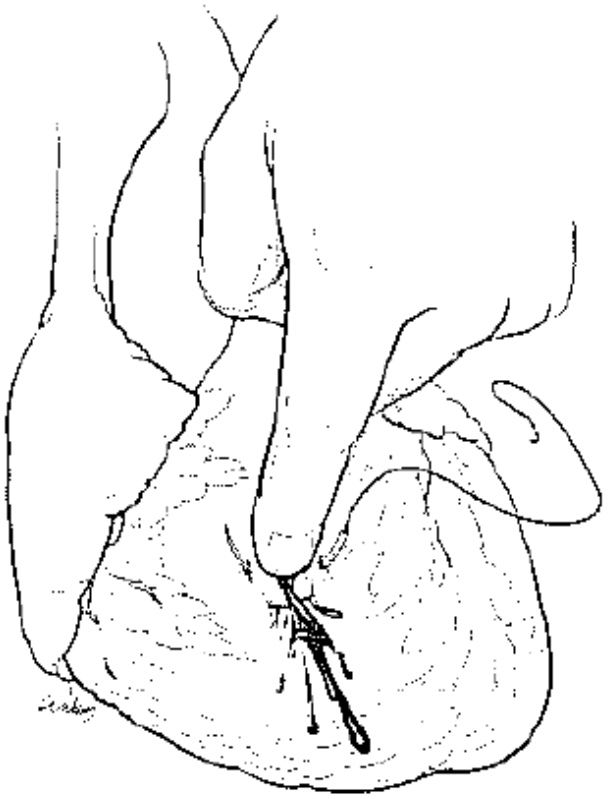
To be quick in ED: stapler





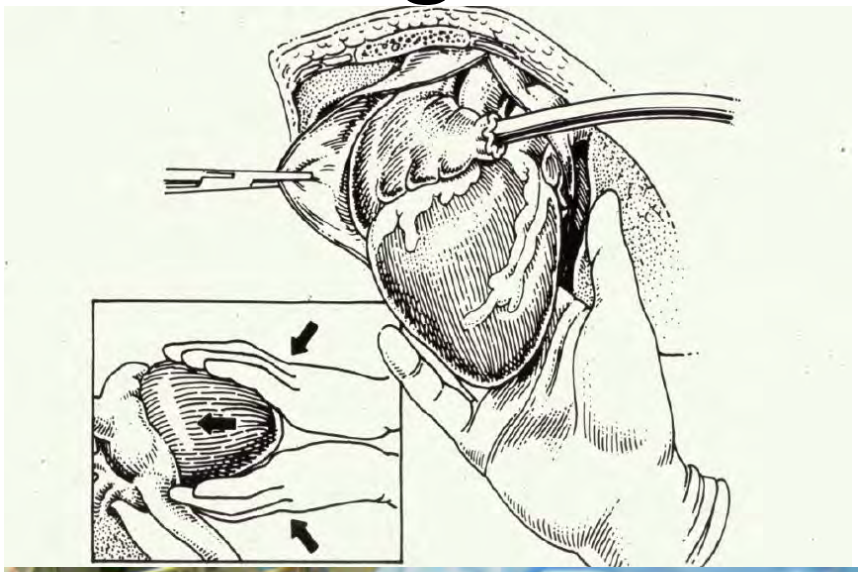
Cardiac Injury

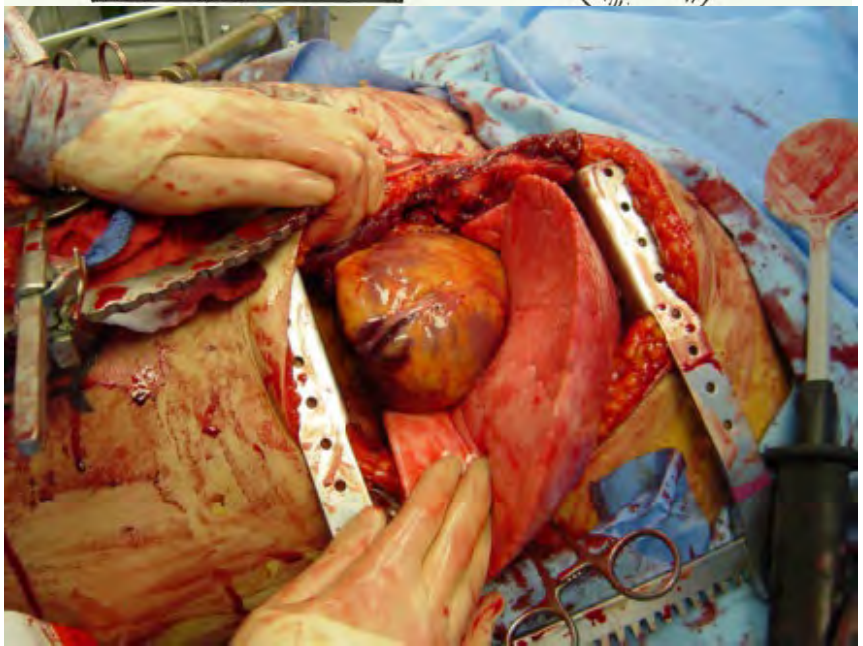




Open Cardiac Massage

- Compress the heart between two flat hands in a hinged clapping motion
- Defibrillate using small internal paddles either side of the heart with 15-30 J



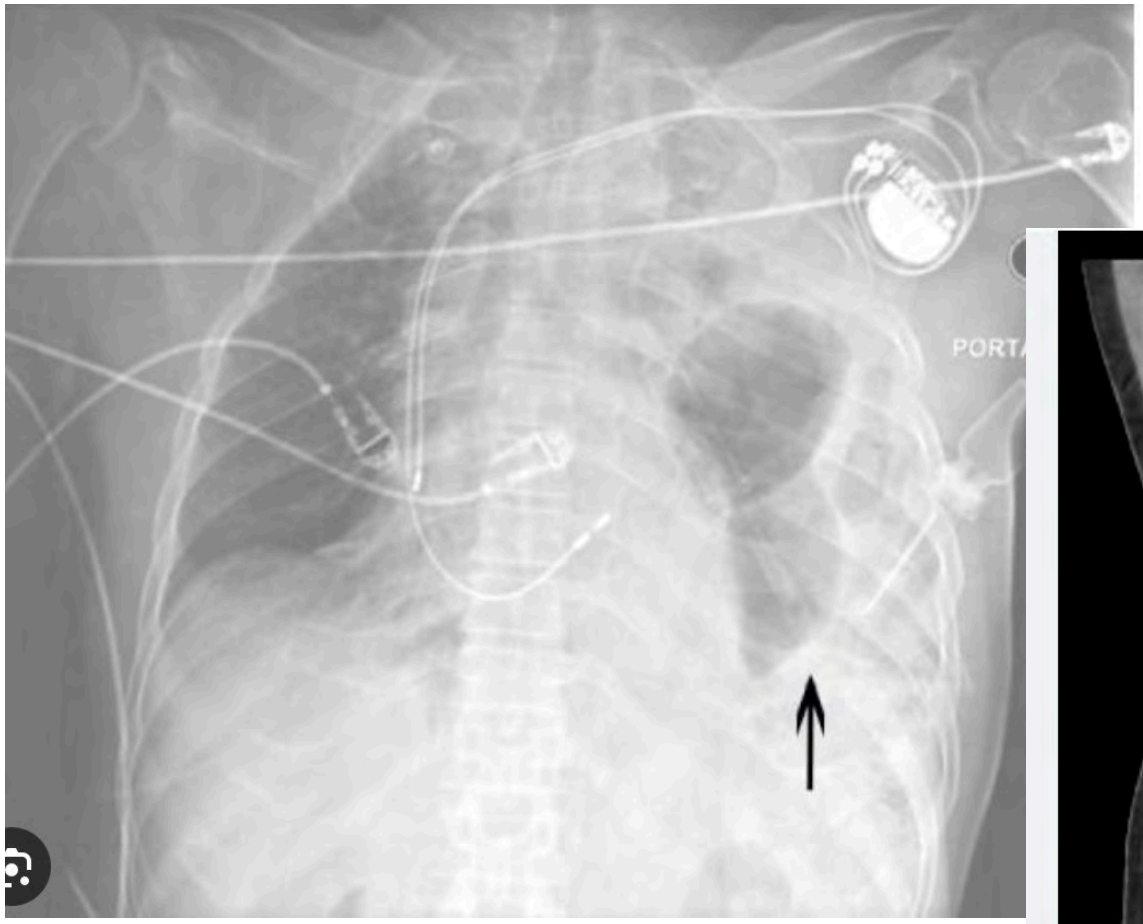


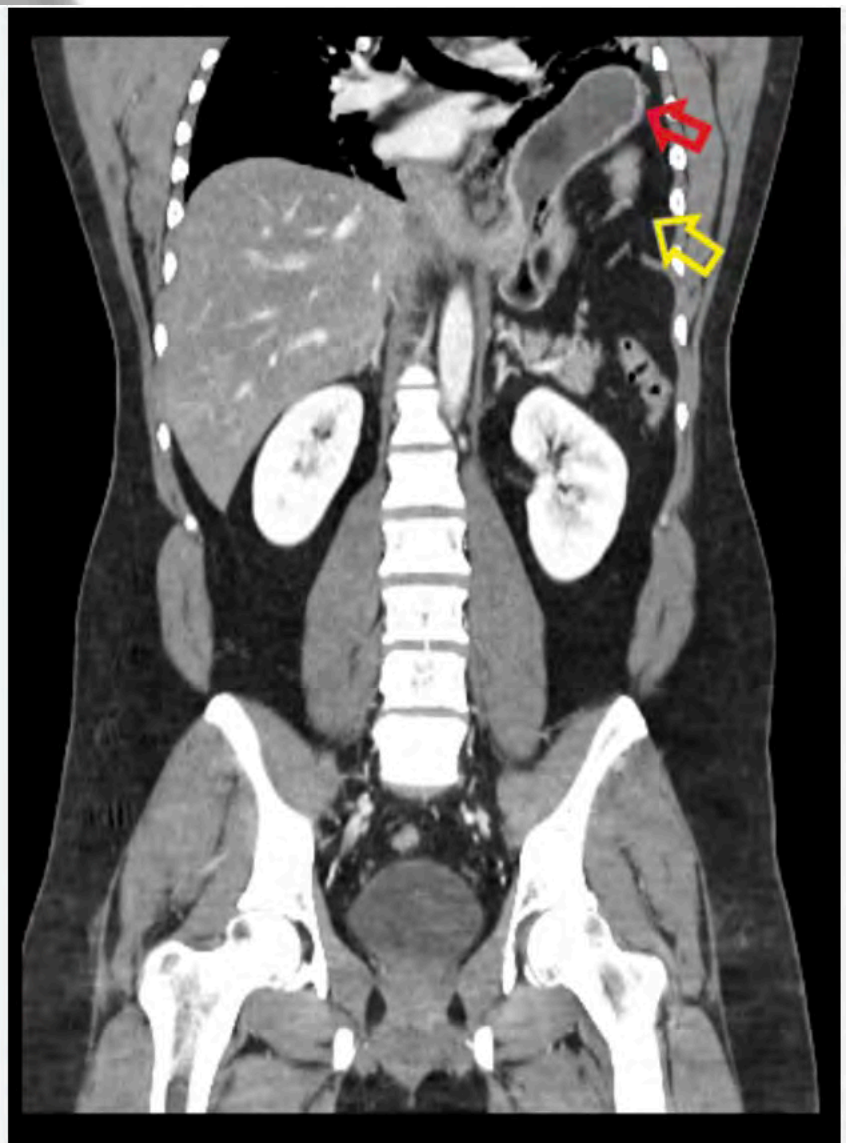
Diaphragmatic Rupture

- A tear in the Diaphragm that allows the abdominal organs enter the chest cavity
- More common on Left side due to liver helps protect the right side of diaphragm
- Associated with multipile injury patients

Warning Signs of Diaphragmatic Rupture

- Abdominal Pain
- Shortness of Air
- Decreased Breath Sounds on side of rupture
- Bowel Sounds heard in chest cavity





Treatment of Diaphragmatic Rupture

- ABC's
- Recognize injury
- High Association with other injuries
- Don't put in a chest tube!

Summary

- ABCDE first
- High index of suspicion
- Accurate diagnosis

Extra slides if time

Treatment of Pericardial Tamponade

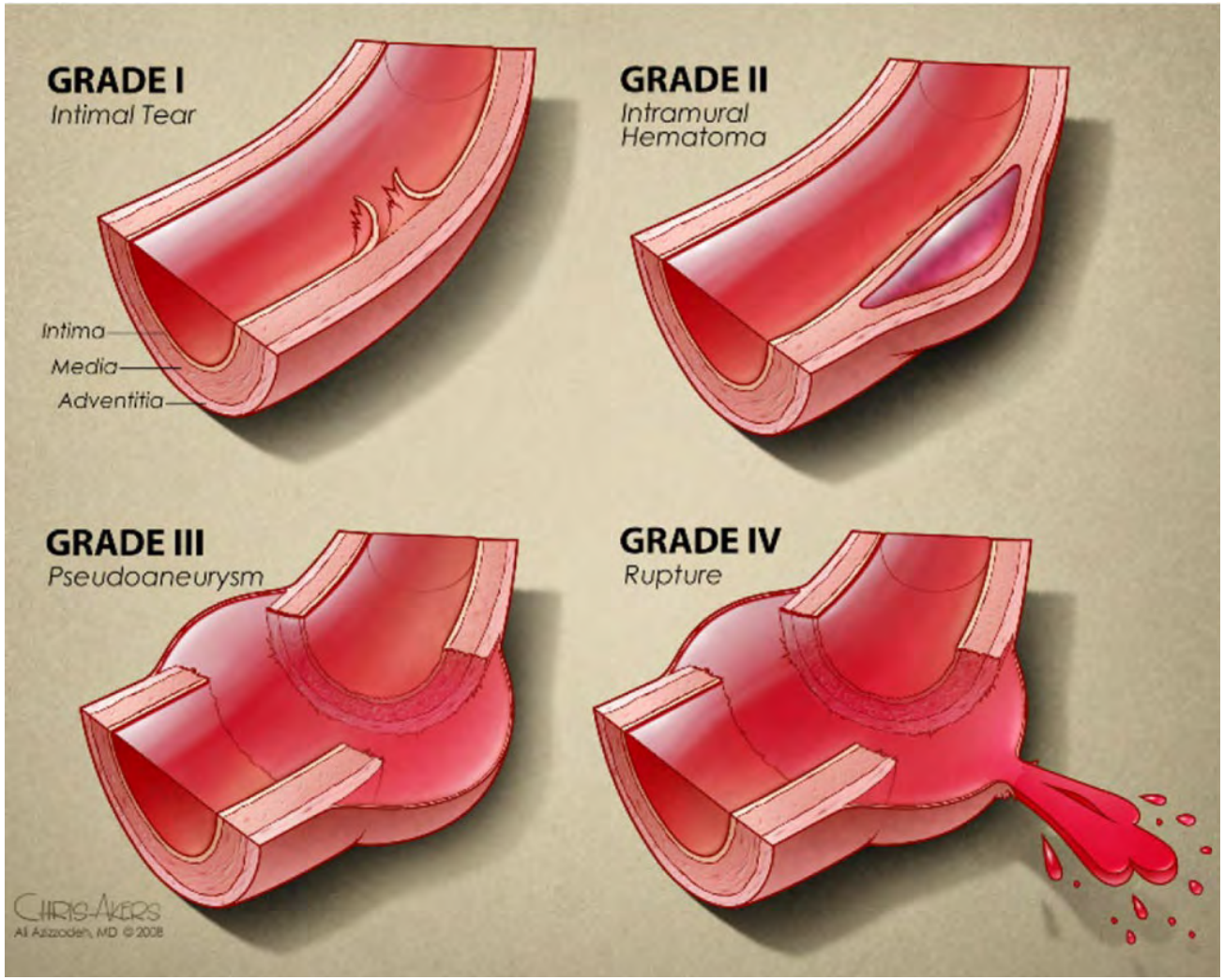
- ABC’s
- Surgical Treatment
- Temporizing method = Pericardiocentesis

Blunt Aortic Injury

- Pre-hosp mortality = 85%
- Most at Isthmus

Repair Open or Endovascular?

Blunt Aortic Injury Types



Warning Signs Of Traumatic Aortic Rupture

- Burning or Tearing Sensation in chest or shoulder blades
- Rapidly dropping Blood Pressure
- Pulse Rapidly Increasing
- Decreased or loss of pulse or b/p on left side compared to right side
- Rapid Loss of Consciousness

Imaging Signs of Traumatic Aortic Rupture

- CXR
  - Fxs.
  - Obliteration of aortic knob
  - Wide mediastinum (>8cm)*
  - Depression of L main bronchus
  - Loss of perivertebral stripe
  - Lateral displacement of trachea
  - Loss of AP window*
  - Apical cap
  - Large L hemothorax
  - Diaphragmatic Injury

Sleep in the ICU

Shazia Jamil, MD
Professor
Scripps Health

Shazia M. Jamil, MD is the Head of Academic Affairs in the Division of Pulmonary, Critical Care and Sleep Medicine and Professor of Medicine at Scripps Clinic where she practices as Intensivist and Sleep specialist. She is a Clinical Professor of Medicine at UCSD school of Medicine. Dr Jamil was named CTS Outstanding Clinician (2021-2022) and received national recognition for receiving ATS Outstanding Clinician Award in 2022. She is a recipient of multiple teaching and research awards from USC, Scripps Clinic, University of California, and American Lung Association. She received her medical degree from Aga Khan Medical School in Pakistan, completed Internal Medicine residency at USC, followed with Pulmonary and Critical Care fellowship, Molecular and Cell Biology post-doctoral fellowship and Sleep Medicine training all at UCSD. She believes that the best approach to diagnosis and management of a patient is integration and application of basic science and physiological knowledge to each clinical setting. Her goal has been to bridge the knowledge gap between community and academic clinicians in the hope of providing evidence-based medical care to all patients, no matter which setting they are being cared for. She has developed several ICU protocols for management of complex liver, transplant and COVID-19 patients and multidisciplinary measures to improve sleep and founded Circadian Rhythm Sleep Disorders Clinic, one of its first kind in California. Her clinical excellence is matched by her zeal for advancing education. Over the last 15 years, she has worked assiduously to develop clinical programs, curricula, CME conferences and hands-on-skills sessions at the local, regional and national level aimed to teach community physicians, trainees and young faculty. She founded a free CME *San Diego Pulmonary, Critical Care and Sleep Medicine Case Conference* which brings together academic and private physicians, updating them on advancement in medical literature and helping with challenging cases. She co-founded and Chairs *Rapid Response Document Series* that is regularly published in *AJRCCM* which addresses emerging lung health issues and provide rapid, readily used practical information for healthcare professionals and patients. Her group published one of the first public health and clinical documents in the U.S. on SARS Co-V2 and COVID-19. She has chaired Sleep Core Curriculum at ATS Education Committee and currently serves as Sleep Chair for the ATS Fellows Track Symposium and Chairs Education Committee at California Thoracic Society.

SLEEP IN THE INTENSIVE CARE UNIT

CTS Annual Conference, March, 2024

SHAZIA M. JAMIL, MD, FCCP, FAASM, ATSF

HEAD ACADEMIC AFFAIRS, DIVISION OF PULMONARY, CRITICAL CARE AND SLEEP MEDICINE, SCRIPPS CLINIC

CLINICAL PROFESSOR OF MEDICINE, SCRIPPS CLINIC AND

UNIVERSITY OF CALIFORNIA, SAN DIEGO SCHOOL OF MEDICINE

FOUNDING DIRECTOR CIRCADIAN RHYTHM SLEEP DISORDERS CLINIC, SCRIPPS CLINIC

I have no Conflicts of Interest

OUTLINE

Normal Sleep and Changes in Sleep with Aging

Impact of Sleep Deprivation in Critically Ill

Measuring Sleep in ICU

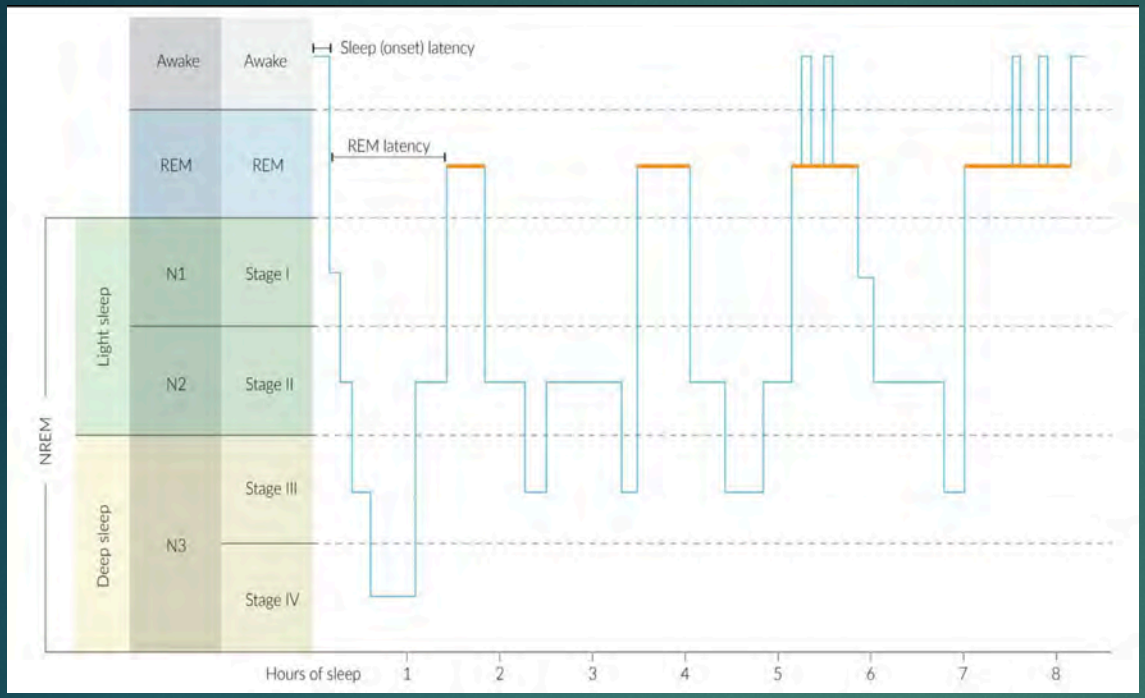
Factors Affecting Sleep in the ICU:

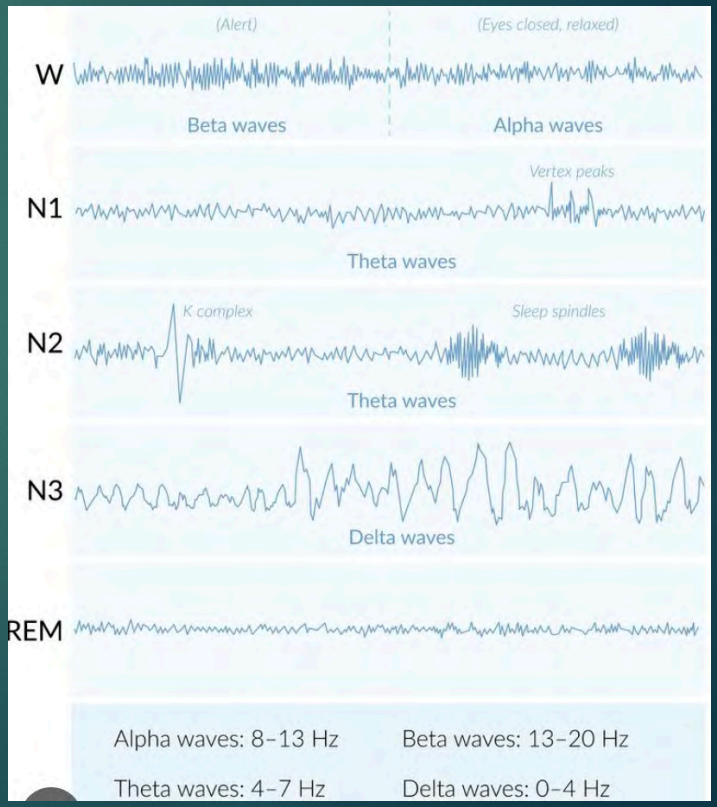
- Endogenous factors and disease processes
- Exogenous Stimuli

Sleep Versus Sedation

Strategies to Improve Sleep in ICU

NORMAL SLEEP ARCHITECTURE IN ADULTS





Sleep = Physiologically recurring state of rest with relative suspension of consciousness & inaction of voluntary muscles

Regulated by Circadian Rhythm (process C) and Homeostatic Drive (process S)

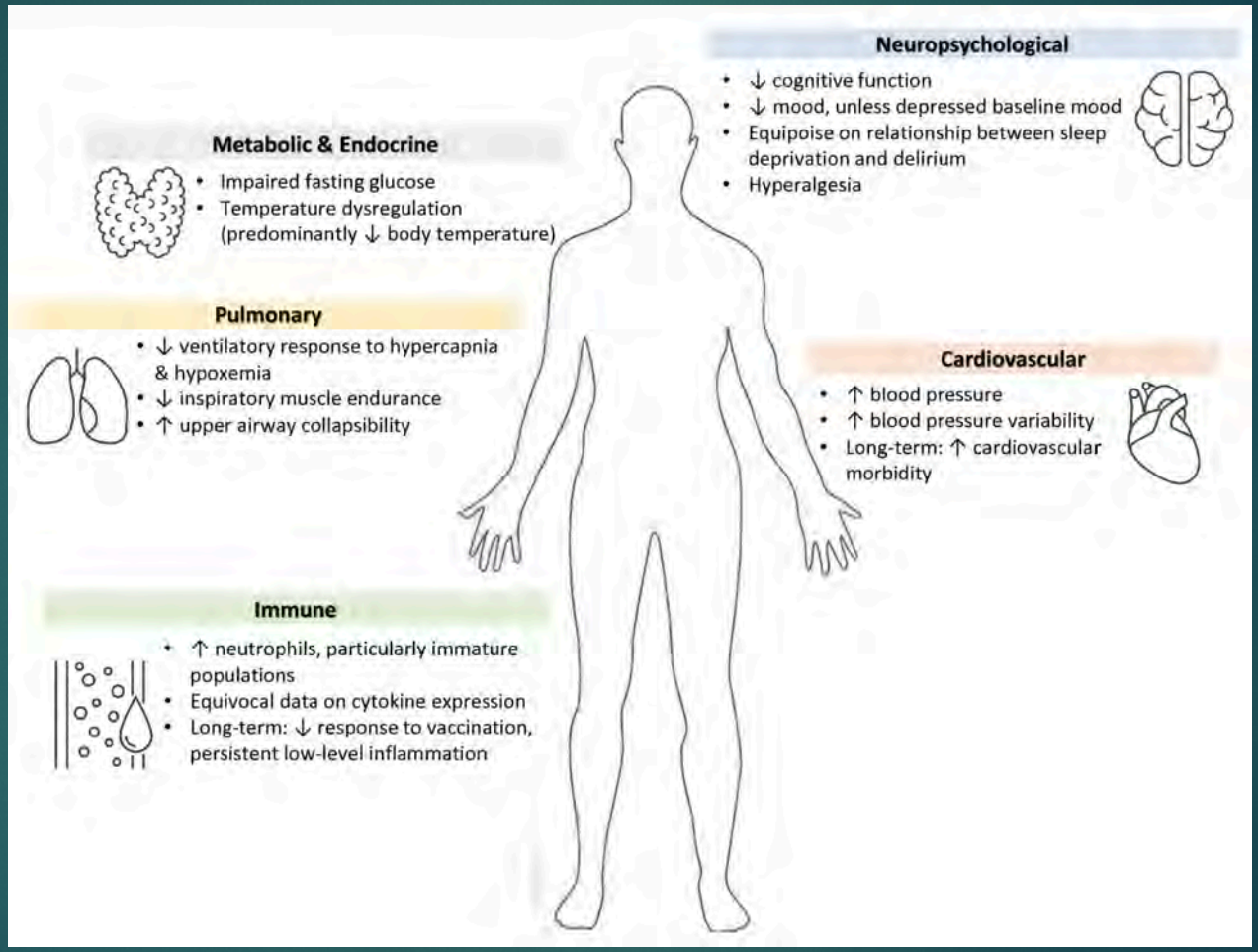
Usually consists of 4-5 sleep cycles

NORMAL CHANGES IN SLEEP ARCHITECTURE WITH AGING

- Decreased time spent in Stage III (deep sleep) and REM sleep
- Increased Sleep Latency
- More frequent nighttime awakenings (nocturia, musculoskeletal pain)
- Advanced Circadian Rhythm: tendency to sleep early and early morning awakening

Li J, Vitiello MV, Gooneratne NS. Sleep in Normal Aging. Sleep Medicine Clinics. 2017; 13 (1): p.1-11.

IMPACT OF SLEEP DEPRIVATION ON ORGAN SYSTEMS IN CRITICALLY ILL



Chang, V.A., Owens, R.L. & LaBuzetta, J.N. Impact of Sleep Deprivation in the Neurological Intensive Care Unit: A Narrative Review. Neurocrit Care ***32****, 596–608 (2020)*

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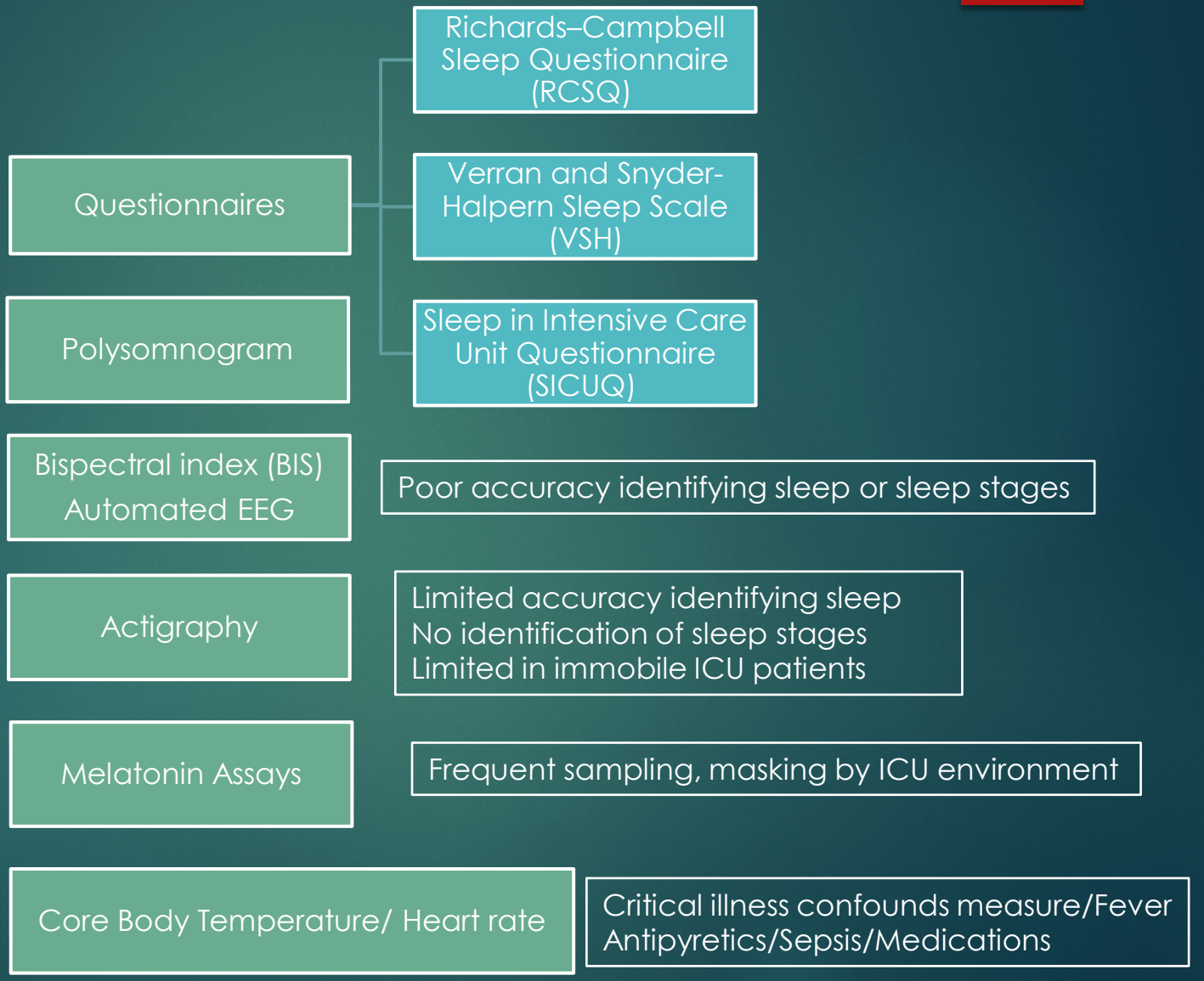
CAN WE ATTEMPT TO MEASURE SLEEP IN ICU ? CURRENT TOOLS



RICHARDS-CAMPBELL SLEEP QUESTIONNAIRE

- 33 patients (MICU)
- 92 patient-nurse assessments
- Mean Sleep Quality, RCSQ **57**
- Lowest score: Sleep depth
- Patient-nurse interrater reliability
  - -slight to moderate
- Nurses tend to overestimate
  - -Sleep quality c/w patients

Measure	Question[a]
1. Sleep depth	My sleep last night was: light sleep (0) … deep sleep (100)
2. Sleep latency	Last night, the first time I got to sleep, I: just never could fall asleep (0) … fell asleep almost immediately (100)
3. Awakenings	Last night, I was: awake all night long (0) … awake very little (100)
4. Returning to sleep	Last night, when I woke up or was awakened, I: couldn't get back to sleep (0) … got back to sleep immediately (100)
5. Sleep quality	I would describe my sleep last night as: a bad night's sleep (0) … a good night's sleep (100)
6. Noise[b]	I would describe the noise level last night as: very noisy (0) … very quiet (100)

[a] Each question is scored by using a 100-mm visual analog scale in which a higher score is better.
[b] Question 6 is not a part of the original 5-item Richards-Campbell Sleep Questionnaire (RCSQ), but was included in this project for consistency with other studies that used the RCSQ.[11]

Richards KC, et al. Measurement of sleep in critically ill patients. J Nurs Meas. 2000; 8(2):131–144
Kamdar, B. et al. Patient-Nurse Interrater Reliability and Agreement of the Richards-Campbell Sleep Questionnaire. Am J Crit Care. 2012 July ; 21(4): 261–269. doi:10.4037/ajcc2012111

CUMULATIVE SLEEP STAGE ANALYSIS IN CRITICALLY ILL VIA POLYSOMNOGRAM

37 ICU patients

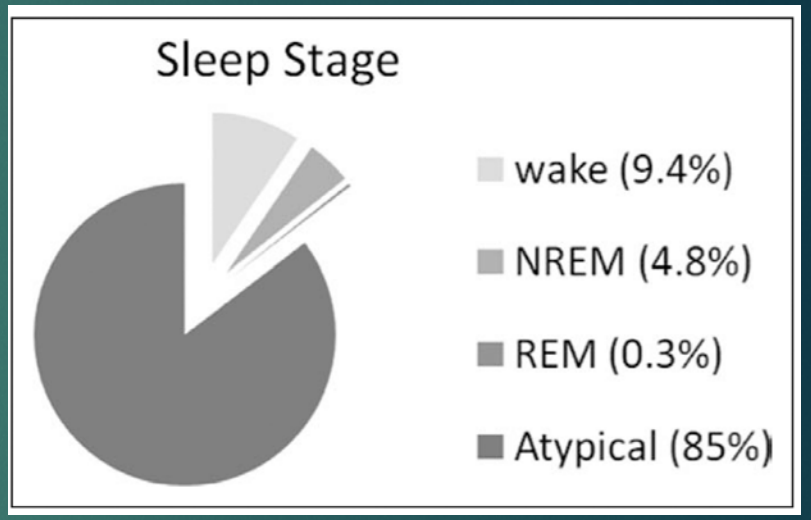
1,945.7 hrs of PSG data

NREM = non-REM sleep(stages I,II,III)

Atypical = could not be scored using gold standard Rechtschaffen & Kales scoring criteria

Lacking sleep spindles,

K complexes

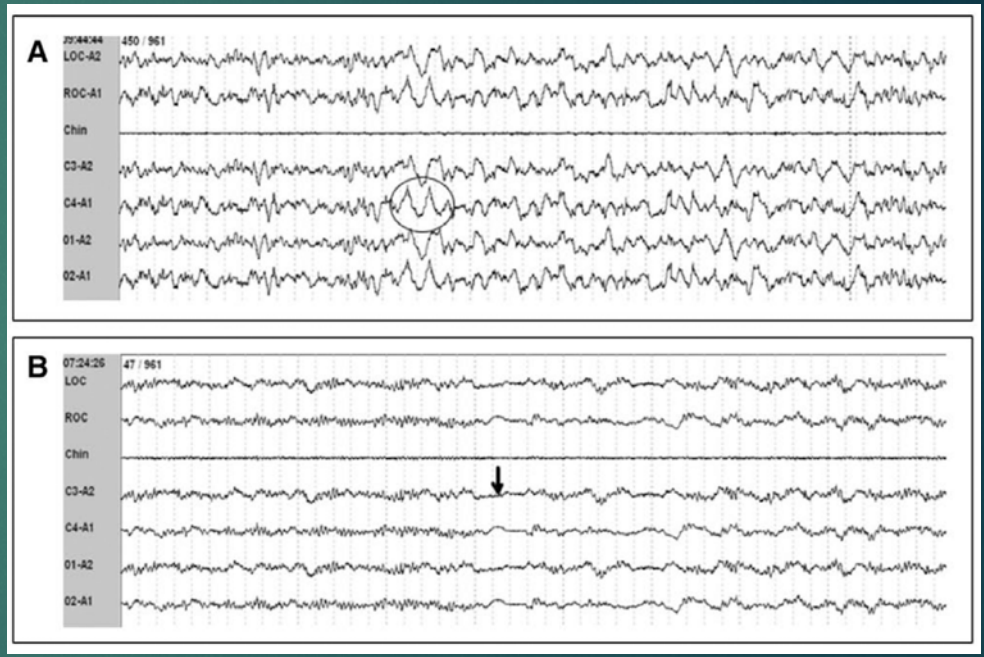


Watson, P et al. Crit Care Med. 2013 August ; 41(8): . doi:10.1097/CCM.0b013e31828a3f75.

DISSOCIATION OF EEG FINDINGS & SLEEP-WAKE STATES

A = Patient awake, following simple commands
Delta waves: N3/deep sleep
Pathologic wakefulness

B = Patient unresponsive
Theta waves: N1 sleep
Isoelectric activity



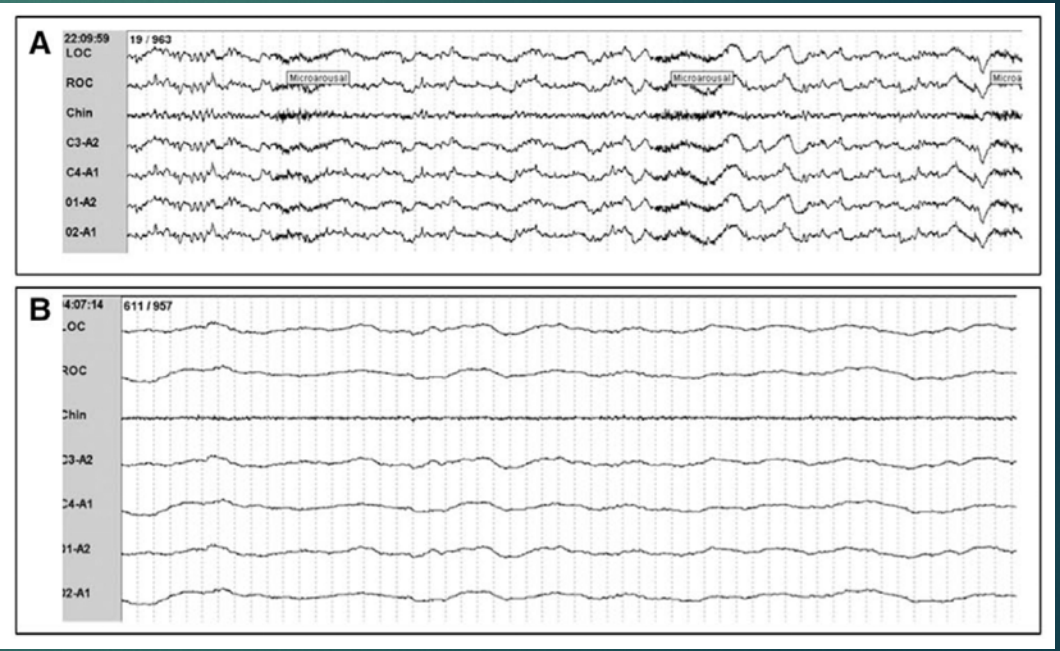
Watson, P et al. Crit Care Med. 2013 August ; 41(8): . doi:10.1097/CCM.0b013e31828a3f75.

VARIABILITY IN EEG CHARACTERISTICS IN COMATOSE PATIENTS ON PSGs

Both Patients A & B with RASS -5 (unresponsive to verbal/ physical stimuli)

A = Theta waves: N1 sleep
Micro arousals

B = Isoelectric activity



Sedation is NOT synonymous to Sleep

Watson, P et al. Crit Care Med. 2013 August ; 41(8): . doi:10.1097/CCM.0b013e31828a3f75.

CONCLUSION

Atypical PSG characteristics were prevalent

Absent: Cyclic progression of sleep stages and ultradian rhythm that is characteristic of sleep in healthy individuals

Inability to determine the sleep or wake states **solely** on EEG criteria

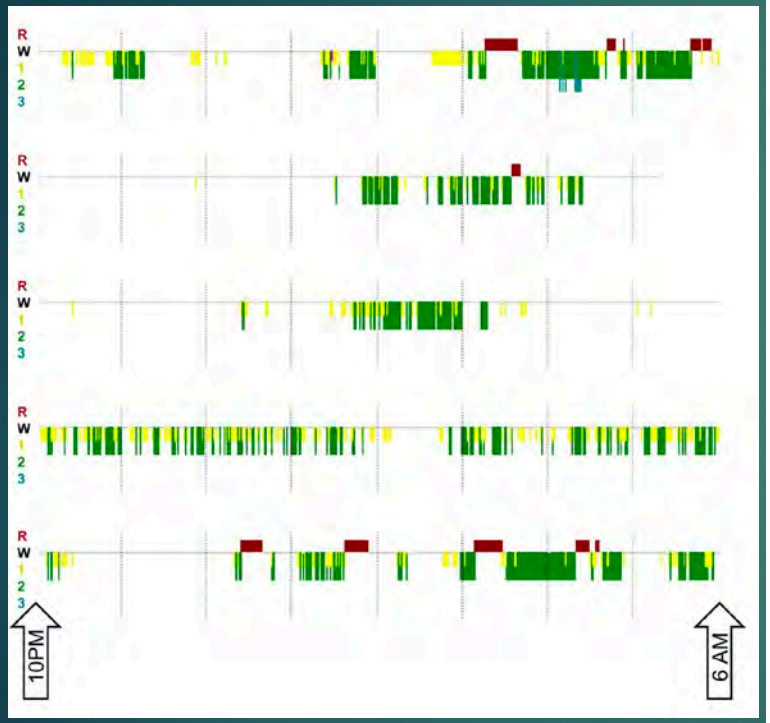
EEG frequencies of beta, alpha, theta, and delta were seen in **both the behavioral wake and sleep states**

In critically ill patients, **there is not a validated method** at this time for scoring sleep

Watson, P et al. Crit Care Med. 2013 August ; 41(8): . doi:10.1097/CCM.0b013e31828a3f75.

SLEEP ARCHITECTURE IN CRITICALLY ILL

PSG Hypnograms of 5 ICU patients



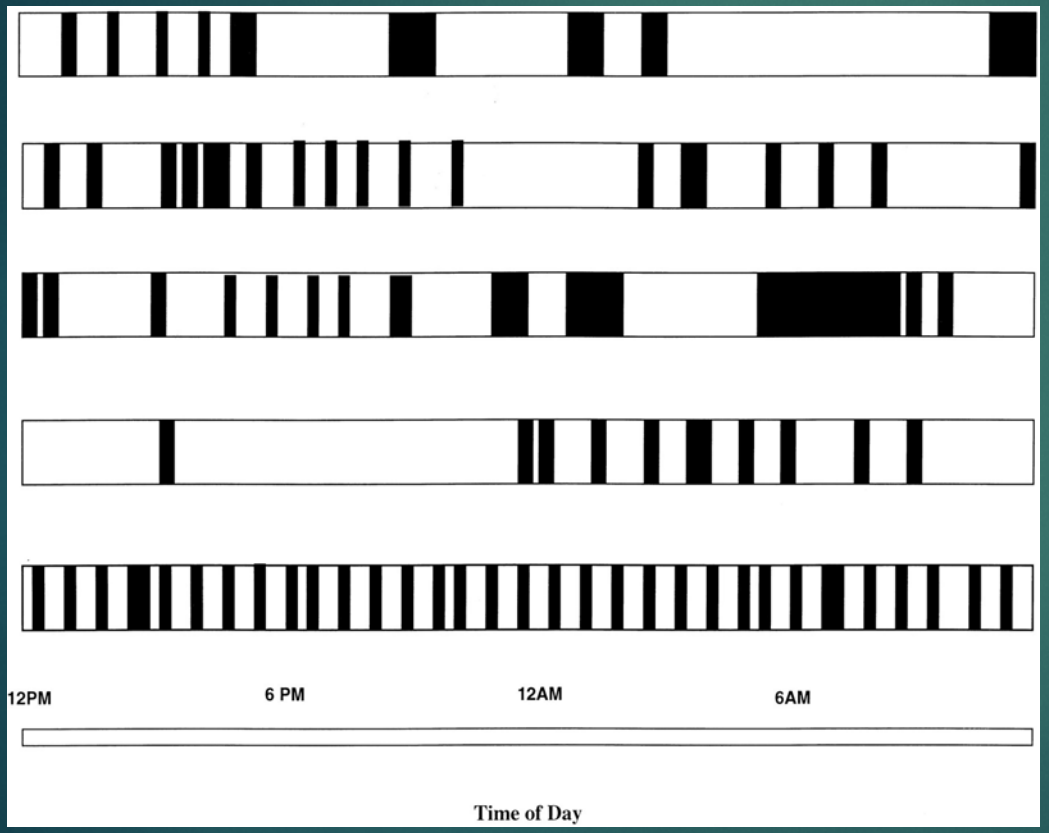
Sleep duration, architecture and arousal indices.

A. Sleep characteristics of MICU patients with Typical Sleep (n=14).	Whole Study Mean (SD)	Overnight Mean (SD)	Daytime Mean (SD)
Sleep Quantity			
Hours Available for Sleep	14.8 (4.1)	7.6 (1.2)	7.2 (3.4)
Hours of Total Sleep Time	6.2 (1.5)	4.2 (1.6)	2.0 (1.1)
Sleep Efficiency (%)	44.1 (13.7)	54.3 (18.2)	35.2 (25.5)
Sleep Architecture			
Proportion REM (%)	12.9 (14.2)	11.8 (12.2)	18.5 (24.3)
Proportion N1 (%)	22.7 (13.4)	23.4 (13.8)	24.3 (13.3)
Proportion N2 (%)	60.4 (12.3)	60.7 (11.4)	54.6 (20.5)
Proportion N3 (%)	3.9 (5.9)	4.1 (6.8)	2.4 (4.9)
Arousals			
NREM Arousal Index	33.0 (13.3)	32.3 (13.8)	34.6 (14.1)
REM Arousal Index	18.7 (16.1)	9.2 (7.3)	23.0 (17.5)
B. Sleep characteristics MICU patients with Atypical Sleep (n=9).			
	Whole Study Mean (SD)	Overnight Mean (SD)	Daytime Mean (SD)
Sleep Quantity			
Hours Available for Sleep	17.2 (5.3)	7.7 (0.9)	9.5 (4.7)
Hours of Total Sleep Time	4.8 (3.4)	3.0 (1.7)	1.7 (2.2)
Sleep Efficiency (%)	28.5 (16.8)	40.1 (21.7)	18.7 (14.8)
Sleep Architecture			
Proportion REM (%)	6.8 (6.0)	7.5 (10.5)	9.4 (10.0)
Arousals			
NREM Arousal Index (per hour)	26.5 (6.0)	27.4 (9.0)	23.4 (4.4)
REM Arousal Index (per hour)	12.4 (4.9)	21.4 (8.6)	4.0 (5.6)

Knauert M, et al. Heart Lung. 2014 ; 43(5): 445–452. doi:10.1016/j.hrtlng.2014.06.049.

REDISTRIBUTION OF SLEEP-WAKE CYCLE IN CRITICALLY ILL

24 hours of PSG Data



Black areas = Sleep

White areas = Awake

57 +/-18% (**Majority**) of Total Sleep Time (TST) was in **daytime**

Only 43 +/-18% of TST was in nighttime

Freedman N et al.; Am J Respir Crit Care Med 2001 163451-457. DOI: 10.1164/ajrccm.163.2.9912128

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Factors Affecting Sleep in ICU

Endogenous factors and disease processes:

- Sepsis
- Ventilators [invasive and non-invasive]
- Pain
- Anxiety/ Depression/stress of being sick and being in ICU
- Known hx of sleep disorders (OSA, insomnia, Circadian rhythm sleep disorders, RLS, dependence on sleeping aids)

Exogenous Stimuli:

- Noise [sounds of monitors/ventilator, outdoor noise (carts, talking)]
- Light
- Physical stimuli [suction, blood drawing, nebulizers, sequential compression, urethral, rectal devices]
- Medication use

SOUND IN THE ICU

WHO and EPA recommends that continuous background sound in patient treatment areas not to > 30 dB & peak nocturnal sounds to remain < 40 dB

Reality however is sounds of:

- ventilator (51 dB)
- suction (53 dB)
- syringe pump alarms (63 dB)

ICU studies involving sound report dB levels exceed recommended limits with:

Mean 53-65 dB; Peak > 80 dB in 24 hrs

Freedman N et al.; Abnormal sleep/wake cycles and the effect of environmental noise on sleep disruption in the intensive care unit. AJRCCM 2001 163451-457.
Gabor J, et al. Contribution of the intensive care unit environment to sleep disruption in mechanically ventilated patients and healthy subjects. AJRCCM 2003;167:708-15.

WHAT DISTURBS PATIENTS' SLEEP THE MOST ?

50 Males + 50 Females

at least 2 nights in ICU post-extubation

Fully attentive completed an ICU sleep questionnaire

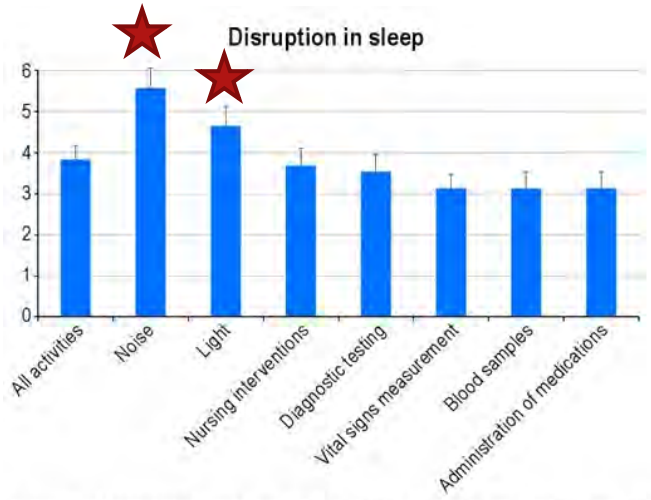


Figure 2: Mean perceived level of disruption of sleep quality from various ICU activities

1 = no disruption to 10 = significant disruption. Bars represent upper 95% confidence limits.

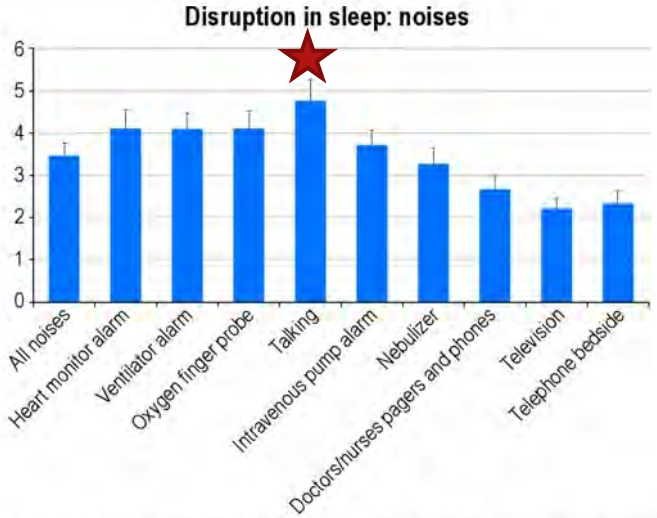


Figure 3: Mean perceived disruption in sleep quality from various sources of noise

Disruptions rated on a scale of 1 = no disruption to 10 = significant disruption. Bars represent upper 95% confidence limits.

Bihari S, et al. Factors affecting sleep quality of patients in intensive care unit. Clin Sleep Med. 2012 Jun 15;8(3):301-7

LIGHT IN THE ICU

Sleep-wake pattern disturbance primarily due to:

- Elevated light levels through-out the night (5-1400 lux)
  - 100-500 lux can suppress endogenous melatonin secretion
- Bright bursts interrupting nighttime darkness
- On the contrary, ICU daytime light levels (42 to 158 lux) w/c is insufficient daytime light

Activity associated with the greatest amount of light exposure at night:

- Obtaining blood samples
- **Light was on due to <u>"NO"</u> reason at all**

Dunn H, et al. Nighttime lighting in intensive care units. Crit Care Nurse. 2010;30(3):31-37
Boivin DB, et al. Dose–response relationships for resetting of human circadian clock by light. Nature.1996;379(6565):540-542

EFFECT OF MECHANICAL VENTILATION ON SLEEP

PSG, Respiratory Mechanics & $EtCO_2$

were measured in 11 mechanically ventilated patients

AC, PS, PS with dead space

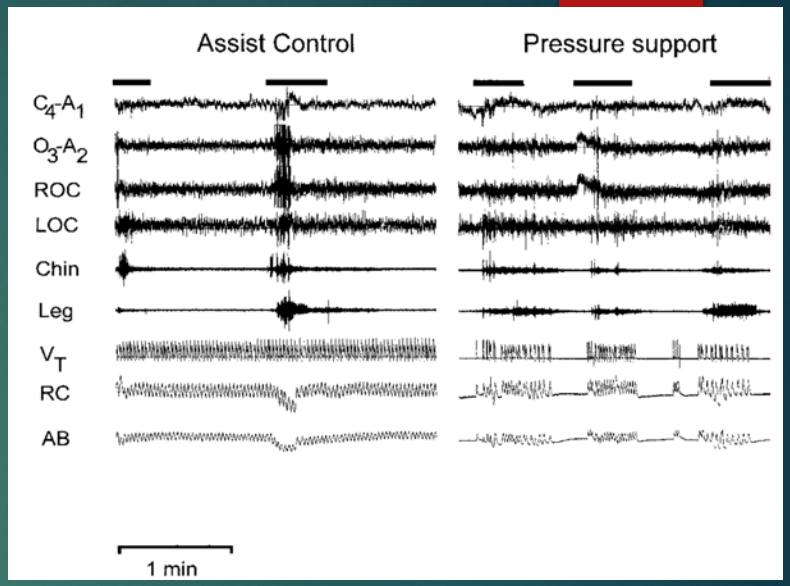
Conclusions

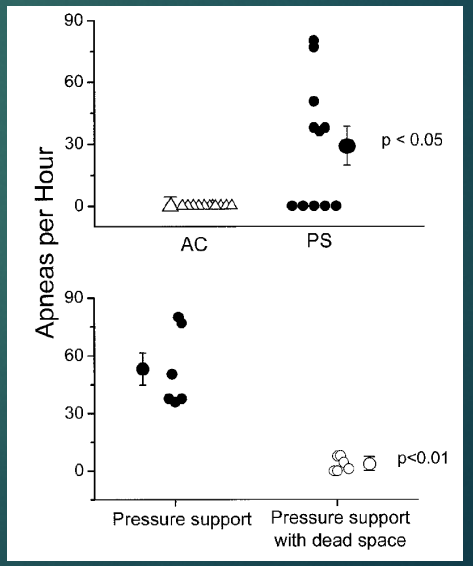
Mech ventilated pts had significant arousals (EEG) during all modes

PS mode further aggravated the fragmented sleep (awakening + arousals)

Central apneas were noted during PS and worsened awakenings: correlated with $EtCO_2$

(Hypocapnia)

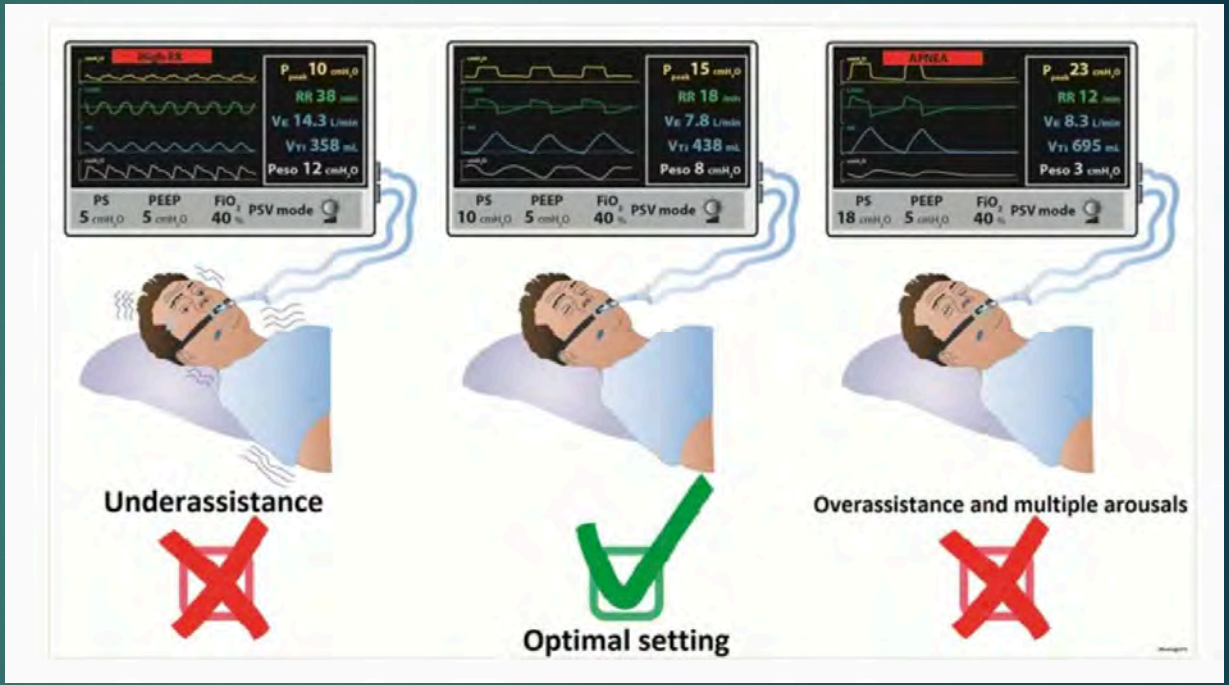




Parthasarathy S, et al. Effect of ventilator mode on sleep quality in critically ill patients. AJRCCM.2002 1;166(11):1423-9
Cooper AB, et al. Sleep in critically ill patients requiring mechanical ventilation. Chest. 2000 Mar;117(3):809-18

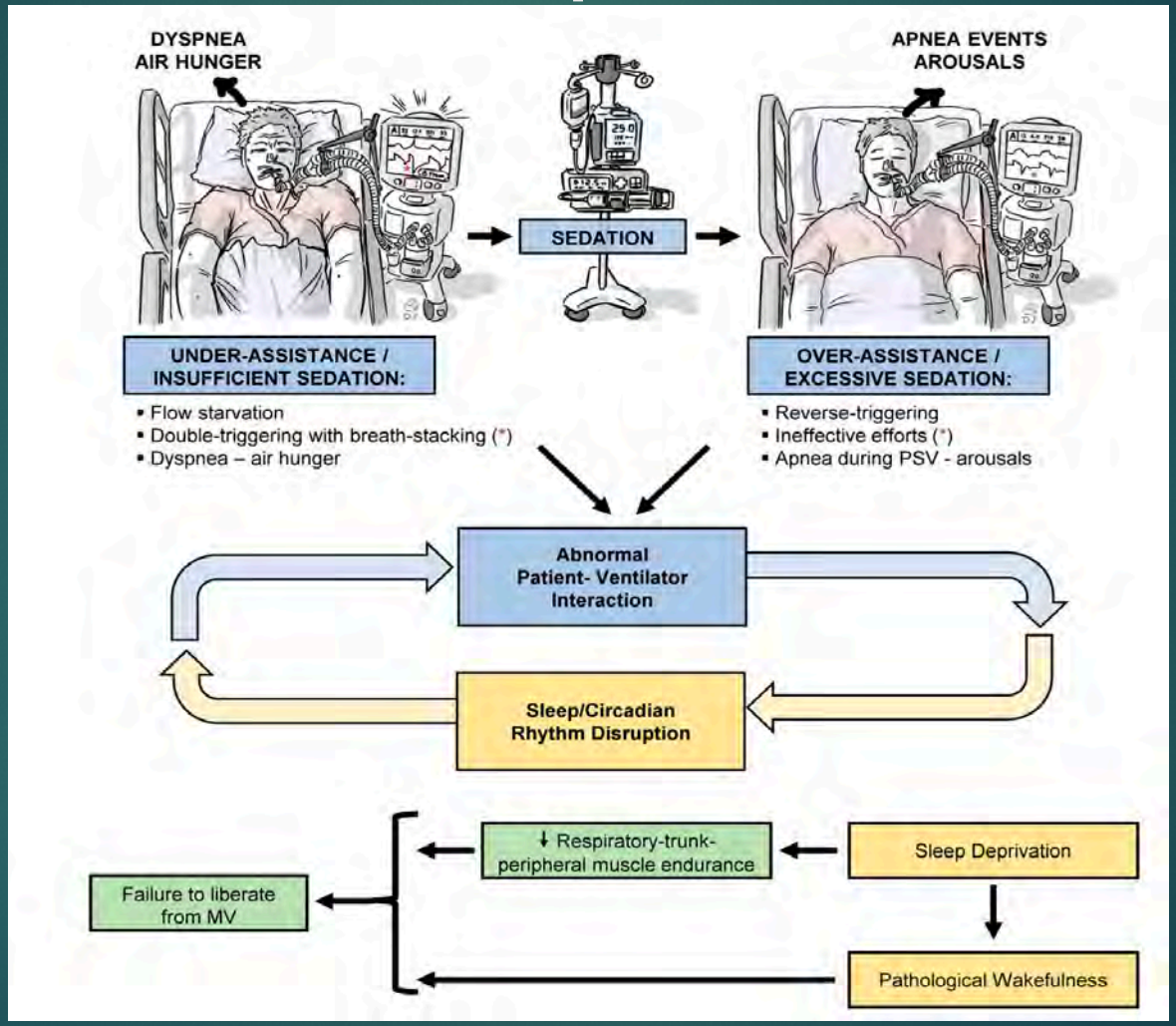
EFFECT OF PRESSURE SUPPORT VENTILATION ON SLEEP

Mode of Ventilation VS Achievement of Physiologic Principles
-Synchrony
-Resp muscle rest
-Relief of dyspnea



Rittayamai, N., Wilcox, E., Drouot, X. et al. Positive and negative effects of mechanical ventilation on sleep in the ICU: a review with clinical recommendations. Intensive Care Med 42, 531–541 (2016)

Proposed Bidirectional Relationship b/w Respiratory Dysfunction and Sleep/Circadian disruption



Knauert MP, et al. Causes, Consequences, and Treatments of Sleep and Circadian Disruption in the ICU: An Official American Thoracic Society Research Statement. Am J Respir Crit Care Med. 2023 Apr 1;207(7):e49-e68

EFFECT OF SEPSIS ON SLEEP-WAKE STATES

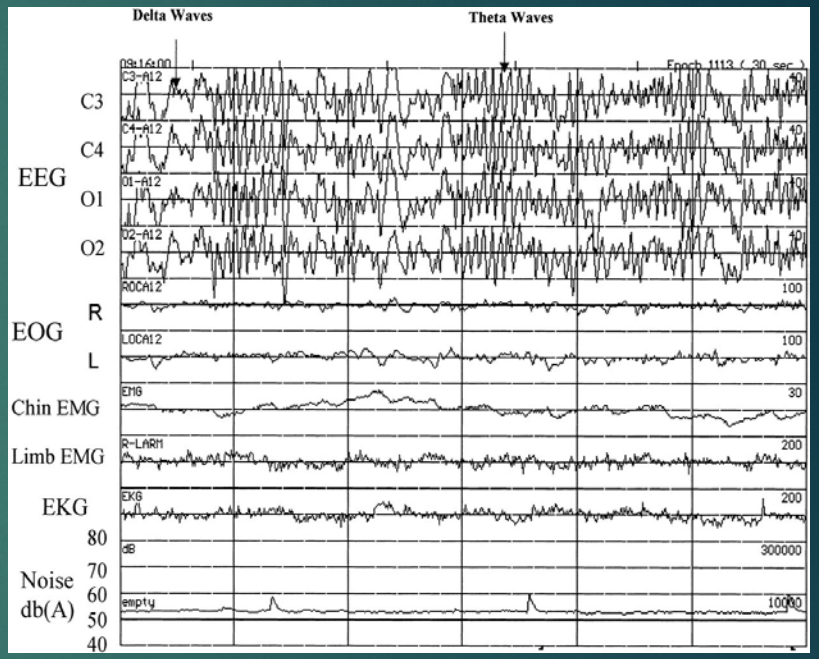
↑ sleep promoting cytokines
TNF, IL-1β

↓ REM sleep

↑ NREM sleep

Altered EEG: low-voltage, mixed-frequency waves with variable theta and delta ("septic encephalopathy")

Loss of normal circadian melatonin secretion



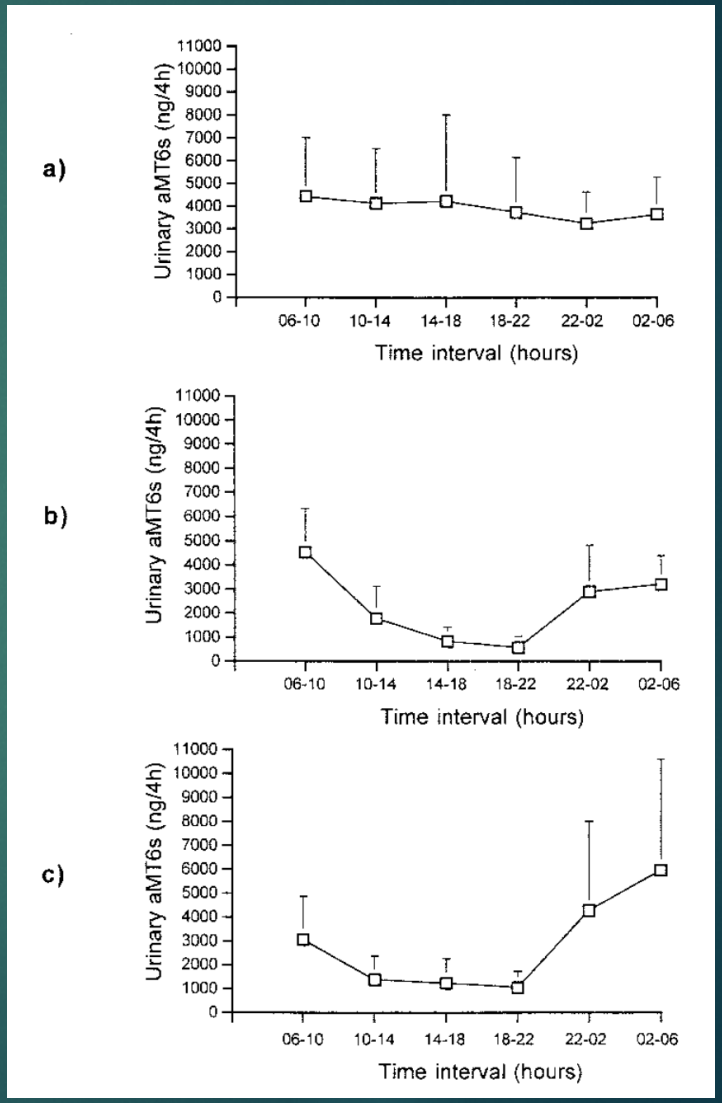
Freedman, N et al. Am J Respir Crit Care Med 2001 163451-457. DOI: 10.1164/ajrccm.163.2.9912128

MELATONIN SECRETION IN CRITICALLY ILL PATIENTS WITH SEPSIS

a = Critically ill patients with severe sepsis

b = Critically ill patients without sepsis

c = Age-Matched healthy volunteers



Mundigler G, et al. Impaired circadian rhythm of melatonin secretion in sedated critically ill patients with severe sepsis. Crit Care Med. 2002 Mar;30(3):536-40.

EFFECT OF COMMON ICU MEDICATIONS ON SLEEP

Benzodiazepines (BDZ), antipsychotics, opiates ↓REM

Abrupt withdrawn can cause REM rebound (nightmares/vivid dreams)

Withdrawal of β-blocker or a-agonist (clonidine) -> ↑ sympathetic activity -> ↑ sleep fragmentation

β-adrenergic receptor agonists

And steroids can cause insomnia

Amiodarone can cause nightmares/sleep fragmentation

Table 1 Commonly used medications and effects on sleep

Drug class or individual drug	Effects on sleep
Benzodiazepines	↓REM, ↓SWS
Opiods	↓REM, ↓SWS
Norepinephrine/epinephrine	↓REM, ↓SWS
Dopamine	↓REM, ↓SWS
β-Blockers	↓REM, ↓SWS
Corticosteroids	↓REM, ↓SWS
Selective serotonin, reuptake inhibitors	↓REM, ↓TST, ↓SE

REM, rapid eye movement; SE, sleep efficiency; SWS, slow-wave sleep; TST, total sleep time.

Weinhouse GL; Schwab RJ. Sleep in the critically ill patient. SLEEP 2006;29(5): 707-716.
Bijwadia J S. et al. Sleep and Critical Care. Curr Opin Crit Care 2009, 15:25–29

SLEEP AND SEDATION

Biological need for sleep & therapeutic need for sedation almost universally coexist in critically ill patients (via propofol/ BDZ)

Similarities

- Overlapping neurophysiologic pathways
- Muscle hypotonia
- Temperature dysregulation
- Disconjugate eye movements (REM)
- Altered sensorium and mentation
- Respiratory depression

Differences

- Sleep is spontaneous; sedation is not
- Sleep is circadian; sedation is not
- Sleep is an essential biologic function; sedation is not
- Sleep is completely reversible with external stimuli; sedation is not
- Sleep ↓ release of Norepi from locus coeruleus; Norepi release continues during sedation
- Sleep is associated with cyclic progression of EEG stages; sedation variably alters normal sleep architecture

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PHARMACOLOGIC INTERVENTIONS COMMONLY USED IN ICU TO IMPROVE SLEEP

- Dexmedetomidine
- Propofol
- Melatonin
  - Melatonin Receptor Agonists
- Trazadone
- Opiates
- Antihistamine
- Benzodiazepines
- Hypnotics: (similar to BDZ)
  - zolpidem, eszoplicone, zaleplon
- Typical Antipsychotic:
  - Haloperidol
- Atypical Antipsychotics:
  - Olanzapine, Quetiapine

EFFECTS OF PHARMACOLOGICAL INTERVENTIONS ON SLEEP

Medications commonly used to promote sleep

Medication	Mechanism of action	Route of administration	Side effects	Sleep effects
Dexmedetomidine	*a*2-agonist	Intravenous	Bradycardia, hypotension	↑N2 with sleep spindles, ?↑N3/SWS, ↓REM, ↑SE, ↓SL
Propofol	GABA receptor agonist	Intravenous	Bradycardia, hypotension, propofol infusion syndrome, respiratory depression	↓REM, ↓SL, ↑TST, ↓W ★
Opiates	CNS opioid receptor agonist	Oral or Intravenous	Dependency, delirium-inducing, hypotension, respiratory depression, withdrawal	↓N3, ↓REM, ↓TST, ↑W ★
Melatonin and melatonin receptor agonists	Melatonin 1 and 2 receptor agonist	Oral	Dizziness, hallucinations, nausea, vivid dreams	↑SE, ↓SL, ↑TST
Atypical antipsychotics	$5HT_2$, D_2–receptor antagonist	Oral	Dizziness, extrapyramidal Symptoms, neuroleptic malignant syndrome, orthostatic hypotension	↑N3, ↑REM, ↑SE, ↓SL, ↑TST, ↓W ★
Typical antipsychotics	Dopamine receptor antagonist	Oral or intravenous	Anticholinergic effects, extrapyramidal symptoms, neuroleptic malignant syndrome, QT prolongation, tardive dyskinesia	↑N2, ↑N3, ↑SE, ↓SL, ↑TST, ↓W
Trazodone	Serotonin reuptake inhibitor, 5-HT1A,1C,2, H1 receptor antagonist	Oral	Anticholinergic syndrome, arrhythmias, orthostatic hypotension	↑N3, ↑↓REM, ?↑SE, ↓SL
Antihistamines	H1-receptor antagonist	Oral or intravenous	Anticholinergic syndrome, dizziness, impaired coordination	?↑N3, ↓REM, ?↑SE, ↓SL
Benzodiazepines	GABA receptor agonist	Oral or intravenous	Dependency, delirium-inducing, dizziness, hypotension, withdrawal	↓N3, ↓REM, ↓SL, ↑TST, ↓W ★
Nonbenzodiazepine hypnotics	GABA receptor agonist	Oral	Daytime somnolence, dizziness, confusion	↓N2, ↓N3, ↑↓REM, ↓SL ↑TST, ↓W ★

Abbreviations: CNS, central nervous system; GABA, gamma-aminobutyric acid; N2, deeper sleep; N3/SWS, restorative, slow wave sleep; REM, rapid eye movement; SE, sleep efficiency; SL, sleep latency; TST, total sleep time; W, wake; ↓, decreased; ↑, increased; ↑↓, equivocal; ?↑, may increase; ?↓, may decrease.

Opiates, BDZ and non-BDZ hypnotics: ↓ N3/Deep sleep
Opiates, BDZ, Propofol: ↓ REM sleep

Dorsch et al. Semin Respir Crit Care Med. 2019 October ; 40(5): 614–628. doi:10.1055/s-0039-1698378.

MELATONIN AND MELATONIN-RECEPTOR AGONIST

Trials of melatonin and ramelteon (melatonin-receptor agonist) in critically ill patients

Medication	First author year	Study type	Sample size	Population	Dose (mg)	Medication timing	Assessment	Results
Melatonin	Shilo[176] 2000	RCT	8	ICU	3	22:00	Actigraphy	↑ Sleep quality ↑ sleep time
	Ibrahim[177] 2006	RCT	32	ICU tracheostomy	3	20:00	Nurse observation	No change in nocturnal sleep duration
	Bourne[178] 2008	RCT	24	ICU tracheostomy	10	21:00	BIS	↓ BIS AUC ↑ sleep quality ↑ sleep time
	Mistraletti[179] 2010	RCT	82	Mixed ICU mechanically ventilated	3 and 3	20:00 and 00:00	Nurse observation	↑ Nocturnal TST
	Huang[131] 2015	NR	40	Healthy volunteers subjected to ICU environment	1	21:00	PSG	↑ REM ↑ TST ↓ SOL ↓ awakenings
	Foreman[101] 2015	RCT	12	Neuro ICU	3	20:00	EEG	UTD; only one patient in each group had scorable sleep
Ramelteon	Hatta[173] 2014	RCT	67	Elderly patients ICU and general wards	8	21:00	DRS-R-98	↓ Delirium
	Nishikimi[174] 2018	RCT	88	ICU	8	20:00	CAM-ICU nurse observation	↓ Delirium ↓ nocturnal awakenings Trend toward ↓ ICU LOS

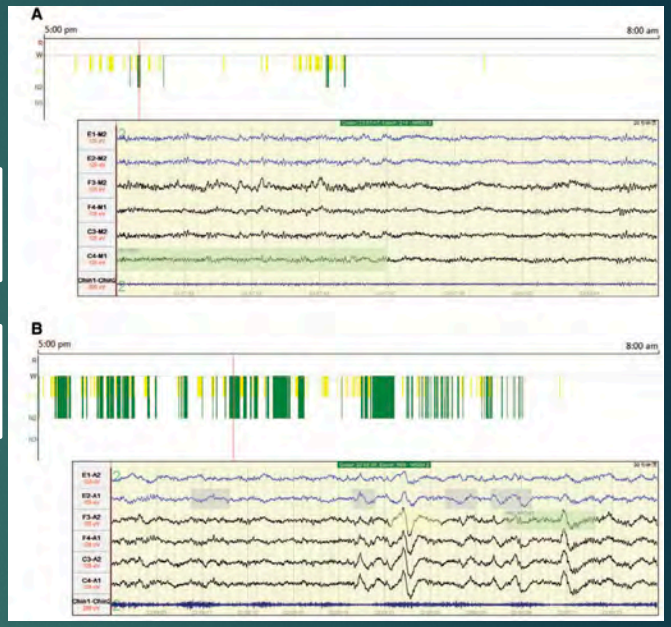
Abbreviations: BIS, bispectral index area; CAM-ICU, confusion assessment method-ICU; DRS-R-98, delirium rating scale-revised-98; EEG, electroencephalogram; ICU, intensive care unit; LOS, length of stay; NR, nonrandomized; PSG, polysomnography; RCT, randomized control trial; SE, sleep efficiency; SOL, sleep onset latency; TST, total sleep time; WASO, wake after sleep onset.

Dorsch et al. Semin Respir Crit Care Med. 2019 October ; 40(5): 614–628. doi:10.1055/s-0039-1698378.

DEXMEDETOMIDINE and SLEEP

Age > = 65 yrs

- Randomized Post surgical, Non-ventilated
- 38 patients received15 hrs:
  - Placebo
  - Dexmedetomidine 0.1 µg/kg/hr
- Completed PSGs
- **Dexmedetomidine ↑ % stage II sleep, ↑sleep time, ↑SE, ↓ stage I**



Representative PSG
A = Placebo
B = Dexmedetomidine

Wu Xin-Hai, et al. Low-dose Dexmedetomidine Improves Sleep Quality Pattern in Elderly Patients after Noncardiac Surgery in the Intensive Care Unit: A Pilot Randomized Controlled Trial. Anesthesiology 2016; 125:979–991

A MULTICOMPONENT MULTIDISCIPLINARY BUNDLE OF INTERVENTIONS ON SLEEP AND DELIRIUM

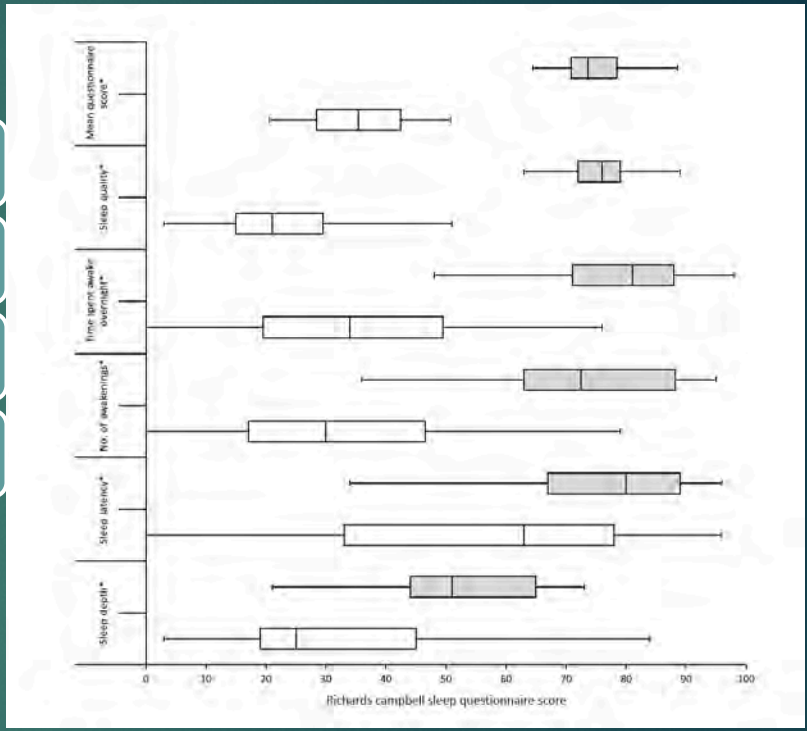
59 ICU patients completed RCSQ

Before (30 pt);After Sleep Intervention(29 pt)

RCSQ Higher score = Positive response

p<0.05:

- Sleep depth
- Sleep latency
- No. of awakenings
- Time spent awake overnight
- Sleep quality
- Mean RCSQ score



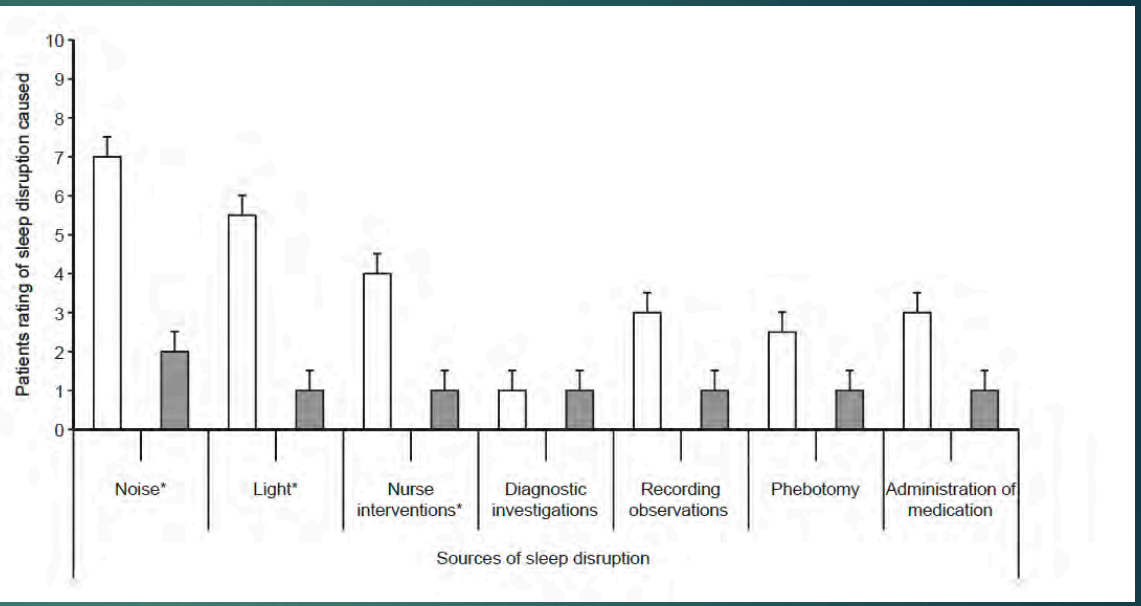
Patel, J. et al. The effect of a multicomponent multidisciplinary bundle of interventions on sleep and delirium in medical and surgical intensive care patients. Anaesthesia 2014, 69, 540–549

Patient rating of sleep disruption

Higher score = ↑ disruption

White bars = before intervention

Grey bars = after intervention



$p<0.05$:

- Noise
- Light
- Nurse interventions

Patel, J. et al. The effect of a multicomponent multidisciplinary bundle of interventions on sleep and delirium in medical and surgical intensive care patients. Anaesthesia 2014, 69, 540–549

CONCLUSION

Compliance with the multicomponent bundle of interventions was **> 90%**

↑ in sleep efficiency (SE)

↑ in sleep quality

↓ **in daytime sleepiness**

↑ **sleep at night**

↑ patient nights contained a 3hr window of un-interrupted sleep

↓ **in Incidence of Delirium** (33% vs 14%)

↓ in mean length of time spent delirious (3.4 vs 1.2 days)

Those reporting ↑ SE showed a ↓ risk of Delirium (O.R 0.9 95% CI 0.84-0.97)

Clinical Practice Guidelines for the Prevention and Management of Pain, Agitation/Sedation, Delirium, Immobility, and Sleep Disruption in Adult Patients in the ICU

Question: **Should a sleep-promoting protocol be used to improve sleep in critically ill adults?**

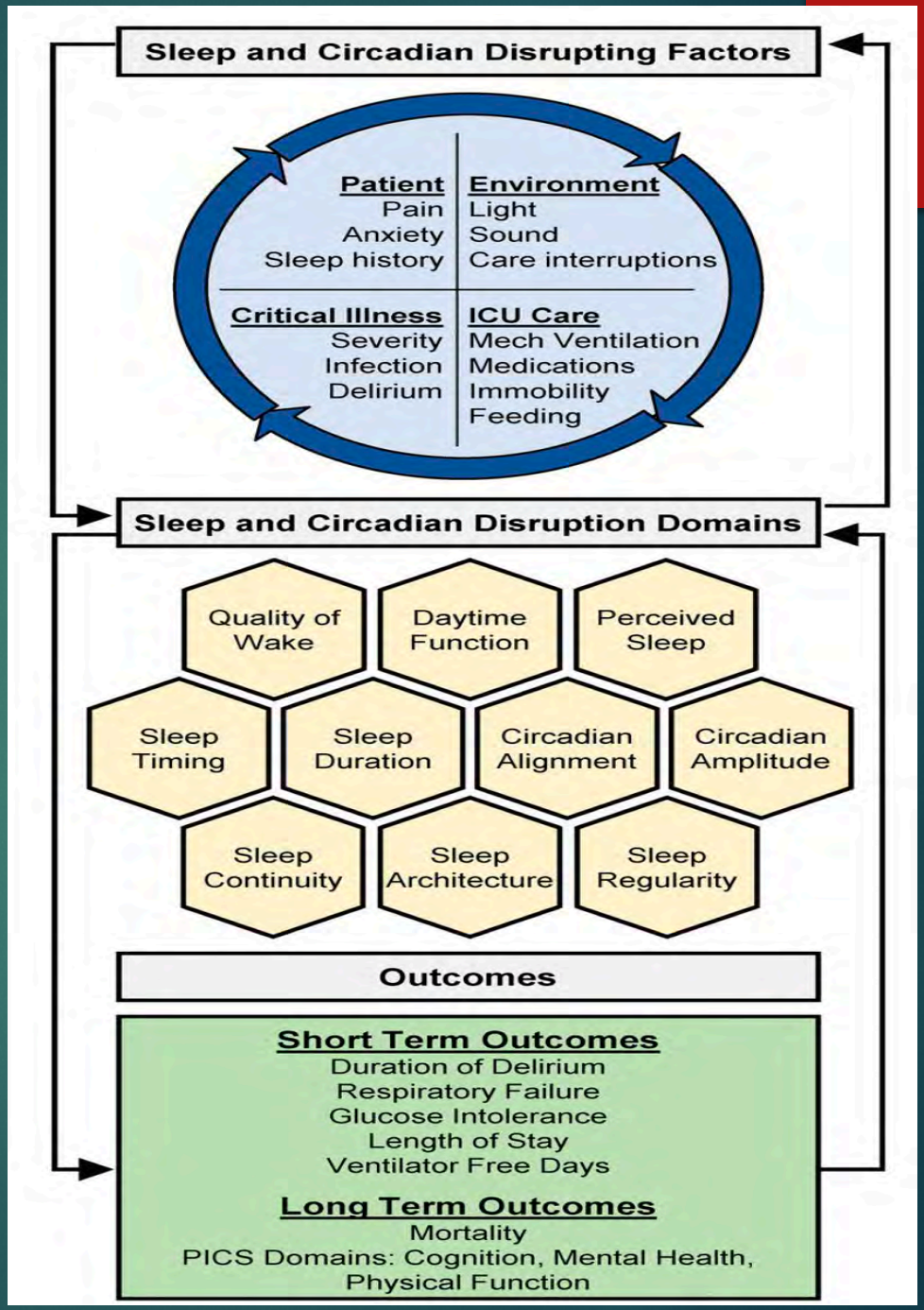
Recommendation: **We suggest using a sleep-promoting, multicomponent protocol in critically ill adults**

Causes, Consequences, and Treatments of Sleep and Circadian Disruption in the ICU
An Official American Thoracic Society Research Statement

Clinical Practice Guidelines for the Prevention and Management of Pain, Agitation/Sedation, Delirium, Immobility, and Sleep Disruption in Adult Patients in the ICU. Critical Care Medicine 46(9):p e825-e873, Sep.2018.

Knauert MP, et al. Causes, Consequences, and Treatments of Sleep and Circadian Disruption in the ICU: An Official American Thoracic Society Research Statement. Am J Respir Crit Care Med. 2023 Apr 1;207(7):e49-e68.

Conceptual Model of ICU Sleep and Circadian Disruption



Knauert MP, et al. Causes, Consequences, and Treatments of Sleep and Circadian Disruption in the ICU: An Official American Thoracic Society Research Statement. Am J Respir Crit Care Med. 2023 Apr 1;207(7):e49-e68.

WHAT CAN WE DO TO IMPROVE SLEEP? SCRIPPS EXPERIENCE

ENGAGEMENT, EDUCATION & COMMITMENT OF RELEVANT DEPARTMENTS:

A COLLOBORATIVE APPROACH AMONG ALL STAKEHOLDERS WHO WORK IN ICU

NURSING/

PHYSICAL/ OCCUPATIONAL THERAPISTS

RESPIRATORY THERAPISTS

PHYSICIANS/ PROVIDERS

RADIOLOGY

LABORATORY/ PHLEBOTOMY

HOUSEKEEPING

PHARMACY/ DIETARY SERVICES

INFORMATION SERVICES/ LIGHTING/OVERHEAD PAGING

WHAT CAN WE DO TO IMPROVE SLEEP?

Optimize Daytime Physical/Mental Activities

- Turn on room lights/open blinds (indoor sunshine therapy)
- Minimize daytime sedation
- Minimize daytime sleeping
- Promote mobilization
- Avoid caffeine after 1 pm
- **Ask patients about their sleep during rounds**
- Daytime time-restricted feeding

SLEEP HYGIENE NIGHTLY ROUTINE

- Optimize time of un-interrupted sleep 2200 – 0500
- Turn off OR dim lights; Turn off television
- Close window blinds
- Minimize noise; reduce alarm level to safe audible level for RN
- Offer ear plugs and eye mask
- Improve room temp. to patient comfort
- Ensure pain/anxiety is controlled

- **Treat pre-existing sleep disorders (OSA/RLS)**
- Cluster activities/nebulizers/medications
- Consider most optimal mech ventilation
- Garbage/linen collection before 2100
- Differ bathing/sponge bath until 0500
- Differ blood drawing/imaging until 0500

THANK YOU FOR YOUR ATTENTION!

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