"SAFE MECHANICAL VENTILATION: "WHAT YOU NEED TO KNOW "...AND DO.

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DISCLOSURES

Past and present Advisory Boards:

- Resmed
- Philips/Respironics
- Hamilton

OBJECTIVES:

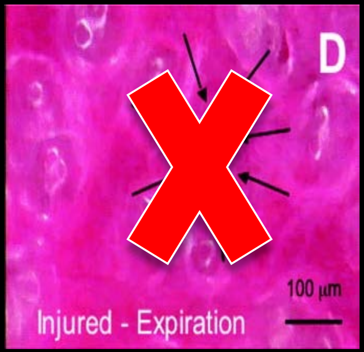
- Describe the physiology of patient (PILI) and/or ventilator induced lung injury (VILI)
- Discuss current evidence of potentially modifiable factors that may limit Lung injury.
- Describe a rational approach to providing safe ventilatory support.

VENTILATORY SUPPORT GOALS

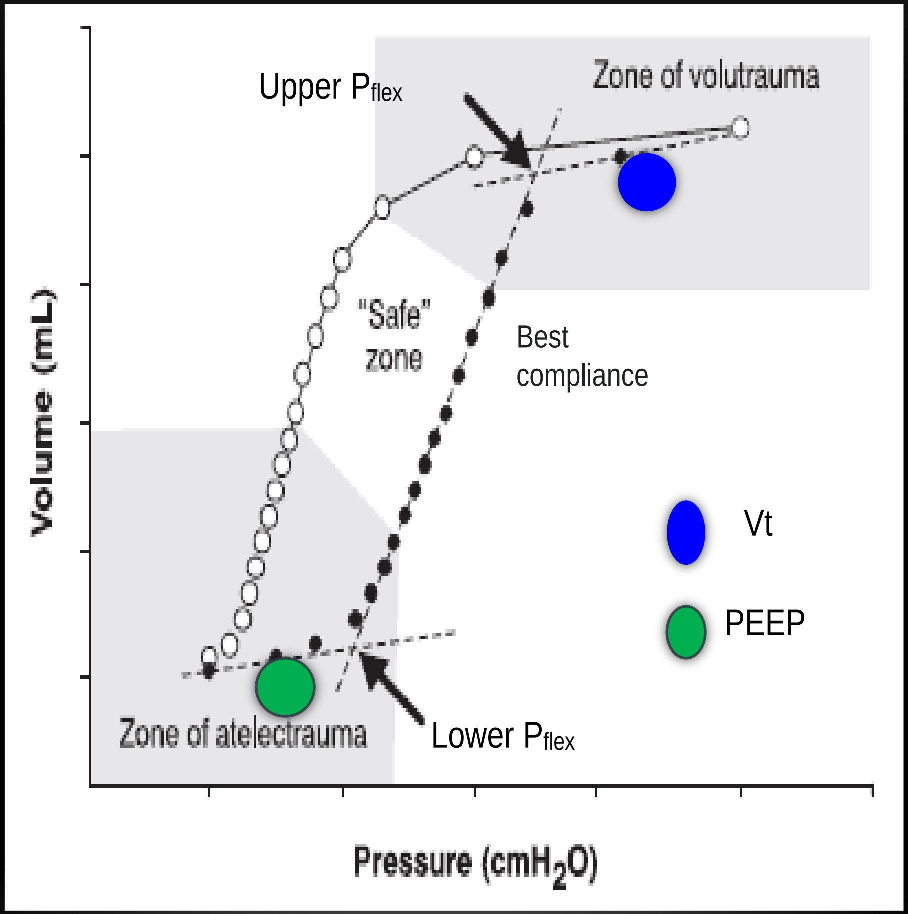
1. Maintain adequate gas exchange
2. Provide appropriate level of support
   a) Limit excessive work of breathing
   b) Pt comfort
   c) Pt/vent synchrony

3. <u>Protect the lung</u>
   a) Limit stress and strain

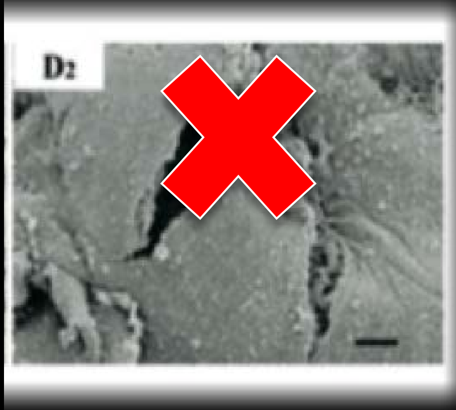
GOALS OF LUNG PROTECTIVE STRATEGY



Cyclic injury



Overdistention

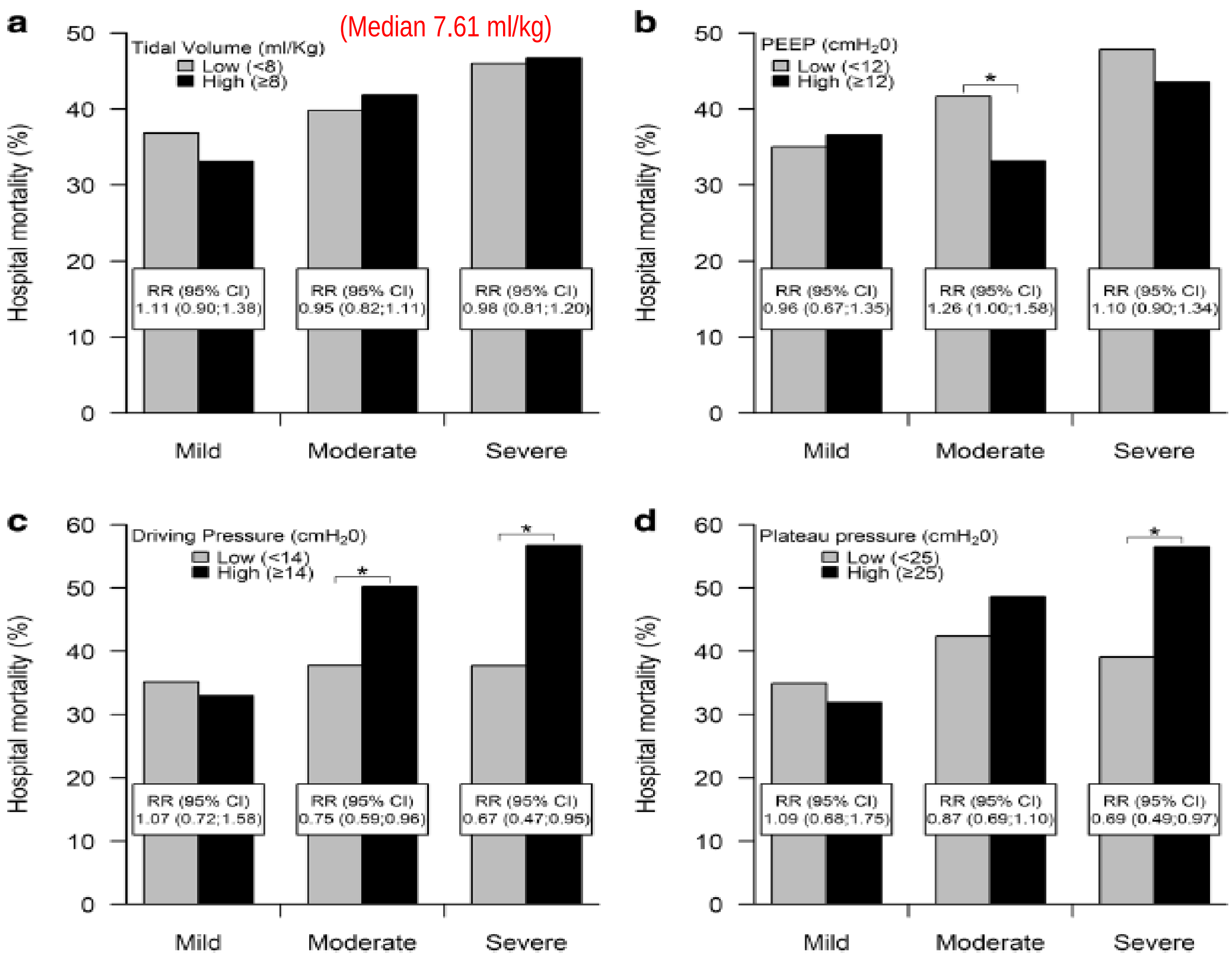


R. Oeckler

Potentially modifiable factors contributing to outcome from acute respiratory distress syndrome: the LUNG SAFE study

John G. Laffey
The LUNG SAFE Investigators

Intensive Care Med
DOI 10.1007/s00134-016-4571-5



Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Epidemiology, Patterns of Care, and Mortality for Patients With Acute Respiratory Distress Syndrome in Intensive Care Units in 50 Countries

JAMA. 2016;315(8):788-800.

LUNG SAFE Investigators and the ESICM Trials Group

- 50 countries, 459 ICUs, 29144 patients receiving mechanical ventilation
- ARDS incidence 10.4 % (3022 patients)
- Recognition of ARDS 60% (40% under recognized)
- Less than 2/3 of ARDS pts received Vt 8ml/kg PBW or less (1/3 received Vt too high)
- 82.6% received PEEP of 12 cmH20 or less (PEEP too low)
- Plateau pressure only monitored in 40.1% (Driving pressure?)
- Mortality ranged from 34.9, 40.3, 46.1 % for mild, moderate, and severe ARDS respectively

CONCLUSIONS AND RELEVANCE Among ICUs in 50 countries, prevalence of ARDS was 10.4% of ICU admissions. This syndrome appeared to be under recognized and undertreated and associated with a high mortality rate. These findings indicate the potential for improvement in the management of patients with ARDS.

TIDAL VOLIUME PER PREDICTED BODY WEIGHT

PREDICTED BODY WEIGHT

MALE

Height (inches)	Height (cm)	PBW (kg)	Tidal Volume 6 ml	Tidal Volume 7 ml	Tidal Volume 8 ml	Tidal Volume 10 ml
48	122	22.4	134	157	179	224
49	124	24.7	148	173	198	247
50	127	27.0	162	189	216	270
51	130	29.3	176	205	234	293
52	132	31.6	190	221	253	316
53	135	33.9	203	237	271	339
54	137	36.2	217	253	290	362
55	140	38.5	231	270	308	385
56	142	40.8	245	286	326	408
57	145	43.1	259	302	345	431
58	147	45.4	272	318	363	454
59	150	47.7	286	334	382	477
60	152	50.0	300	350	400	500
61	155	52.3	314	366	418	523
62	157	54.6	328	382	437	546
63	160	56.9	341	398	455	569
64	163	59.2	355	414	474	592
65	165	61.5	369	431	492	615
66	168	63.8	383	447	510	638
67	170	66.1	397	463	529	661
68	173	68.4	410	479	547	684
69	175	70.7	424	495	566	707
70	178	73.0	438	511	584	730
71	180	75.3	452	527	602	753
72	183	77.6	466	543	621	776
73	185	79.9	479	559	639	799
74	188	82.2	493	575	658	822
75	191	84.5	507	592	676	845
76	193	86.8	521	608	694	868
77	196	89.1	535	624	713	891
78	198	91.4	548	640	731	914
79	201	93.7	562	656	750	937
80	203	96.0	576	672	768	960
81	206	98.3	590	688	786	983
82	208	100.6	604	704	805	1006
83	211	102.9	617	720	823	1029
84	213	105.2	631	736	842	1052

FEMALE

Height (inches)	Height (cm)	PBW (kg)	Tidal Volume 6 ml	Tidal Volume 7 ml	Tidal Volume 8 ml	Tidal Volume 10 ml
48	122	17.9	107	125	143	179
49	124	20.2	121	141	162	202
50	127	22.5	135	158	180	225
51	130	24.8	149	174	198	248
52	132	27.1	163	190	217	271
53	135	29.4	176	206	235	294
54	137	31.7	190	222	254	317
55	140	34.0	204	238	272	340
56	142	36.3	218	254	290	363
57	145	38.6	232	270	309	386
58	147	40.9	245	286	327	409
59	150	43.2	259	302	346	432
60	152	45.5	273	319	364	455
61	155	47.8	287	335	382	478
62	157	50.1	301	351	401	501
63	160	52.4	314	367	419	524
64	163	54.7	328	383	438	547
65	165	57.0	342	399	456	570
66	168	59.3	356	415	474	593
67	170	61.6	370	431	493	616
68	173	63.9	383	447	511	639
69	175	66.2	397	463	530	662
70	178	68.5	411	480	548	685
71	180	70.8	425	496	566	708
72	183	73.1	439	512	585	731
73	185	75.4	452	528	603	754
74	188	77.7	466	544	622	777
75	191	80.0	480	560	640	800
76	193	82.3	494	576	658	823
77	196	84.6	508	592	677	846
78	198	86.9	521	608	695	869
79	201	89.2	535	624	714	892
80	203	91.5	549	641	732	915
81	206	93.8	563	657	750	938
82	208	96.1	577	673	769	961
83	211	98.4	590	689	787	984
84	213	100.7	604	705	806	1007

Association Between Use of Lung-Protective Ventilation With Lower Tidal Volumes and Clinical Outcomes Among Patients Without Acute Respiratory Distress Syndrome

A Meta-analysis

Ary Serpa Neto, MD, MSc

JAMA. 2012;308(16):1651-1659

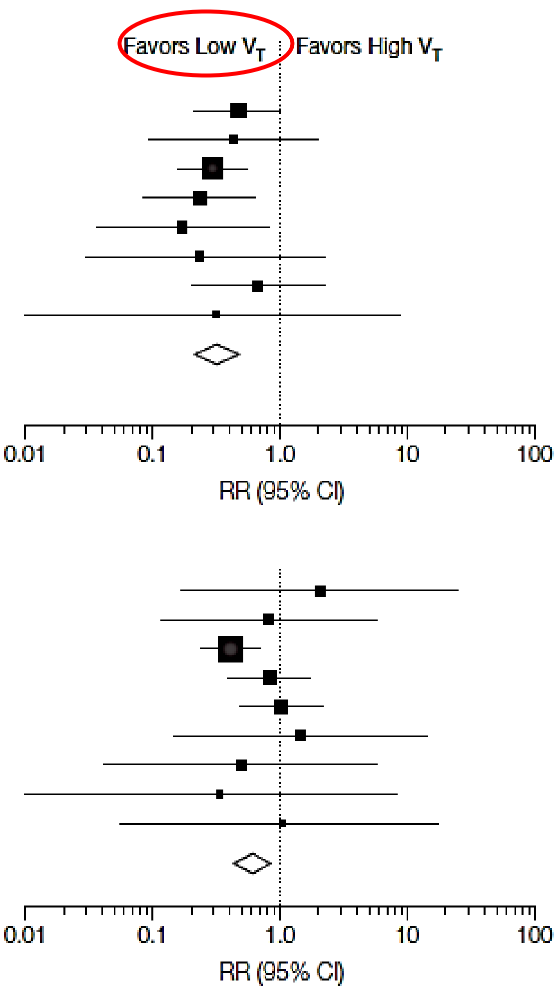
Figure 2. Effect of Ventilation With Smaller Tidal Volume in Patients With Healthy Lungs at the End of the Follow-up Period for Each Study

	High V_T, No.		Low V_T, No.			
	Events	Total	Events	Total	Weight, %	RR (95% CI)
Lung injury						
Gajic et al,[16] 2004	32	100	12	66	18.1	0.47 (0.22-1.00)
Michelet et al,[20] 2006	6	26	3	26	4.6	0.43 (0.10-1.97)
Yilmaz et al,[23] 2007	60	212	17	163	40.7	0.29 (0.16-0.53)
Licker et al,[26] 2009	20	533	5	558	17.7	0.23 (0.09-0.62)
Determann et al,[27] 2010	10	74	2	76	8.6	0.17 (0.04-0.82)
Yang et al,[31] 2011	4	50	1	50	3.4	0.23 (0.03-2.18)
Fernandez-Bustamante et al,[29] 2011	5	75	7	154	5.6	0.67 (0.20-2.17)
Weingarten et al,[32] 2012	1	20	0	20	1.3	0.32 (0.01-8.26)
Subtotal (95% CI)		1090		1113	100.0	0.33 (0.23-0.47)
Total events	138		47			

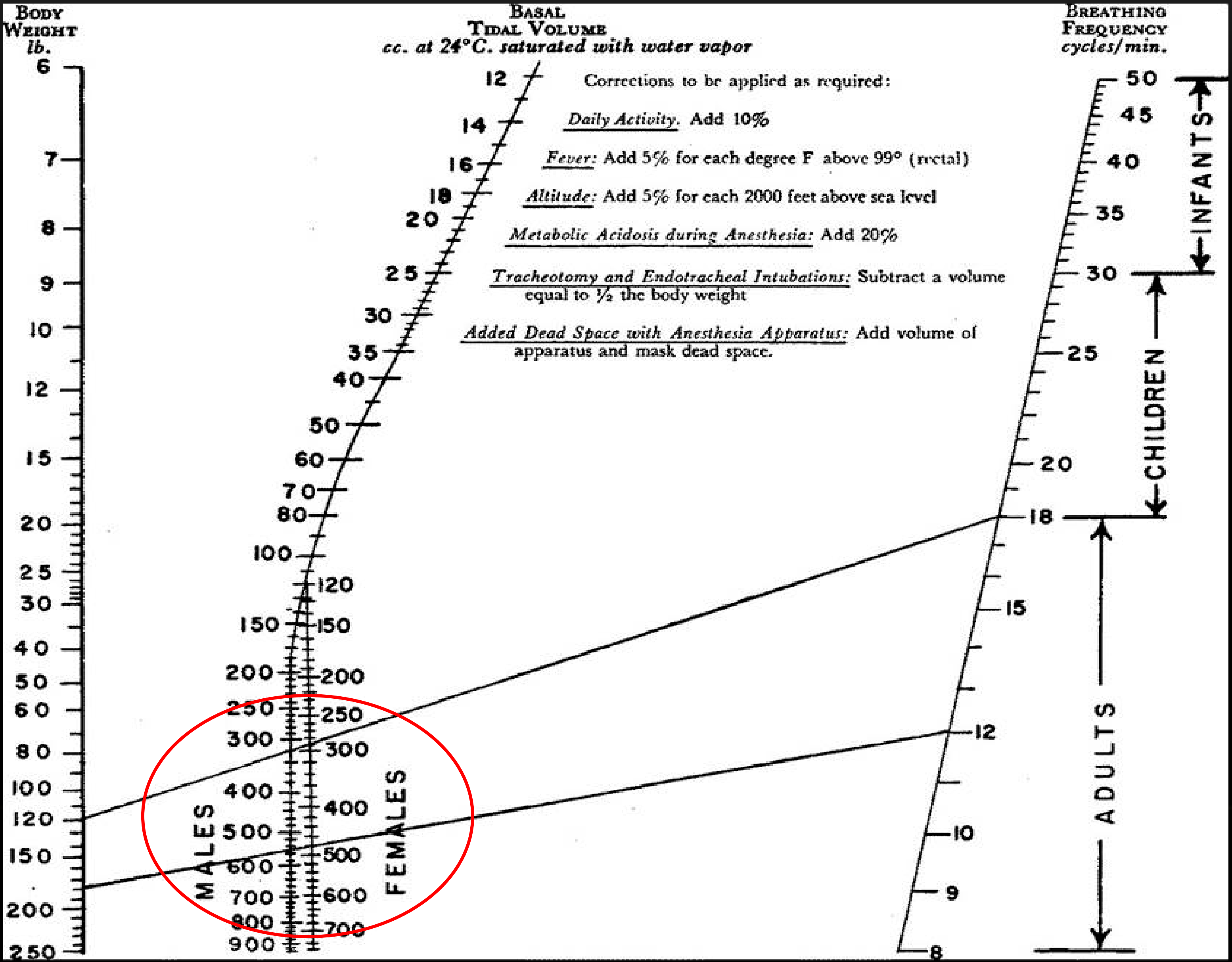
Heterogeneity: $\chi^2_7 = 3.74$; $P = .81$, $I^2 = 0\%$
Test for overall effect: $z = 6.06$; $P < .001$

	High V_T, No.		Low V_T, No.			
	Events	Total	Events	Total	Weight, %	RR (95% CI)
Mortality						
Michelet et al,[20] 2006	1	26	2	26	1.0	2.08 (0.18-24.51)
Wolthuis et al,[22] 2007	2	13	3	23	2.5	0.82 (0.12-5.71)
Yilmaz et al,[23] 2007	69	212	27	163	55.7	0.41 (0.25-0.68)
Licker et al,[26] 2009	15	533	13	558	16.7	0.82 (0.39-1.75)
Determann et al,[27] 2010	23	74	24	76	17.7	1.02 (0.51-2.04)
Fernandez-Bustamante et al,[29] 2011	1	75	3	154	1.5	1.47 (0.15-14.38)
Sundar et al,[30] 2011	2	74	1	75	2.2	0.49 (0.04-5.48)
Yang et al,[31] 2011	1	50	0	50	1.7	0.33 (0.01-8.21)
Weingarten et al,[32] 2012	1	20	1	20	1.1	1.00 (0.06-17.18)
Subtotal (95% CI)		1077		1145	100.0	0.64 (0.46-0.86)
Total events	115		74			

Heterogeneity: $\chi^2_8 = 6.94$; $P = .54$, $I^2 = 0\%$
Test for overall effect: $z = 2.68$; $P = .007$



RADFORD NOMOGRAM N ENGL J MED 1954; 251:877-884



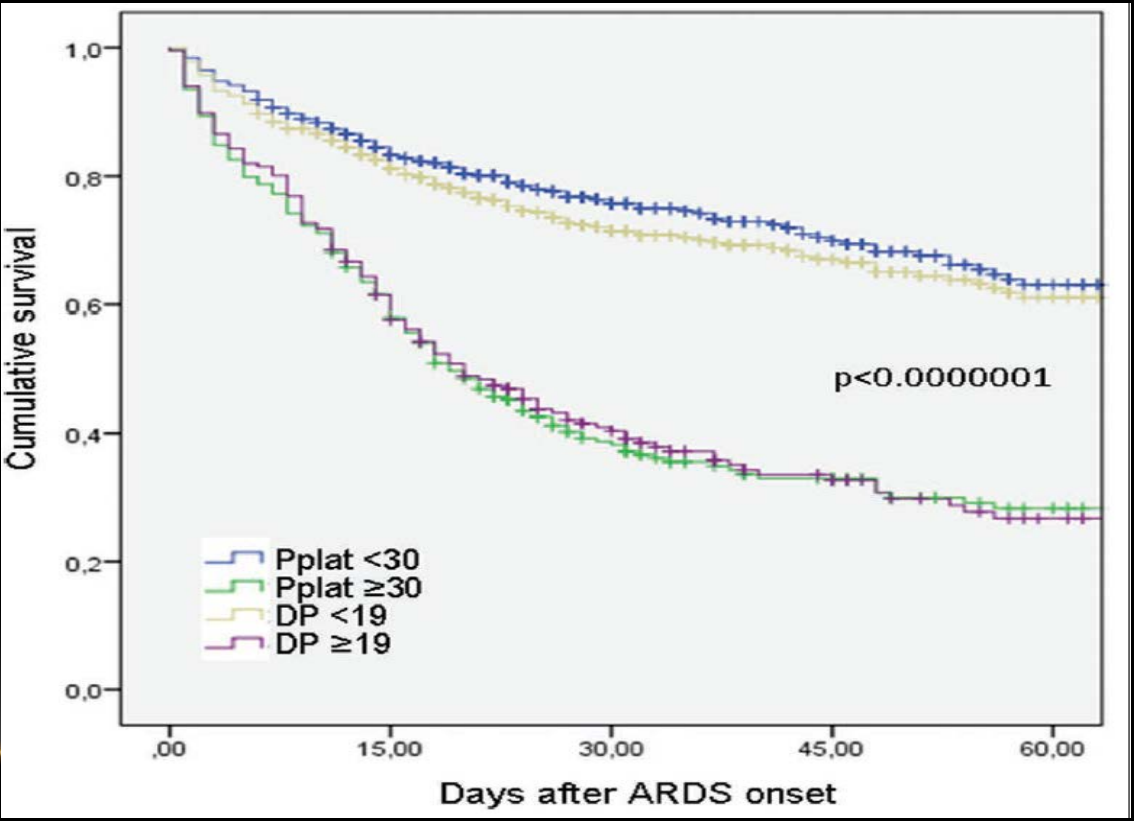
A Quantile Analysis of Plateau and Driving Pressures: Effects on Mortality in Patients With Acute Respiratory Distress Syndrome Receiving Lung-Protective Ventilation*

Jesús Villar, MD, PhD, FCCM[1]

Critical Care Medicine. 45(5):843-850, May 2017.

Spanish Initiative for Epidemiology, Stratification and Therapies of ARDS (SIESTA) Network

- Is driving pressure superior to its' defining variables in predicting outcome? (PEEP, Vt, Pplt)
- Retrospective analysis from 778 pts with moderate to severe ARDS
- Lung protective strategy
  - Vt 4-8ml/kg PBW, Pplt < 30cmH20, PEEP 10 -15cmH20 (Sp02> 90%), PaC02 35-50mmHg



Conclusion: Plateau pressure slightly better than driving pressure in predicting mortality

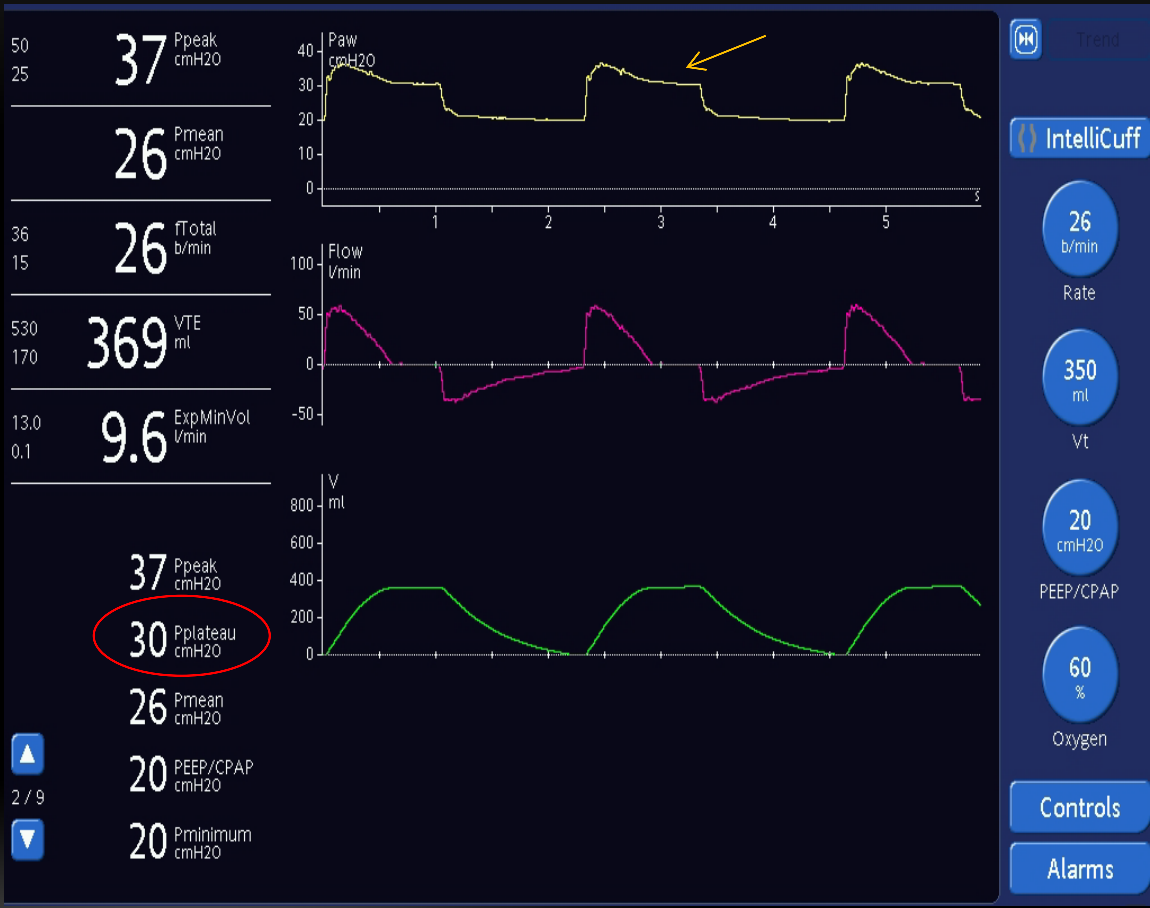
DRIVING PRESSURE

- Driving Pressure (DP) =Tidal volume/Compliance

  or

- Plateau pressure minus PEEP (total)

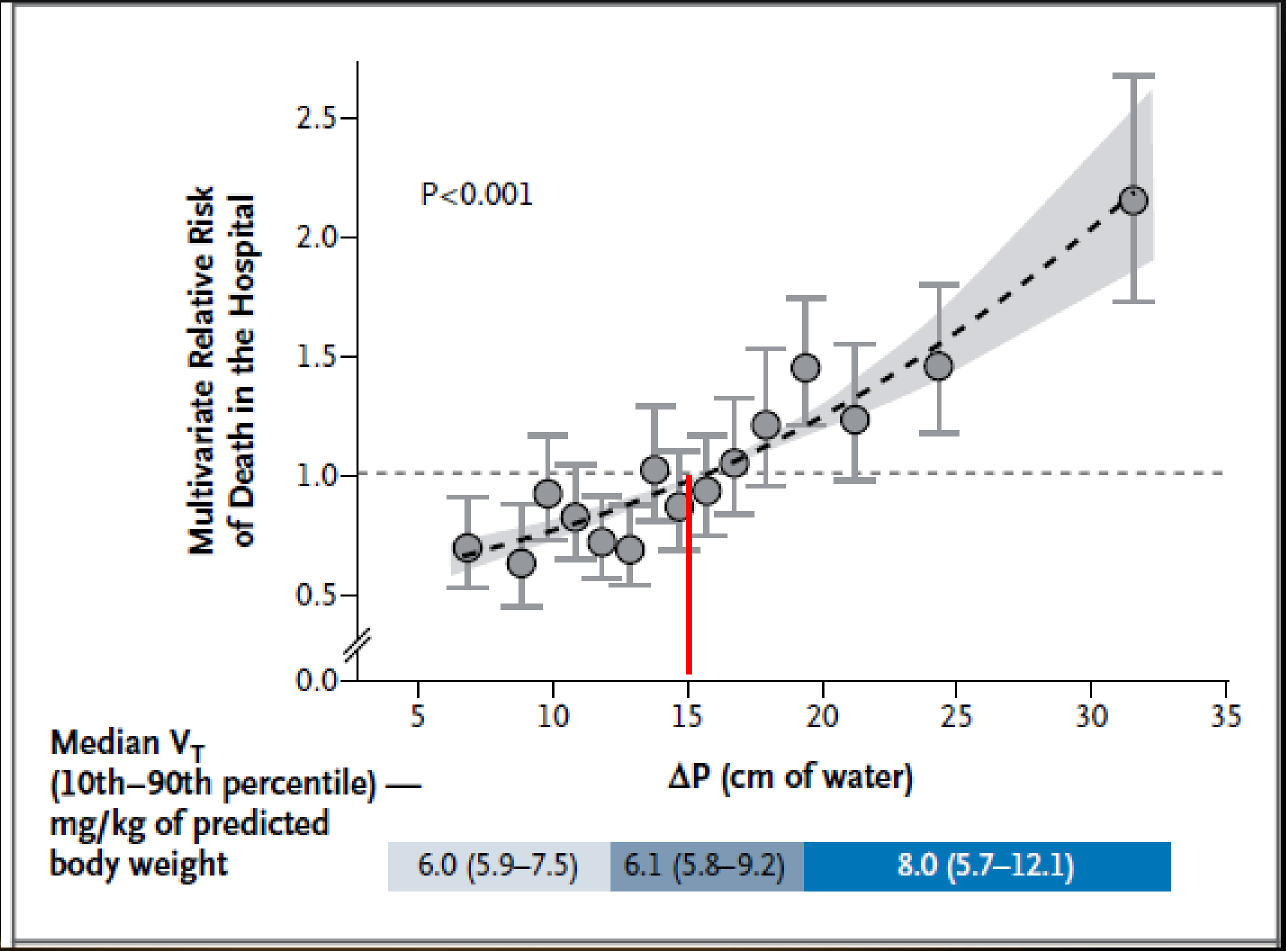
- i.e. Pplt 30cmH20 – 20 cmH20 PEEP = DP 10cmH20



Driving Pressure and Survival in the Acute Respiratory Distress Syndrome

N Engl J Med 2015;372:747-55.

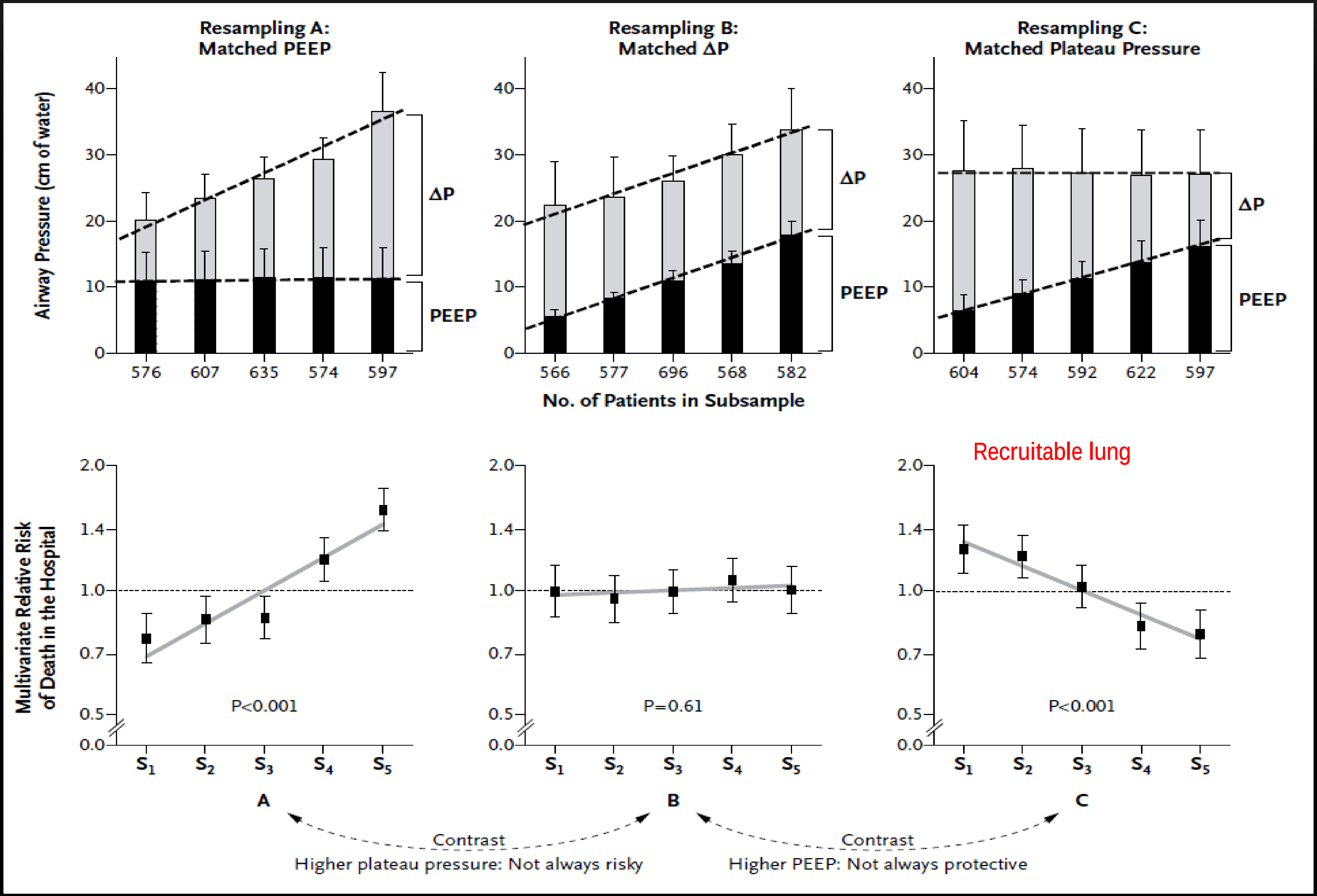
Driving Pressure =
Pplat – PEEP
or
Vt/ Compliance



Driving Pressure and Survival in the Acute Respiratory Distress Syndrome

Marcelo B.P. Amato

N Engl J Med 2015;372:747-55.



METHODS TO SELECT PEEP

- PEEP/Fi02 table
- Stress Index
- Best Compliance (Decremental)
- PV Curve
- Transpulmonary pressure

Goals: Oxygenation, reduce cyclic sheer injury, improve compliance, improve lung homogenaity (reduce stress raisers)

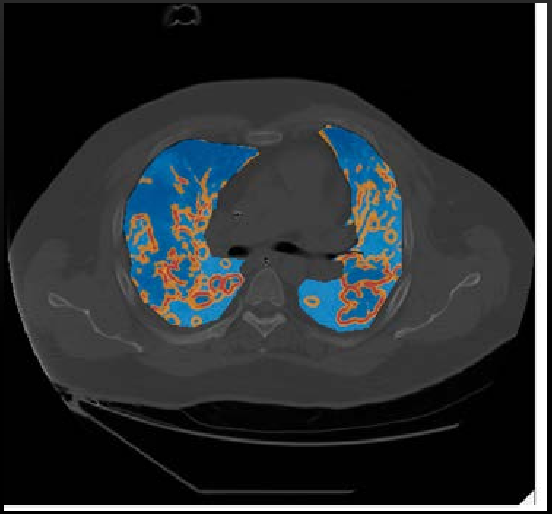
Key point: PEEP is related to FRC

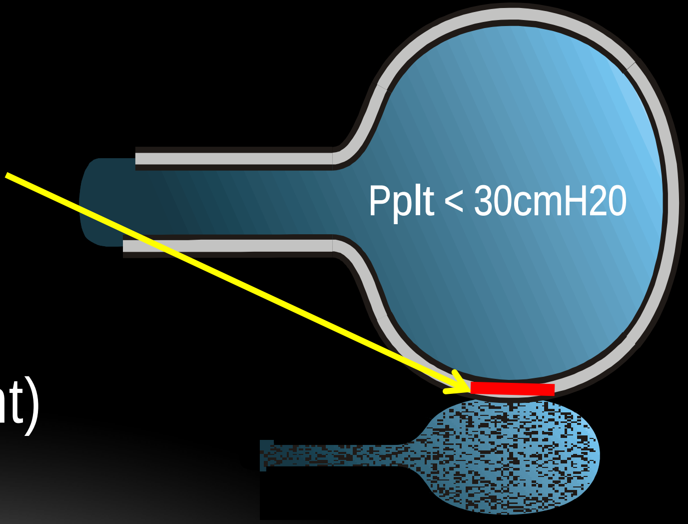
Lung recruitment is an inspiratory phenomenon.

STRESS RAISERS

- Lung Inhomogeneity/heterogeneity increases with ARDS severity
- Dynamic junction pressure increase
  - Multiplied by factors of 2:1 to 4.64:1

  Pplt 30cmH20 x 2 = 60 cmH20,
  Pplt 30 cmH20 x 4 = 120 cmH20!
- Injures normal alveoli
- Decreasing inhomogeneity reduces stress raisers (<u>Early</u> lung recruitment)





Lung Inhomogeneity in Patients with Acute Respiratory Distress Syndrome

Massimo Cressoni

Am J Respir Crit Care Med Vol 189, Iss 2, pp 149–158, Jan 15, 2014

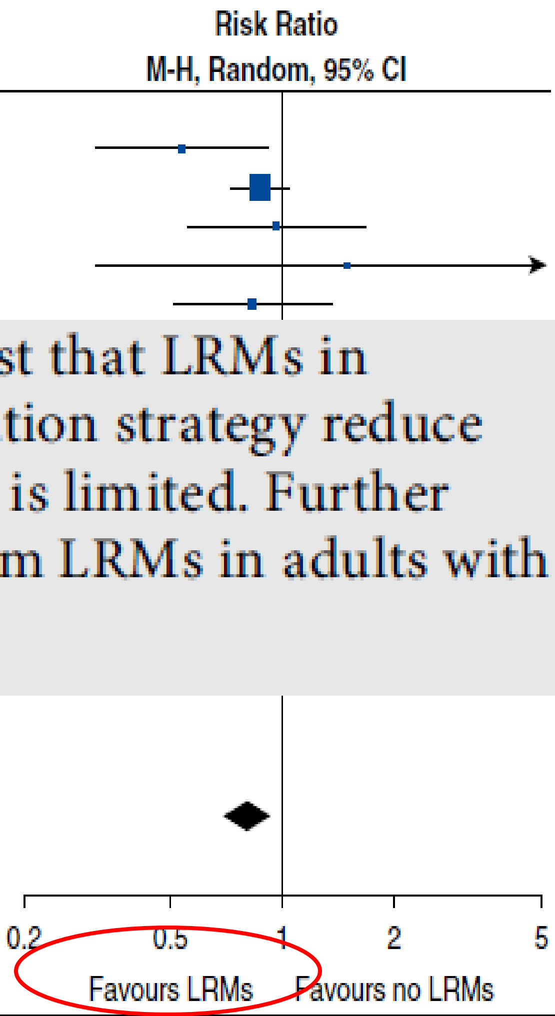
Lung Recruitment Maneuvers for Adult Patients with Acute Respiratory Distress Syndrome

A Systematic Review and Meta-Analysis

Ewan C. Goligher

Ann Am Thorac Soc Vol 14, Supplement 4, pp S304–S311, Oct 2017

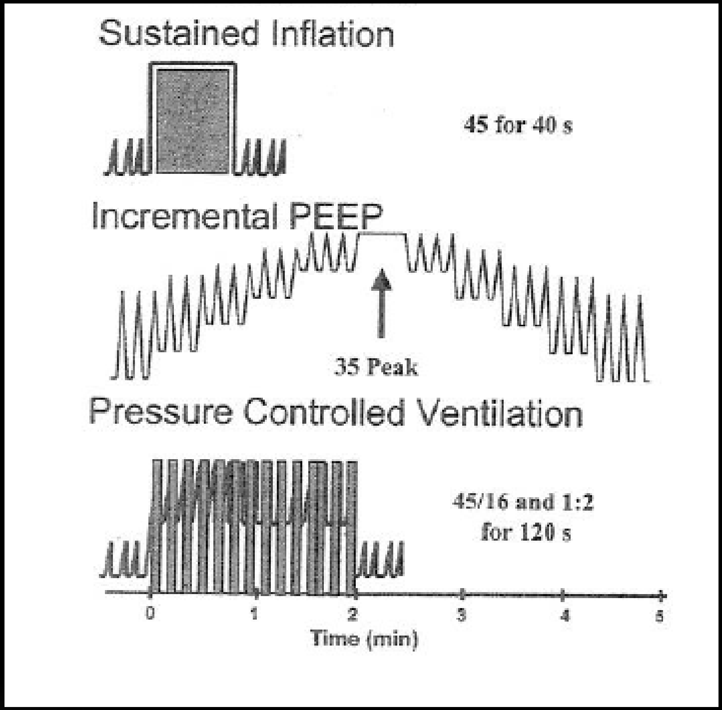
Study or Subgroup	LRMs Events	LRMs Total	No LRMs Events	No LRMs Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Co-intervention with higher PEEP							
Amato 1998	11	29	17	24	8.9%	0.54 [0.31, 0.91]	1998
Meade 2008	145	475	178	508	59.3%	0.87 [0.73, 1.04]	2008
Huh 2009	14	30	13	27	8.4%	0.97 [0.56, 1.68]	2009
Hodgson 2011	3	10	2	10	1.1%	1.50 [0.32, 7.14]	2011
Kacmarek 2016	22	99	27	101	10.4%	0.83 [0.51, 1.36]	2016
Subtotal (9							
Total events							
Heterogenei							
Test for ove							
No co-interv							
Xi 2010							
Subtotal (9							
Total events							
Heterogeneity: Not applicable							
Test for overall effect: Z = 2.06 (P = 0.04)							
Total (95% CI)		**698**		**725**	**100.0%**	**0.81 [0.69, 0.95]**	
Total events	213		266				
Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 5.30$, df = 5 (P = 0.38); $I^2 = 6\%$							
Test for overall effect: Z = 2.57 (P = 0.01)							
Test for subgroup differences: $Chi^2 = 1.58$, df = 1 (P = 0.21), $I^2 = 36.7\%$							



Conclusions: Randomized trials suggest that LRMs in combination with a higher PEEP ventilation strategy reduce mortality, but confidence in this finding is limited. Further trials are required to confirm benefit from LRMs in adults with ARDS.

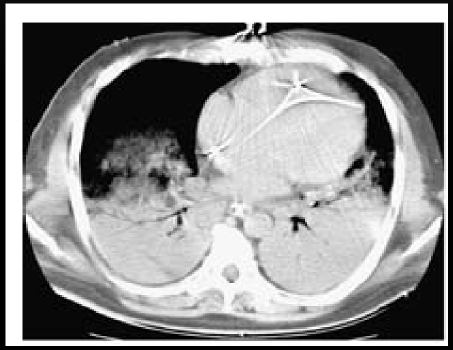
RECRUITMENT MANEUVERS

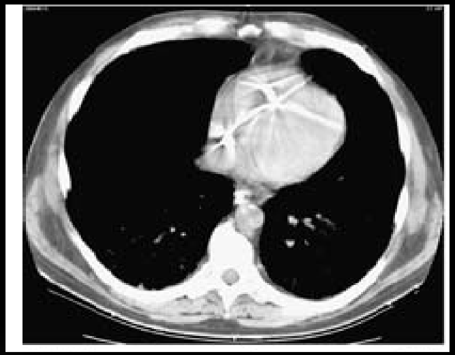
- Sustained inflation (insp hold)
  - 40-45 cmH20 for 10 - 40 sec
- Incremental/decremental PEEP
  - Best compliance
  - Sp02
- Pressure Control Modes
  - Inverse I:E ratio (APRV,Bilevel)
  - HFO
- Slow pressure volume maneuver
  - Super syringe



DETERMINANTS OF RM SUCCESS

- ARDS category
  - Intrapulmonary vs extrapulmonary
- ARDS Stage
  - Early vs. late
- Post maneuver PEEP
  - Maintain recruitment
  - Response duration
- Recruiting method





Effect of Lung Recruitment and Titrated Positive End-Expiratory Pressure (PEEP) vs Low PEEP on Mortality in Patients With Acute Respiratory Distress Syndrome
A Randomized Clinical Trial

Alveolar Recruitment for Acute Respiratory Distress Syndrome Trial (ART) Investigators
JAMA. 2017;318(14):1335-1345. doi:10.1001/jama.2017.14171

Methods

Control group: Vt 6ml/Kg PBW, Pplt 30cmH20

FIO2 %	30	40	40	50	50	60	70	70	70	80	90	90	90	100
PEEP cmH_2O	5	5	8	8	10	10	10	12	14	14	14	16	18	20–24

Intervention group:

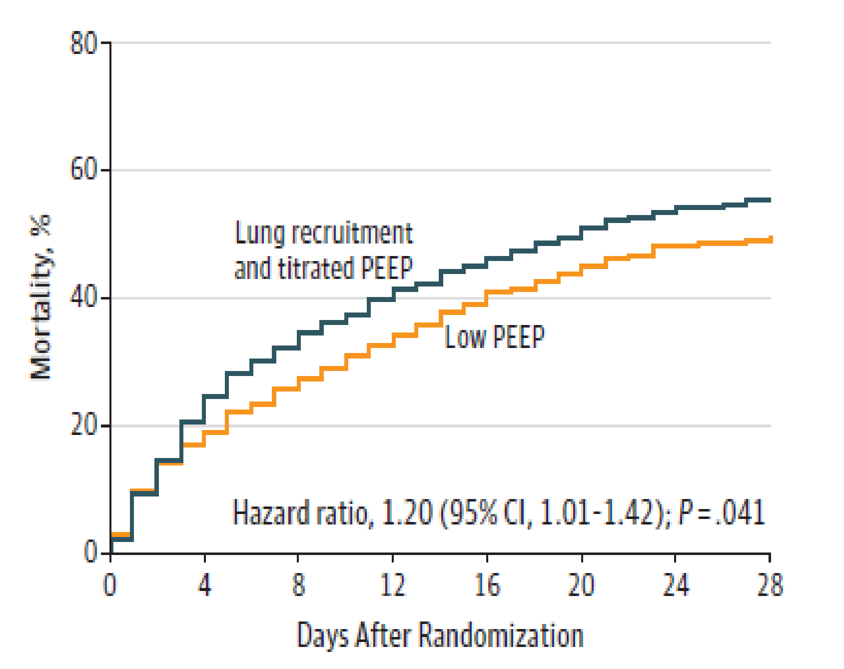
PC driving pressure 15cmH20 and incremental/decremental PEEP recruitment

Incremental PEEP 25 (1 min), 35 (1 min), 45 (2 min) = max Pplt 60cmH20 !

DSMB changed to PEEP 25, 30, 35 = max Pplt 50cmH20 due to SAEs (death, barotrauma, ↑ inotropes)

63% OLV Intrapulmonary ARDS

(56% Pneumonia)



CONCLUSIONS AND RELEVANCE In patients with moderate to severe ARDS, a strategy with lung recruitment and titrated PEEP compared with low PEEP increased 28-day all-cause mortality. These findings do not support the routine use of lung recruitment maneuver and PEEP titration in these patients.

Recruitment Maneuvers Modulate Epithelial and Endothelial Cell Response According to Acute Lung Injury Etiology*

(*Crit Care Med* 2013; 41:e256–e265)

- Animal study comparing effects of rate of increase in Paw and duration of RM on intrapulmonary vs. extrapulmonary ARDS
- RM Methods: 30cmH20/30 seconds, Stepwise of 5 cmH20, 8.5sec/step (step51), Stepwise of 5 cmH20 for 5sec/step with sustained 30cmH20/30sec (STEP 30/30)
- Measured biological markers of apoptosis, fibrogenesis, and cell damage

Results:

- Sustained RM 30cmH20/30 seconds resulted in worse endothelial injury.
- Both RM of 30/30 and stepwise 30/30 with sustained inflation had increased markers of endothelial injury in extra-pulmonary ARDS
- Stepwise RM Stepwise 5cmH20 for 8.5 seconds without sustained pressure resulted in least adverse biological impact
- Key point: Both rate and duration of RM matter: Slower rise and shorter duration least harmful.

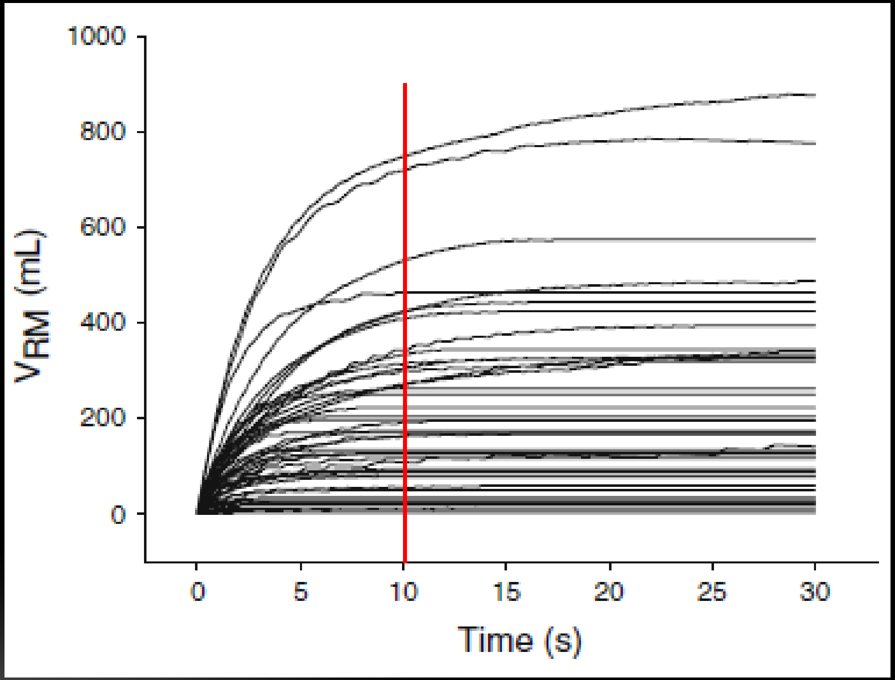
Optimal duration of a sustained inflation recruitment maneuver in ARDS patients

Jean-Michel Arnal Intensive Care Med (2011) 37:1588–1594

- 50 pts early onset ARDS
- Slow inflation (5cmH20/sec) sustained inflation of 40cmH20 for 30 seconds
- Measured HR, BP, Sp02 and recruited volume at 10 sec intervals

Results:

- 98% of volume recruited within first 10 seconds
- No significant decrease in BP at 10 seconds



BUT WHAT ABOUT THE SPONTANEOUS BREATHING PATIENT?

BJORN AAGE IBSEN, INTENSIVIST/ ANESTHESIST ESTABLISHED THE WORLDS FIRST ICU: COPENHAGEN 1953

"And actually it does not matter what is the source of the patient's gasping. You simply have to bring his breathing back in order."

Mechanical Ventilation–induced Diaphragm Atrophy Strongly Impacts Clinical Outcomes

Ewan C. Goligher Am J Respir Crit Care Med Vol 197, Iss 2, pp 204–213, Jan 15, 2018

Table 2. Clinical Outcomes in Relation to Changes in Diaphragm Thickness during Mechanical Ventilation

Outcome	Initial Change in Diaphragm Thickness during First Week of Ventilation Patients with ⩾2 Measurements (*n* = 191)			Statistical Comparisons Adjusted Count Ratio or Adjusted Odds Ratio (95% CI)*	
	⩾10% Decrease in Thickness (*n* = 78; 41%) Atrophy	<10% Change in Thickness (*n* = 66; 35%)	⩾10% Increase in Thickness (*n* = 47; 24%) Excess WOB	⩾10% Decrease in Thickness vs. <10% Change in Thickness	⩾10% Increase in Thickness vs. <10% Change in Thickness
Ventilator-free days to Day 60	46 (0–53)	51 (0–55)	37 (0–51)	0.77 (0.59–1.00)	0.91 (0.67–1.22)
Duration of ventilation (in ICU s	9 (5–17)†	5 (4–9)	10 (6–22)†	1.69 (1.28–2.24)	1.38 (1.00–1.90)
Duration o (in ICU s					31 (0.94–1.83)
Duration o (in hospi					23 (0.71–1.60)
Complicati respirato					84 (0.77–4.43)
Reintubati					24 (0.97–10.88)
Tracheost					11 (0.66–6.70)
Mechanica >14 d, n					16 (0.87–5.40)
Readmission to ICU during same hospital admission, *n* (%)	5 (7)	9 (15)	9 (20)	0.78 (0.21–2.84)	2.32 (0.70–7.67)
Death in ICU, *n* (%)	19 (24)	12 (18)	11 (23)	1.55 (0.61–3.95)	1.28 (0.45–3.65)
Death in hospital, *n* (%)	28 (37)	21 (3)	17 (37)	1.66 (0.73–3.76)	0.94 (0.38–2.34)

Conclusion: Development of diaphragm atrophy during the early course of mechanical ventilation predicts prolonged ventilation and an increased risk of complications. Similar but weaker results were found for increased diaphragm thickness.

Targeting an inspiratory effort level similar to that of healthy subjects at rest might accelerate liberation from ventilation.

Ventilator-Associated Lung Injury during Assisted Mechanical Ventilation

Felipe Saddy, MD, MSc

Semin Respir Crit Care Med 2014;35:409–417.

- Assisted ventilation may minimize VALI by:
  - Recruitment of dependent lung regions
  - More homogenous distribution of pleural pressure
- Assisted modes may exacerbate VALI:
  - Increased asynchrony
  - Allows inappropriate tidal volumes and transpulmonary pressures

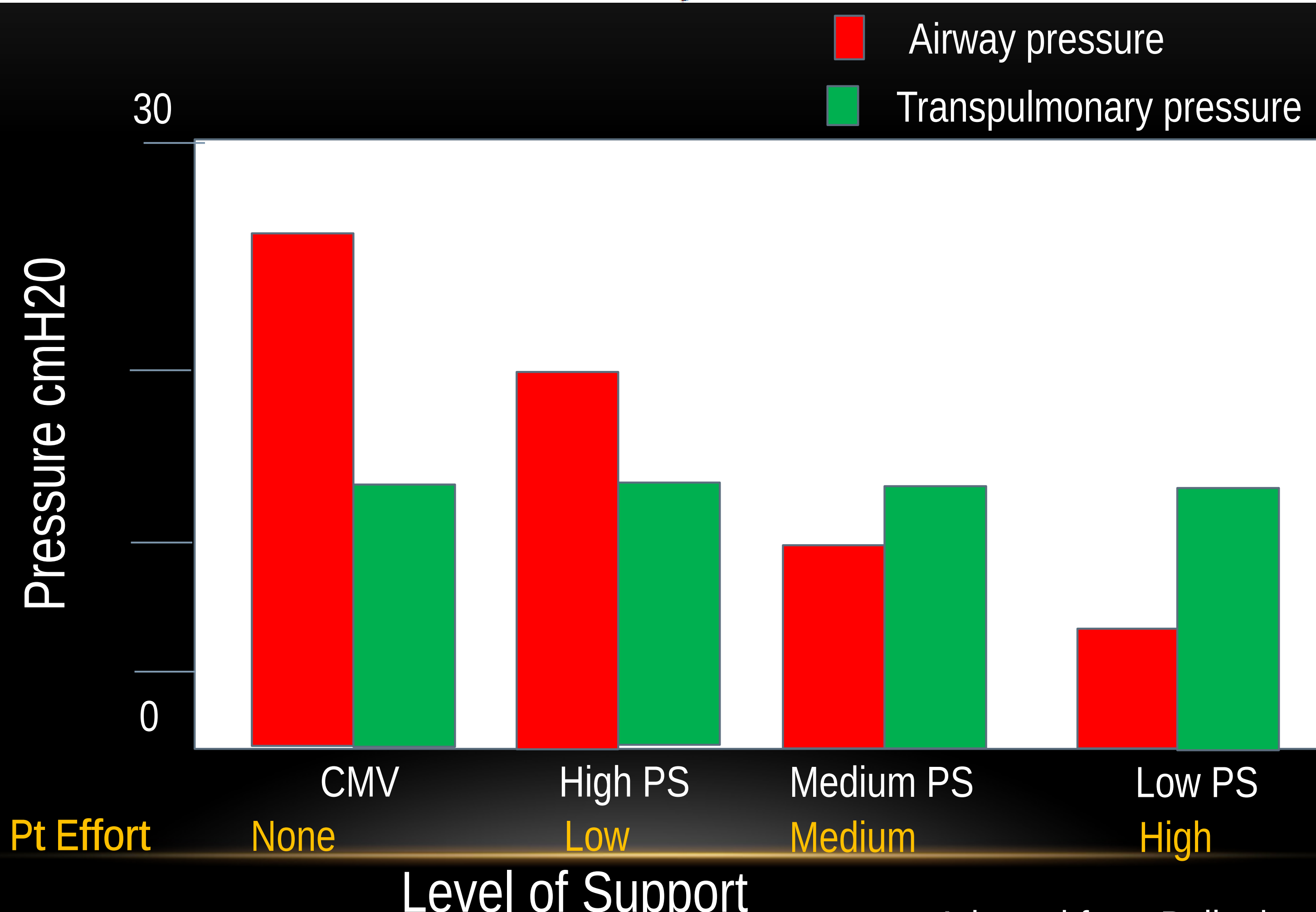
Conclusions:

- Assisted pressure limited time cycled modes that allow spontaneous ventilation may be used in patients with mild to moderate ARDS: P/F >150 (APRV, ACPC).
- The following parameters should be monitored:
  - Inspiratory drive (driving pressure)
  - Transpulmonary pressure
  - Tidal volume (6ml/kg PBW)

  Stress and Strain
- Assisted modes should not be used in severe ARDS.

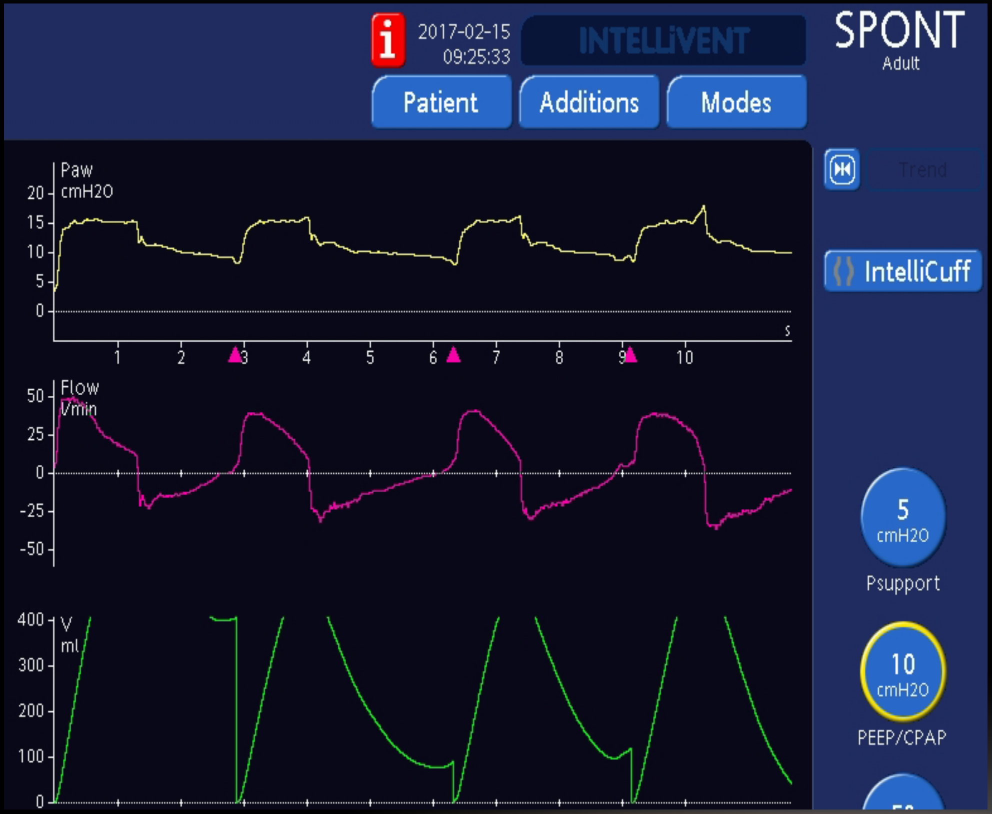
Do spontaneous and mechanical breathing have similar effects on average transpulmonary and alveolar pressure? A clinical crossover study

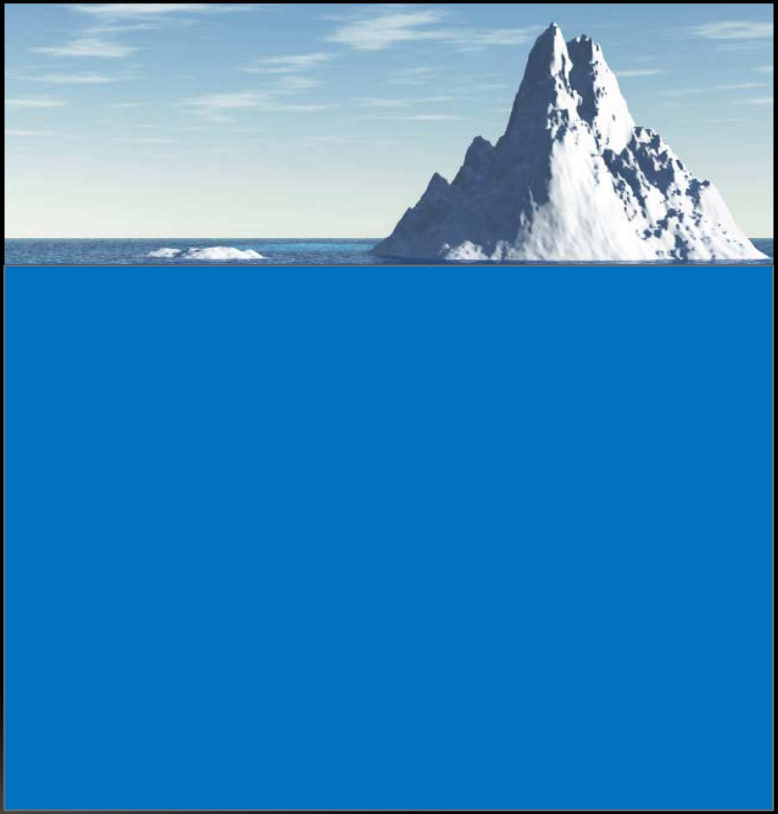
Bellani *et al. Critical Care* (2016) 20:142
DOI 10.1186/s13054-016-1290-9



Adapted from Bellani

VENTILATOR MONITORING
RESPIRATORY SYSTEM





DEFINING VENTILATOR PRESSURES

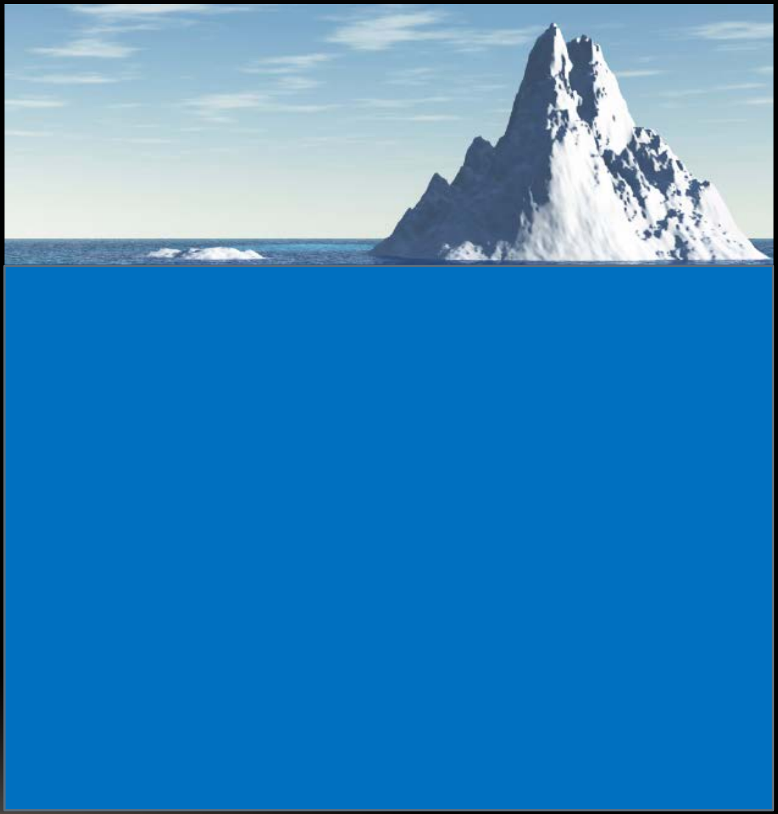
- Pressure
  - Peak Paw
    - Ventilator Circuit Pressures Resistance
    - Respiratory System Pressures Lung, chest wall, abdomen
    - Alveolar Distending Pressure
  - Plateau Pplt
    - Respiratory System Pressures Lung,chest wall, abdomen
    - Alveolar Distending Pressure
  - Transpulmonary Ptp
    - Alveolar Distending Pressure

The Application of Esophageal Pressure Measurement in Patients with Respiratory Failure

Am J Respir Crit Care Med Vol 189, Iss 5, pp 520–531, Mar 1, 2014

- Determination of "better PEEP"
- Tailor tidal volume to lung size
- Differentiate between chest wall and lung compliance
- Estimate true driving pressure
- Assess work of breathing
- Improve patient ventilator synchrony

VENTILATOR MONITORING
TRANSPULMONARY PRESSURES



BERLIN DEFINITION

	Acute Respiratory Distress Syndrome
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mm Hg < PaO_2/FIO_2 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H_2O[c]
Moderate	100 mm Hg < PaO_2/FIO_2 ≤ 200 mm Hg with PEEP ≥5 cm H_2O
Severe	PaO_2/FIO_2 ≤ 100 mm Hg with PEEP ≥5 cm H_2O

Abbreviations: CPAP, continuous positive airway pressure; FIO_2, fraction of inspired oxygen; PaO_2, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure.

[a] Chest radiograph or computed tomography scan.

[b] If altitude is higher than 1000 m, the correction factor should be calculated as follows: [PaO_2/FIO_2 × (barometric pressure/760)].

[c] This may be delivered noninvasively in the mild acute respiratory distress syndrome group.

Non-invasive Ventilation (NIV) of Patients with ARDS: Insights from the LUNG SAFE Study AJRCCM 2016 Oct

- 436/2813 (15.5%) of ARDS patients were initially managed with NIV regardless of severity
- NIV use independently associated with under recognition of ARDS 23.2%
- NIV Vt too large
  - 8.46 $\pm$ 2.77 ml/Kg vs. 7.53 $\pm$ 1.75 ml/kg invasive MV ($p << .001$)
- NIV PEEP inadequate
  - NIV 7cmH20 $\pm$ 2 vs. 8 cmH20 $\pm$ 3.1 invasive MV ($p < .001$)
- NIV failure occurred in 22.2% of mild, 42.3% moderate, 47.1% severe ARDS
- ICU mortality 10.6% NIV success vs. 42.7% NIV failure ($p < .001$)
- Hospital mortality 16.1% NIV success vs. 45.4 % NIV failure ($p < .001$)
- ICU Mortality P/F <150: NIV 36.2% vs. 24.7% Invasive MV ($p = 0.033$)

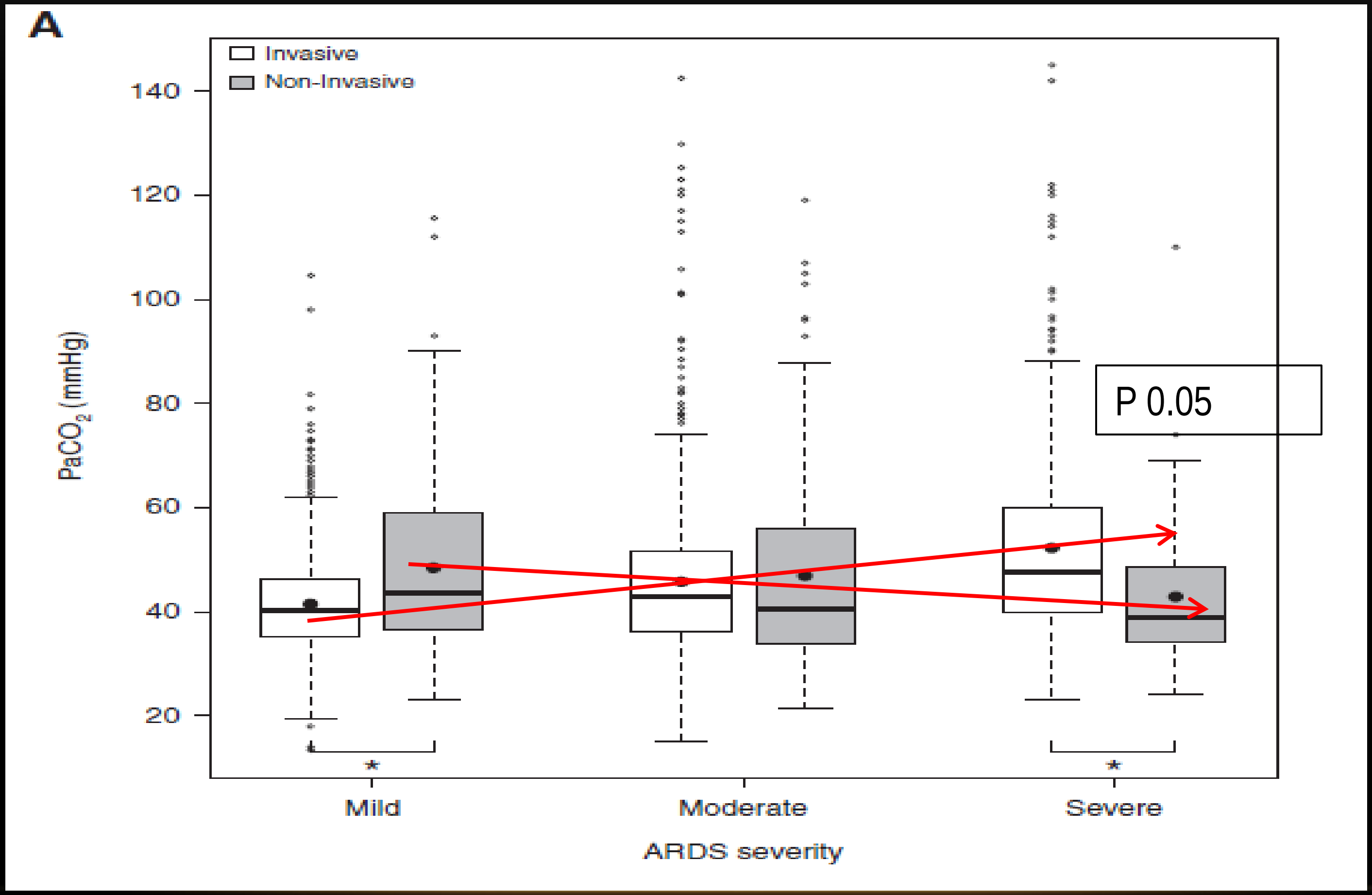
Conclusions: NIV was used in 15% of patients with ARDS, irrespective of severity category. NIV appears to be associated with higher ICU mortality in patients with a PaO_2/FiO_2 lower than 150 mmHg.

Noninvasive Ventilation of Patients with Acute Respiratory Distress Syndrome

Insights from the LUNG SAFE Study

Giacomo Bellani

Am J Respir Crit Care Med Vol 195, Iss 1, pp 67–77, Jan 1, 2017



NIV Pts had ↑ Vt, ↑ driving pressures

Timing of Intubation and Clinical Outcomes in Adults With Acute Respiratory Distress Syndrome*

Kirsten Neudoerffer Kangelaris
Crit Care Med. 2016 Jan;44(1):120-9

- Prospective observational study of 457 pts meeting ARDS criteria

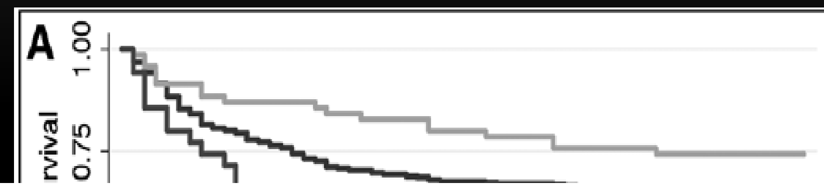


TABLE 3. Clinical Outcomes in Three Intubation Groups

	Early Intubation	Never Intubated	Late Intubation
n	351	70	36
Death at 60 d, *n* (%)	128 (36)	18 (26)	20 (56)[a,b]
Died in the hospital, *n* (%)	104 (30)	10 (14)[a]	18 (50)[a,b]
Ventilator-free days, median (IQR)	16 (0–23)	28 (23–28)[a]	7 (1–20)[b]
ICU days, median (IQR)[c]	9 (6–16)	4 (3–7)[a]	11.5 (9–17)[b]
Days of MV, median (IQR)[c]	6 (3–12)	0 (0–0)[a]	8 (4–15)[b]

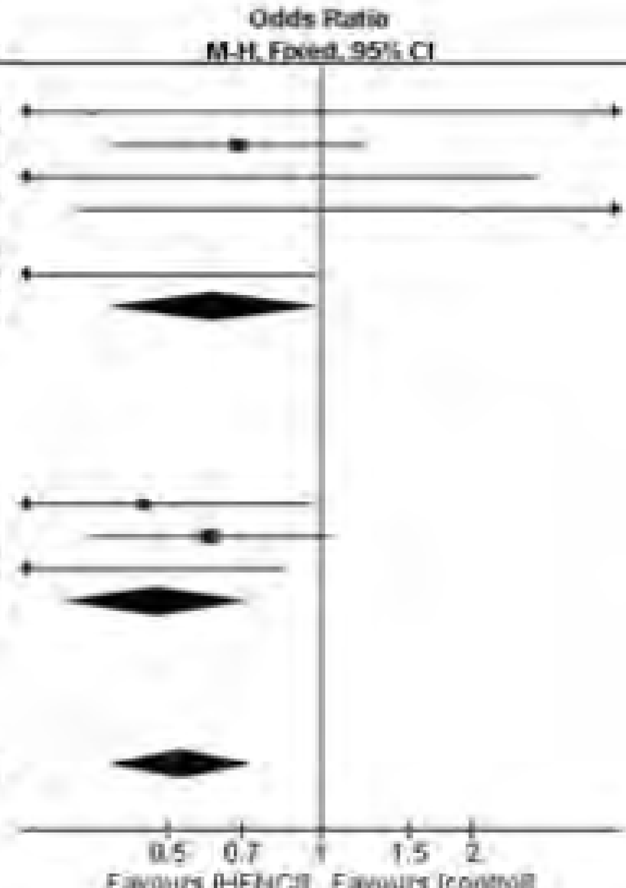
Conclusions: A substantial proportion of pts with ARDS were not intubated in their initial days of intensive care, and many were never intubated. Late intubation was associated with increased mortality. Criteria defining the ARDS prior to need for positive pressure ventilation are required so that these patients can studied and to facilitate early recognition and treatment of ARDS.

The Effect of High-Flow Nasal Cannula in Reducing Mortality and Rate of Endotracheal Intubation When Used Before Mechanical Ventilation Compared With Conventional Oxygen Therapy and Noninvasive Ventilation. A Systematic Review and Meta-Analysis.

AARC Sept 2017

Yuenan Ni, Jian Luo, Binmiao Liang, Zongan Liang; West China Hospital, Chengdu, China

Study or Subgroup	HFNC Events	HFNC Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
1.1.1 HFNC vs. COT						
Bell 2015	0	49	1	52	1.3%	0.35 [0.01, 8.90]
Frat 2015	40	106	44	94	27.4%	0.69 [0.39, 1.21]
Jones 2016	1	165	3	138	3.1%	0.27 [0.03, 2.67]
Lemiale 2015	4	52	2	48	1.9%	1.92 [0.33, 10.97]
Rittayamai 2015	0	20	0	20		Not estimable
Roca 2015	13	22	16	18	6.8%	0.18 [0.03, 0.99]
Subtotal (95% CI)		**413**		**370**	**40.5%**	**0.62 [0.38, 0.99]**
Total events	58		66			
Heterogeneity: $Chi^2 = 4.39$, df = 4 (P = 0.36); $I^2 = 9\%$						
Test for overall effect: Z = 1.98 (P = 0.05)						
1.1.2 HFNC vs. NIPPV						
Coudroy 2016	21	60	30	55	19.2%	0.45 [0.21, 0.95]
Frat 2015	40	106	55	110	31.8%	0.61 [0.35, 1.04]
Nagata 2015	0	33	10	43	8.5%	0.05 [0.00, 0.05]
Subtotal (95% CI)		**199**		**208**	**59.5%**	**0.48 [0.31, 0.73]**
Total events	61		95			
Heterogeneity: $Chi^2 = 3.25$, df = 2 (P = 0.20); $I^2 = 38\%$						
Test for overall effect: Z = 3.44 (P = 0.0006)						
Total (95% CI)		**612**		**578**	**100.0%**	**0.53 [0.39, 0.73]**
Total events	110		161			
Heterogeneity: $Chi^2 = 7.94$, df = 7 (P = 0.34); $I^2 = 12\%$						
Test for overall effect: Z = 3.90 (P < 0.0001)						
Test for subgroup differences: $Chi^2 = 0.63$, df = 1 (P = 0.43), $I^2 = 0\%$						



Conclusions: When used before MV, HFNC can improve the prognosis of patients compared both with the COT and NIPPV.

Hypoxemic Patients With Bilateral Infiltrates Treated With High-Flow Nasal Cannula Present a Similar Pattern of Biomarkers of Inflammation and Injury to Acute Respiratory Distress Syndrome Pa

853;

Comp
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simila MV.

This s
considered as mild ARDS patients and biomarker analysis may help identify patients who will fail on HFNC and will need to be intubated.

Failure of high-flow nasal cannula therapy may delay intubation and increase mortality

Byung Ju Kang

Intensive Care Med
DOI 10.1007/s00134-015-3693-5

Observational study of 175 pts who failed NHF

Classified as early intubation < 48 hrs vs. late intubation > 48 hrs

Results: ICU Mortality: early 39.2 vs. 66.7 % late (P 0.006)

Also significant: longer weaning, less vent free days, longer ICU LOS in late extubation group

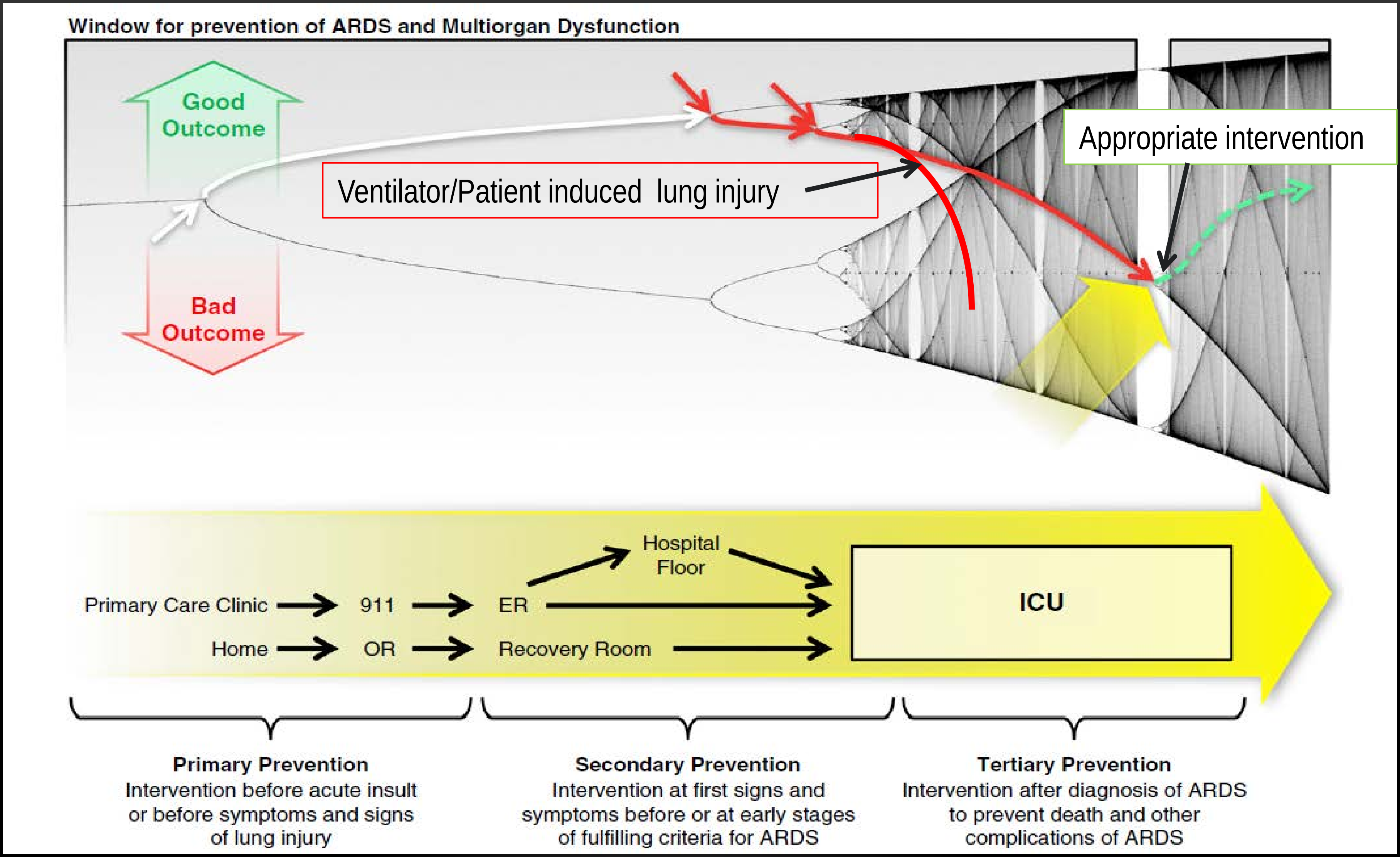
Conclusions: NHF may cause delayed intubation and worse clinical outcomes.

WHAT WE NEED TO DO AT THE BEDSIDE.

CASE STUDIES

WHAT'S THE MOST IMPORTANT FACTOR IN ENSURING SAFE VENTILATORY SUPPORT?

TIMING IS EVERYTHING



The golden hours of critical care resuscitation. Delays of even a few hours in the treatment of critical care syndromes can result in irreversibly large physiologic changes that become increasingly difficult to overcome.

FIFTY YEARS OF RESEARCH IN ARDS Am J Respir Crit Care Med Vol 195, Iss 6, pp 725–736, Mar 15, 2017

Is Acute Respiratory Distress Syndrome a Preventable Disease?

NIV/NHF IN RESPIRATORY FAILURE

1. Careful patient selection (hypercapnic vs. hypoxemic)
2. NIV settings to target
   a) IPAP: tidal volume (Vt) 6-8 ml/kg PBW
   b) EPAP: Sp02 88-95% with lowest Fi02
3. NHF setting: Highest flow lowest Fi02
4. <u>Recognize ARDS !</u>
5. <u>Closely monitor for 1- 4 hours!</u>
   a) Work of breathing:
      a) Accessory muscles
      b) Respiratory rate
   b) High likelihood of failure:
      a) P/F $\leq$ 175 after 1 hr
      b) Vt > 9.5 ml/kg PBW

Role of Noninvasive Ventilation in
Acute Lung Injury/Acute Respiratory Distress Syndrome:
A Proportion Meta-analysis

Respir Care 2010;55(12):1653–1660.

WHAT MODE SHOULD I USE?

There are currently 200+ Modes

Ventilator Mode Translator v7.2

Standard Modes on

ICU VENTILATORS

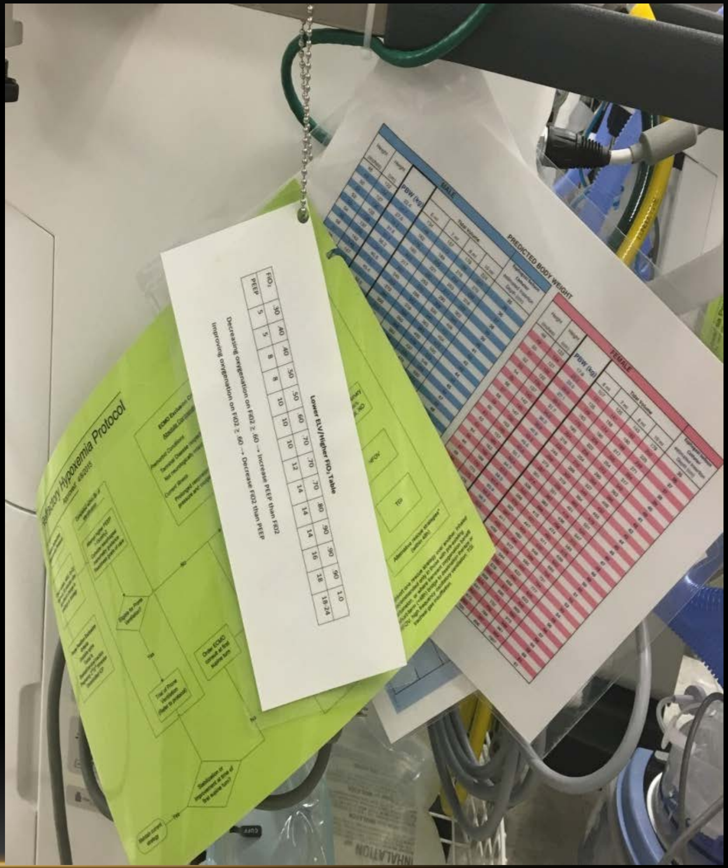
Manufacturer	Model	
CareFusion	Avea	Volume A/C
CareFusion	Avea	Volume SIMV
CareFusion	Avea	Pressure A/C
CareFusion	Avea	Pressure SIMV
CareFusion	Avea	CPAP/Pressure Support
Covidien	Newport e360T	Volume Control A/C Man
Covidien	Newport e360T	Volme Control SIMV
Covidien	Newport e360T	Pressure Control A/C Ma
Covidien	Newport e360T	Pressure Control SIMV
Covidien	Newport e360T	Pressure Control Spont
Covidien	Newport e360T	Volume Control Spont
Dräger	Babylog VN500	Pressure Control A/C
Dräger	Babylog VN500	Pressure Control Continu
Dräger	Babylog VN500	Pressure Control SIMV
Dräger	Babylog VN500	Pressure Control Pressu
Dräger	Babylog VN500	Spontaneous CPAP/Pres
Dräger	Evita Infinity V500	Volume Control Continuo
Dräger	Evita Infinity V500	Volume Control Assist C
Dräger	Evita Infinity V500	Volume Control Synchro
Dräger	Evita Infinity V500	Pressure Control Assist
Dräger	Evita Infinity V500	Pressure Control Continu
Dräger	Evita Infinity V500	Pressure Control Synchr
Dräger	Evita Infinity V500	Pressure Control Airway
Dräger	Evita Infinity V500	Pressure Control Pressu
Dräger	Evita Infinity V500	Spontaneous CPAP/Pres
Dräger	Evita XL	Continuous Mandatory V
Dräger	Evita XL	SIMV
Dräger	Evita XL	Pressure Controlled Ven
Dräger	Evita XL	Pressure Controlled Ven
Dräger	Evita XL	Continuous Positive Airw
GE Healthcare	Engström Carestation	Volume Controlled Ventil
GE Healthcare	Engström Carestation	Synchronized Intermitten
GE Healthcare	Engström Carestation	Pressure Controlled Ven
GE Healthcare	Engström Carestation	Synchronized Intermitten
GE Healthcare	Engström Carestation	Bilevel Airway Pressure
GE Healthcare	Engström Carestation	Constant Positive Airway
Hamilton	C3	Pressure Controlled Ven
Hamilton	C3	Pressure Controlled Syn
Hamilton	C3	Spontaneous
Hamilton	G5	Synchronized Controlled
Hamilton	G5	SIMV
Hamilton	G5	Pressure Controlled Con
Hamilton	G5	Pressure SIMV
Hamilton	G5	Spontaneous
Hamilton	Galileo	Synchronized Controlled
Hamilton	Galileo	SIMV
Hamilton	Galileo	Pressure Controlled Con
Hamilton	Galileo	Pressure SIMV
Hamilton	Galileo	Spontaneous
Maquet	Servo i	Pressure Control
Maquet	Servo i	SIMV (Pressure Control)
Maquet	Servo i	Automode (Pressure Co
Maquet	Servo i	Spontaneous/CPAP
Maquet	Servo i	Pressure Support
Philips Respironics	V200	Volume Control Ventilatio
Philips Respironics	V200	Volume Control Intermitte
Philips Respironics	V200	Pressure Control Intermi
Philips Respironics	V200	Continous Positive Airw
Philips Respironics	V60	Spontaneous/Timed
Philips Respironics	V60	CPAP

lode Classification

Family Breath equence	Genus Primary Breath Targeting Scheme	Species Secondary Breath Targeting Scheme	Tag
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
IMV	set-point	set-point	VC-IMVs,s
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CMV	set-point	N/A	VC-CMVs
CMV	set-point	N/A	VC-CMVs
CMV	set-point	N/A	PC-CMVs
IMV	set-point	set-point	VC-IMVs,s
IMV	set-point	set-point	PC-IMVs,s
IMV	set-point	set-point	PC-IMVs,s
IMV	set-point	set-point	PC-IMVs,s
CSV	set-point	N/A	PC-CSVs
CSV	set-point	N/A	PC-CSVs

CURRENT VENTILATION GUIDELINES LUNG PROTECTION FOR ALL PATIENTS

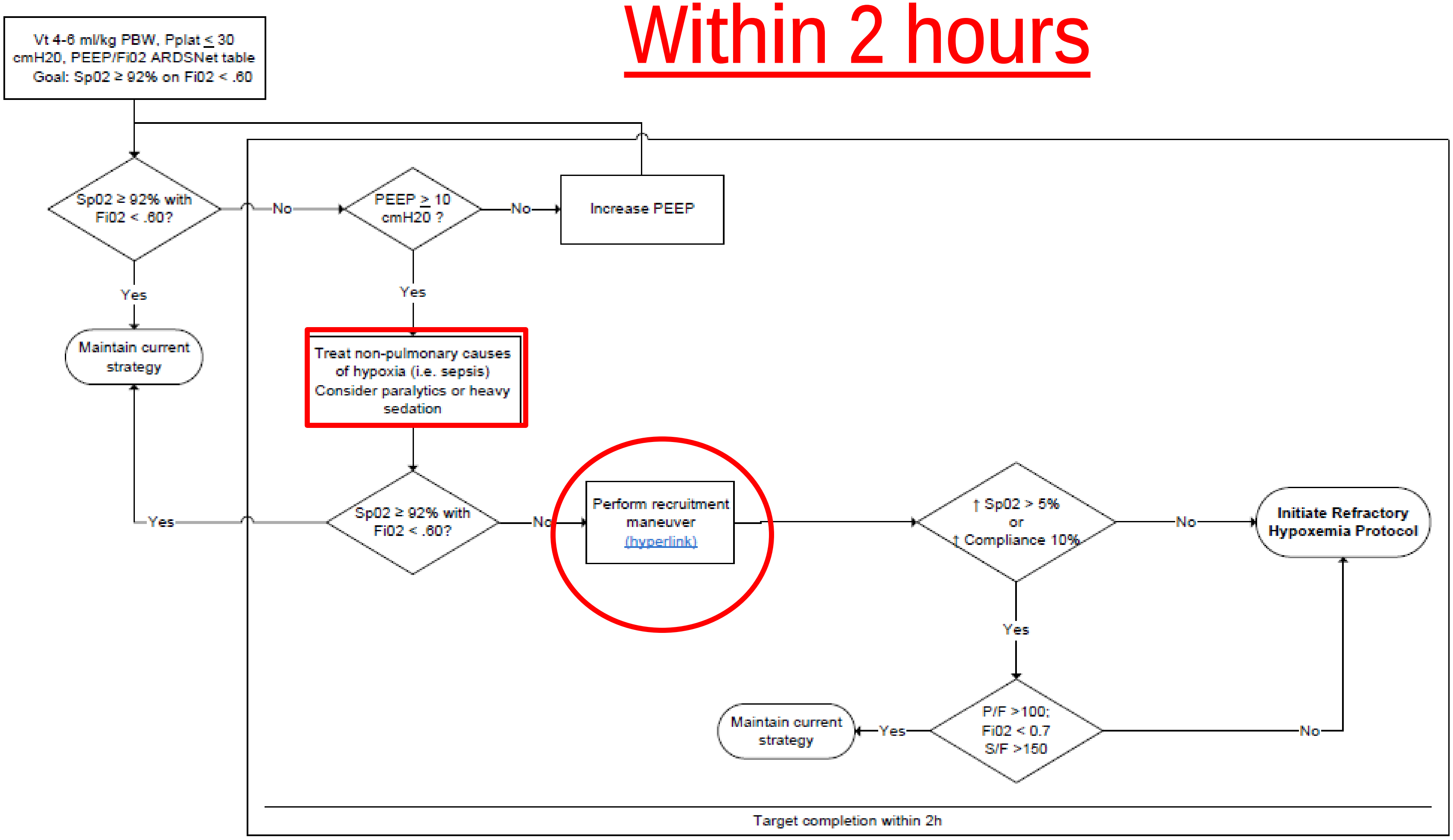
- Maintain Sp02 target (92-96%), Fi02 < .60
- Target tidal volume 6 (4-8) ml/Kg PBW (all pts unless MD directed)
- Plateau pressure (Pplt) < 30 cmH20
- Respiratory rate PaC02 35-45 mmHg (COPD exception)
- PEEP/Fi02 table
- Driving pressure < 15 cmH20



Refractory Hypoxemia Identification

Approved: 4/8/2015

Within 2 hours



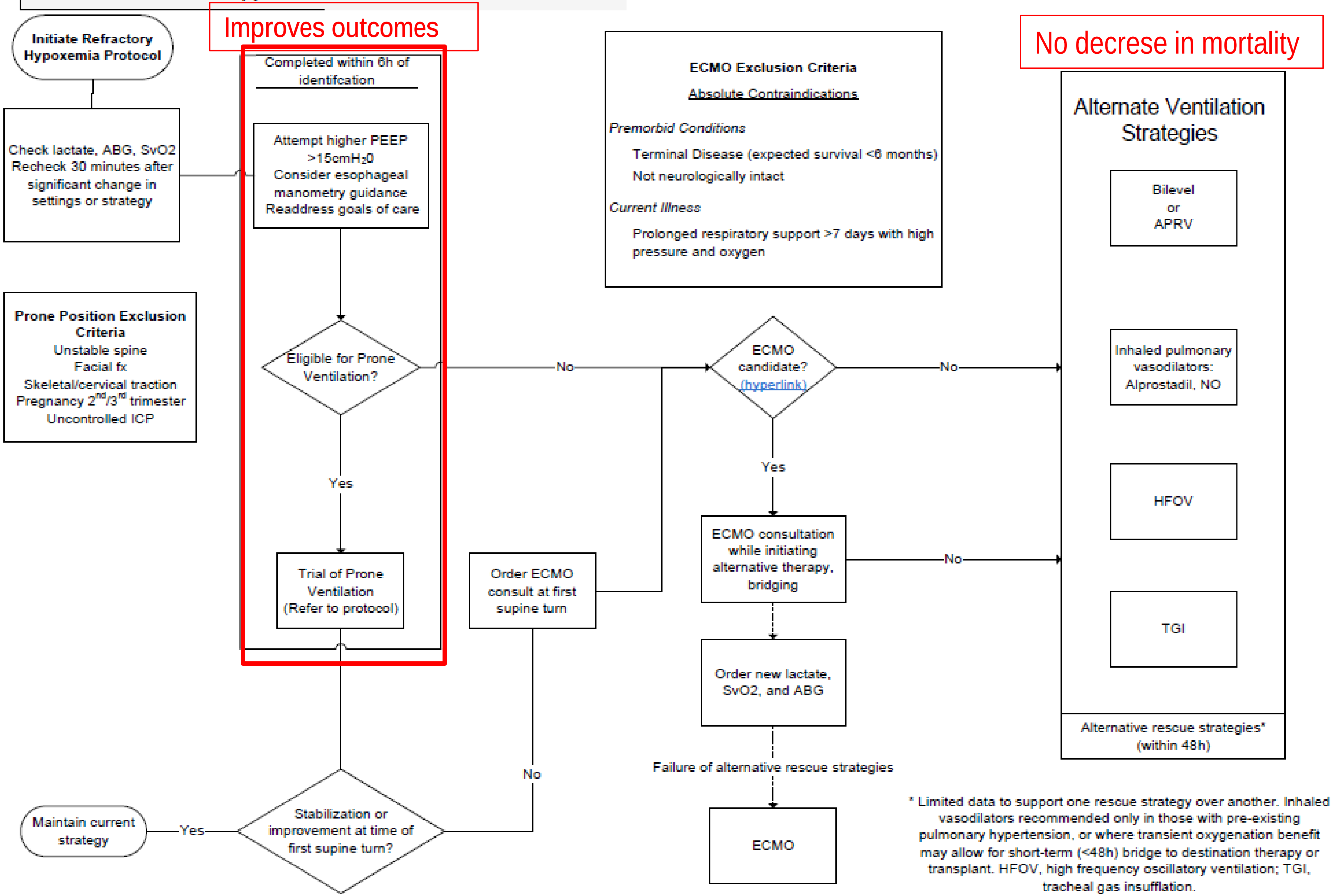
PV CURVE AND RECRUITMENT

- Lower inflection point (LIP)
  - Alveolar opening
- Point of derecruitment (PDR)
  - Alveolar derecruitment
- Upper inflection point (UIP)
  - Lung over distention
- Recruitable volume

Refractory Hypoxemia Protocol

Approved: 4/8/2015

Within 6 hours



TRANSPULMONARY PRESSURE MONITORING

- Respiratory system pressures
  - Peak, plateau <30, PEEP
  - Driving pressure (DP) < 15 cmH20 (respiratory system)
- Esophageal pressure
  - (pleural pressure)
- Transpulmonary pressures
  - Inspiratory <20
  - Expiratory 0-5
  - "True" DP trans <15 (10-12)

PRONE POSITION

- New (old) protocol 2017
  - No Rotoprone initially
- 16 consecutive hours per day (at least)
- Discontinue when:
  - Resolution of underlying processes
  - P/F >150 mmHg
  - PEEP $\leq$ 10cmH20
  - Fi02 $\leq$ .60

CASE 1 OPTIMIZING VENTILATOR SETTINGS

- 62yo male with multiple comorbidities
- bilateral pneumonia
- worsening hypoxia
- Pa02 63, PaC02 53, pH 7.41

CASE 1 PEEP TITRATION

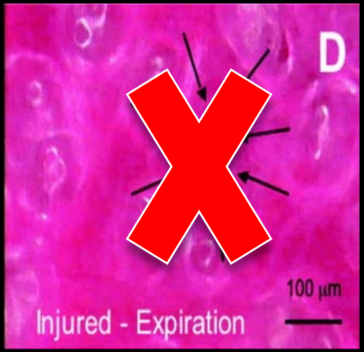
- End expiratory transpulmonary pressure (PtransE) – 3.5 cmH20
- Driving pressure 15-16 cmH20 (resp system)
- Intervention: Increase PEEP

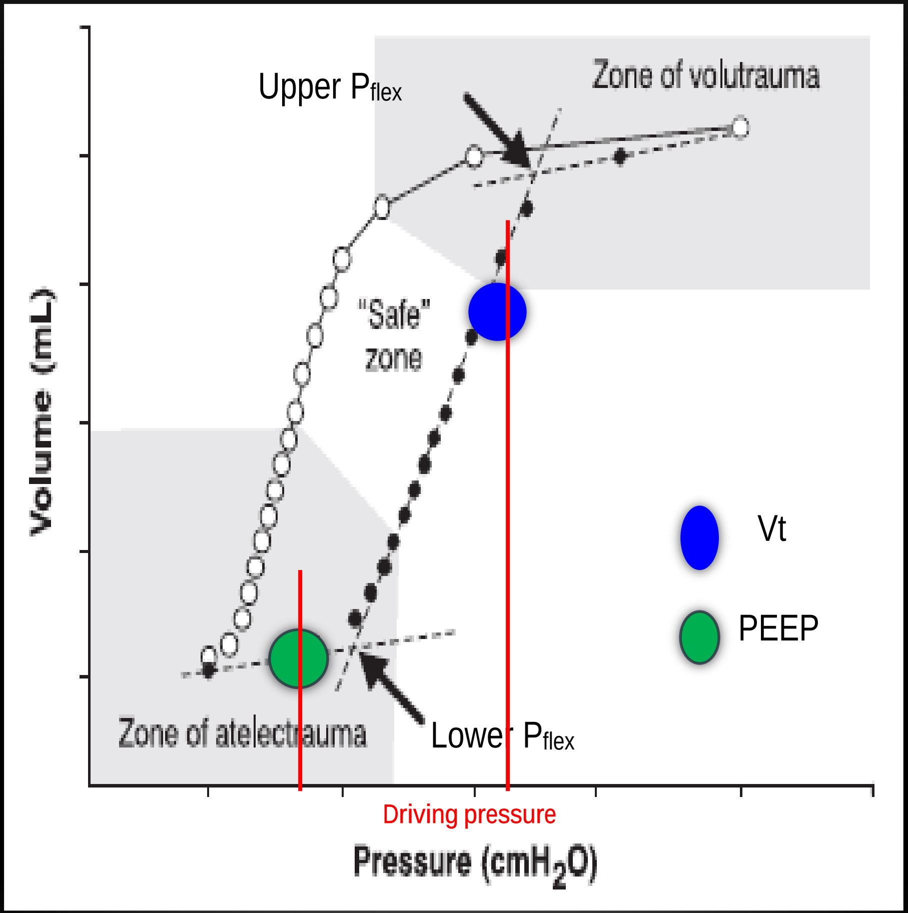
CASE 1 DRIVING PRESSURE

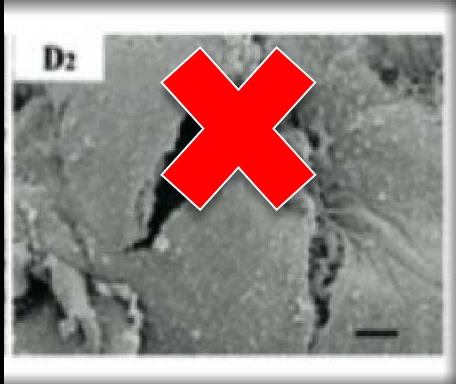
- High Plateau pressure (Pplt) 40cmH20
- Driving pressure airway (DPaw) 24cmH20
- Driving pressure Transpulmonary (DPtp) 24cmH20

Why is driving pressure increasing so much?

DRIVING PRESSURE







↑ PEEP ↑ FRC
"Baby lung"

CASE 1 WHAT IS SAFE TIDAL VOLUME

- Vt decreased to 380ml (5ml/kg)
- Ptrans E 0-2 cmH20
- Ptrans I < 20 cmH20
- Driving pressure:

  < 15cmH20

Fi02 >.60, marginal Sp02, Now what?

Refractory Hypoxemia Protocol

Approved: 4/8/2015

Initiate Refractory Hypoxemia Protocol

Check lactate, ABG, SvO2
Recheck 30 minutes after significant change in settings or strategy

Completed within 6h of identifcation

Attempt higher PEEP >15cm$H_2$0
Consider esophageal manometry guidance
Readdress goals of care

Eligible for Prone Ventilation?

No

Yes

Trial of Prone Ventilation (Refer to protocol)

Order ECMO consult at first supine turn

No

Stabilization or improvement at time of first supine turn?

Yes

Maintain current strategy

Prone Position Exclusion Criteria
Unstable spine
Facial fx
Skeletal/cervical traction
Pregnancy 2nd/3rd trimester
Uncontrolled ICP

ECMO Exclusion Criteria
Absolute Contraindications

Premo
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Alternate Ventilation
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ors:
, NO

strategies*
8h)

ECMO

* Limited data to support one rescue strategy over another. Inhaled vasodilators recommended only in those with pre-existing pulmonary hypertension, or where transient oxygenation benefit may allow for short-term (<48h) bridge to destination therapy or transplant. HFOV, high frequency oscillatory ventilation; TGI, tracheal gas insufflation.

CASE 1 PRONE POSITION

- Less cardiac oscillations
- Significant improvement in respiratory mechanics
- DPtp 11cmH20
- Fi02 .50, Pa02 > 153mmHg
- Pt proned for 2 days (16 hrs)
- Returned supine and successfully weaned 3 days later.

CASE 2 RH PROTOCOL RESCUE

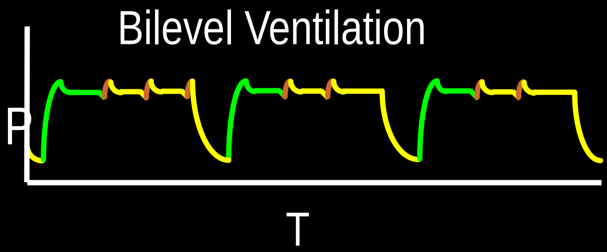
37 yo from OSH at 03:30 severe ARDS

Sp02 during transport low 70s

Rescue therapies initiated:

- Fi02 .80 Bilevel (APRV) 30/15 cmH20
- Nitric oxide 20 PPM
- HFOV on standby
- ECMO consult

07:00 Sp02 low 90s

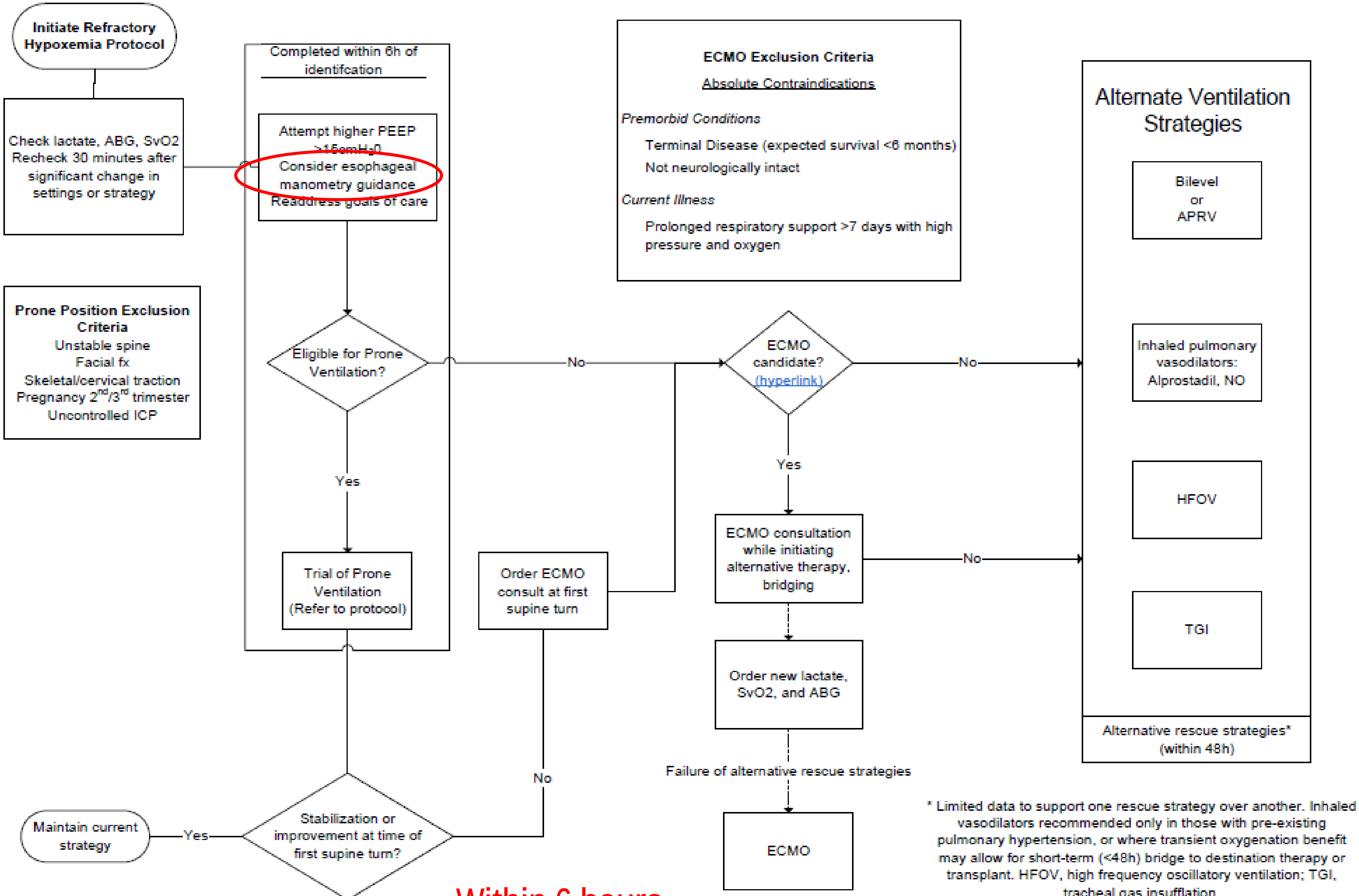


Nitric Oxide

HFOV

Refractory Hypoxemia Protocol

Approved: 4/8/2015



Within 6 hours

RECRUITMENT MANEUVER

- Slow inflation with 10 second inspiratory hold at 40 cmH20

- Recruitable lung
  - Large hysteresis
  - Recruited volume > 400ml

CASE 2 08:30 TRANPULMONARY MONITORING

Within 2 hrs

- Fi02 .60
- PEEP 20 cmH20
- DPtp 7-8 cmH20
- NO off
- Sp02 94%
- ECMO consult cancelled

EARLY intervention is Key!

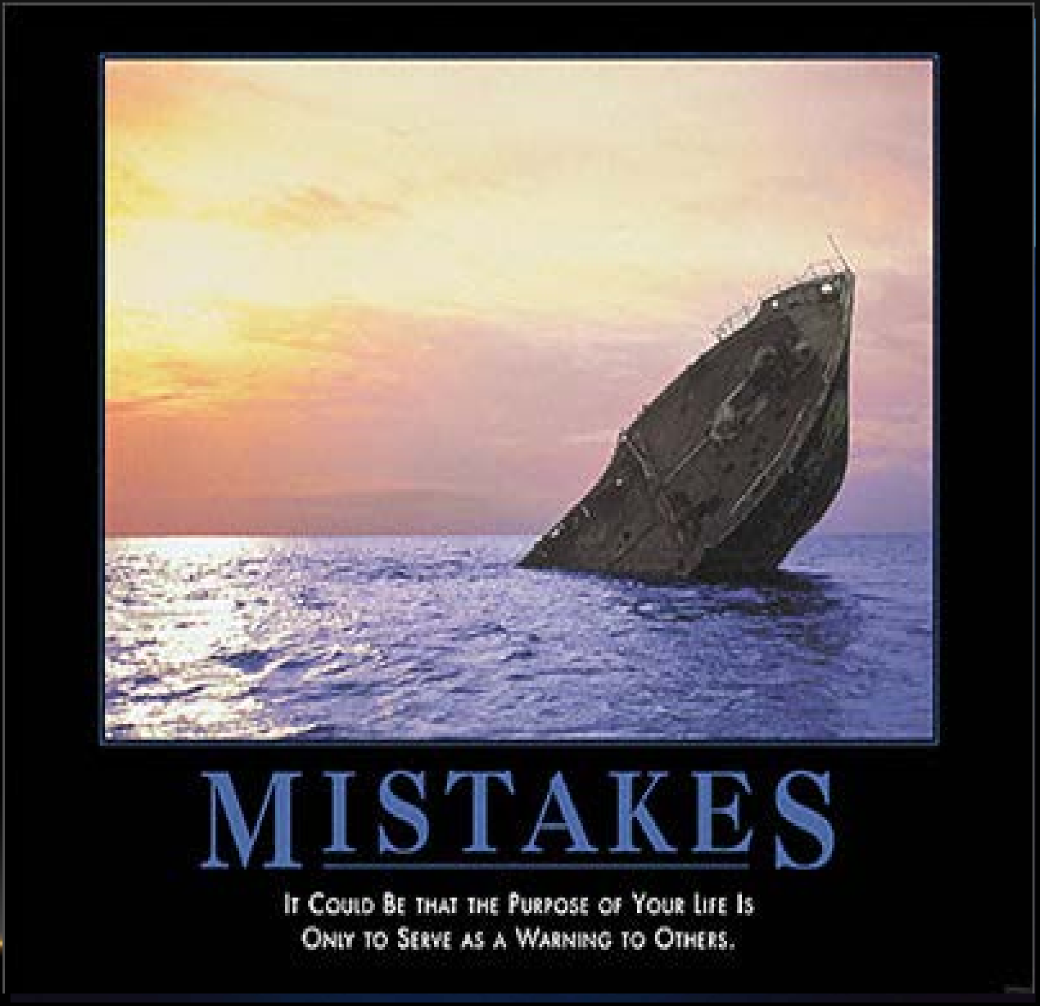
CASE 2 DAY 3 SBT

- PS 8 cmH20 PEEP 10 cmH20
- 2 hrs later emergent call to room
- Vt > 12 ml/kg !
- Driving pressure (DPtp) 30 cmH20 !
- Temp 39.9 C
- Fi02 1.0, PEEP 18, paralytic, prone position, 2 week recovery
- Patient-Self Induced Lung Injury !

HAVE A PLAN !

Predefine SBT success vs. failure criteria:

- RR, Vt, Sp02
- Work of breathing
  - Physical assessment
  - P 0.1, PTP, Ptp
- When to reinstitute sedation and/or paralytics
- Avoid Icebergs



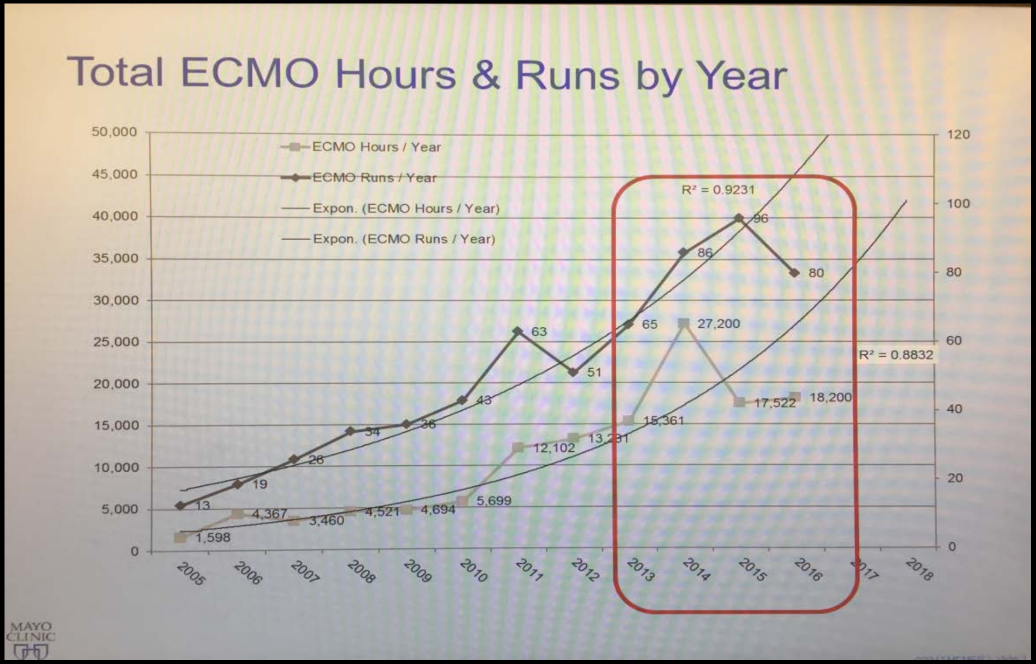
REFRACTORY HYPOXEMIA GUIDELINES

PRELIMINARY (UNPUBLISHED) DATA

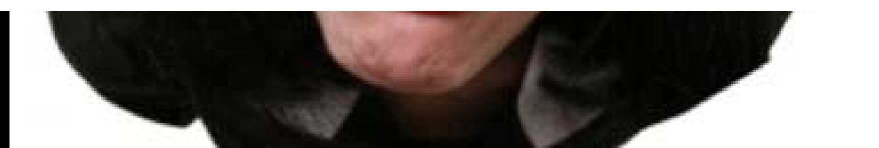
50+ catheters so far
2017

Decreased:

- ECMO
- Nitric oxide usage
- Driving pressures
- Ventilator time ?
- Mortality?



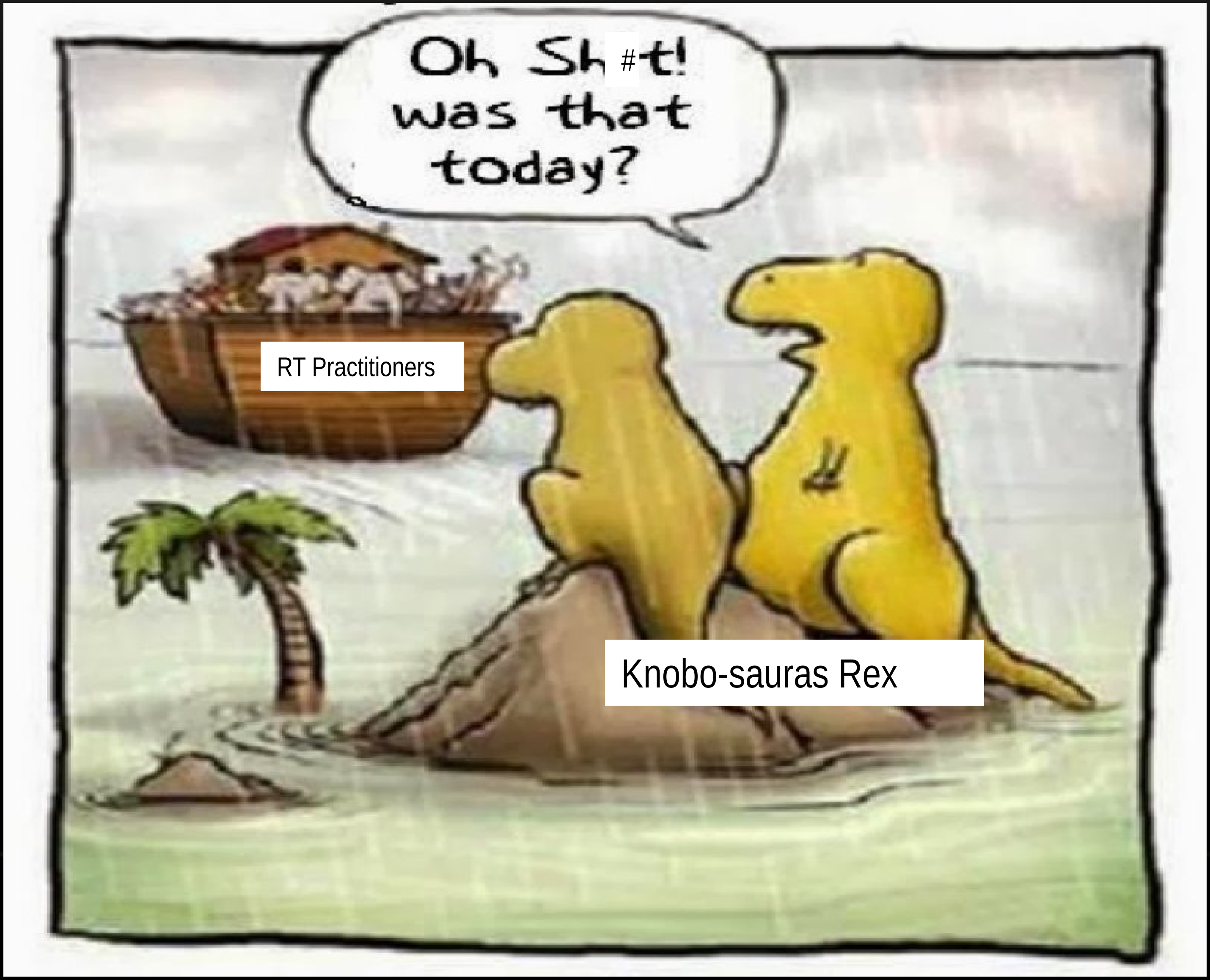
What’s Next?



SUMMARY

- Excessive stress and strain causes lung injury regardless of how generated.
- Early lung stabilization and limiting lung distending pressure are the key components to preventing lung injury.
- During spontaneous breathing early recognition and alleviation of excessive WOB is crucial in preventing P-SILI.
- What <u>YOU</u> do makes a difference!

IT'S TIME TO GET ONBOARD



WHAT ABOUT CARDIAC OUTPUT?

15 minutes

☑ Show only rows with data

☑ Show subheadings

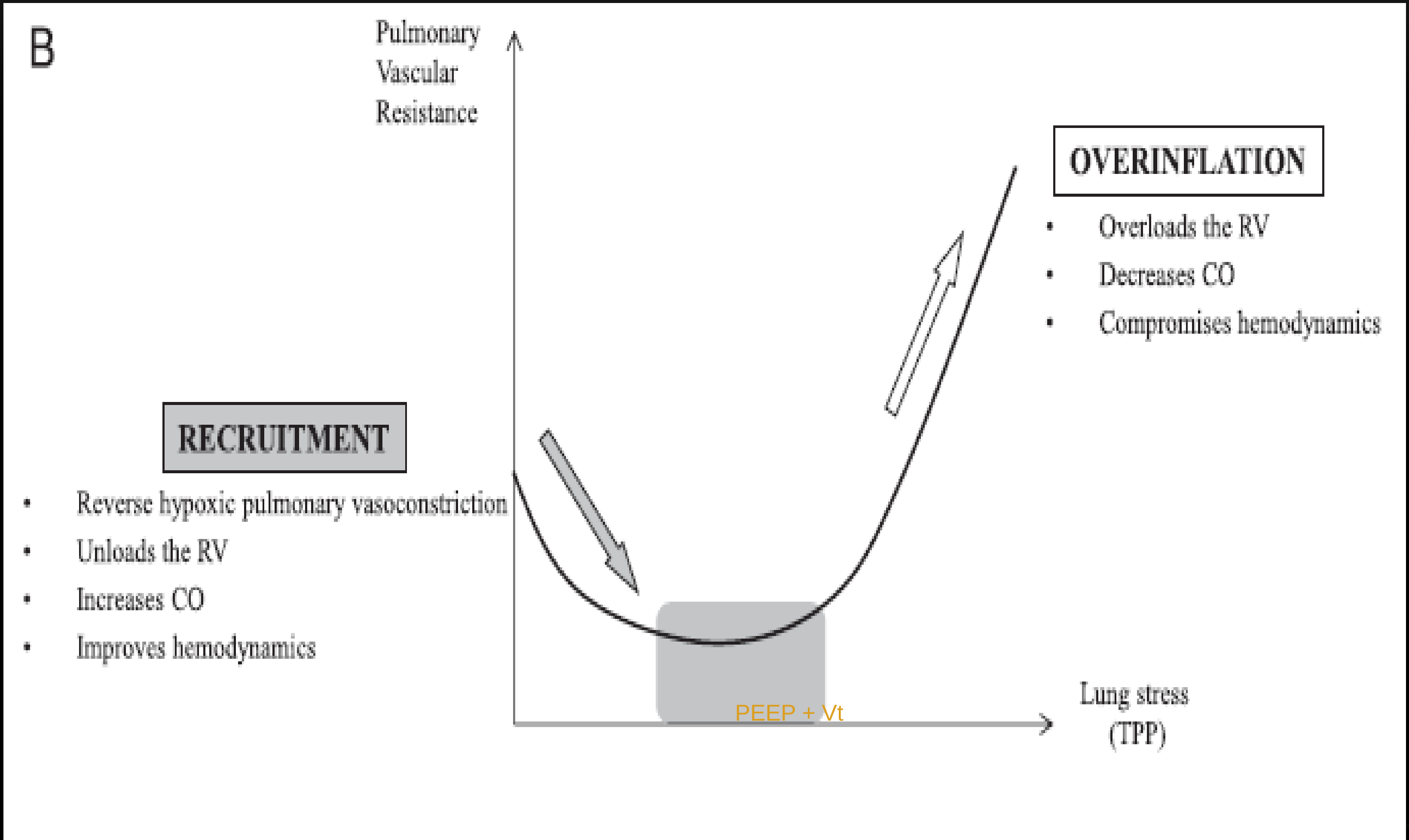
Monitored Care (

12/21

Main Flowsheet

Physiologic Variables	08:45	09:00	09:15	09:30	09:45	10:00
Heart Rate	112	111	82	110	113	113
Arterial BP-Systolic (ABP)	97	95	96	91	95	92
Arterial BP-Diastolic (ABP)	61	62	58	58	63	61
Arterial BP-Mean (ABP)	75	75	72	71	75	73
SpO2	95	95	95	96	96	94
Respiratory Rate	26	26	26	26	26	26
Temperature-Manual					38.0	
Pulse Rate	111	111	81	110	113	113
Ventilator Mode	SIMV	SIMV	SIMV	SIMV	SIMV	SIMV
FIO2/O2%	60.00	60.00	60.00	60.00	50.00	40.00
Tidal Volume Preset	450.0	450.0	450.0	450.0	450.0	450.0
Tidal Volume Expired	470.0	440.0	443.0	444.0	431.0	452.0
Spontaneous Min. Volume	0.00	0.00	0.00	0.00	0.00	0.00
Resp. Rate Set - Vent	26	26	26	26	26	26
Resp. Rate Total - Vent	26	26	26	26	26	26
PEEP	15.0	15.0	15.0	15.0	23.0	21.0
Pressure Support	10	10	10	10	10	10
Peak Flow Setting	70	70	70	70	70	70
Peak Inspiratory Pressure	32.0	31.0	31.0	34.0	39.0	37.0
Mean Airway Pressure	21.0	21.0	22.0	22.0	30.0	28.0
Plateau Pressure	26.0	27.0	28.0	28.0	37.0	34.0
Sensitivity Flow Setting	3.0	3.0	3.0	3.0	3.0	3.0
Respiratory Compliance	41	38	39	40	35	38
Respiratory Resistance	12.0	11.0	10.0	12.0	9.0	10.0
Inspiratory Tidal Volume	473.0	451.0	456.0	452.0	454.0	451.0
Expiratory Minute Volume	12.20	11.40	11.50	11.30	11.30	11.70
Q-T Interval	160.00					
PR Interval	0.15					
QRS Width	0.05					
QTc	220.00					
Inspiratory Pressure	17	16	13	14	14	13
Inspiratory Time	0.77	0.77	1.07	1.07	1.07	1.07
PVC Rate	0	0	0	0	0	0
ST Segment Lead III	0.20	0.20	0.10	0.20	0.20	0.20
ST Segment Lead V	0.80	0.80	0.80	0.90	0.90	0.90
Continuous Cardiac Output			7.60	8.10	7.50	7.50
SVV (CCO)			15	12	14	13
SV (CCO) ml/beat			68	74	66	67
SVI (CCO) ml/beat m(2)			28	31	28	28

EFFECT OF MECHANICAL VENTILATION ON HEMODYNAMICS



Cardiorespiratory Interactions: The Relationship Between Mechanical Ventilation and Hemodynamics

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