

Acute Respiratory Failure

Phil Factor, D.O.
 Associate Professor of Medicine
 Pulmonary, Allergy, and Critical Care Medicine
 Director, Medical Intensive Care Unit
 Columbia University Medical Center

Respiratory Failure

Physiologic Definition:
Inability of the lungs to meet the metabolic demands of the body

 Can't take in enough O_2
 or
 Can't eliminate CO_2 fast enough to keep up with production

Respiratory Failure

- Failure of Oxygenation: $P_aO_2 < 60$ mmHg
- Failure of Ventilation*: $P_aCO_2 > 50$ mmHg


* P_aCO_2 is directly proportional to alveolar minute ventilation

Acute Respiratory Failure

Physiologic Classification

Mechanism	↓		↓
Etiology			
Clinical Setting			

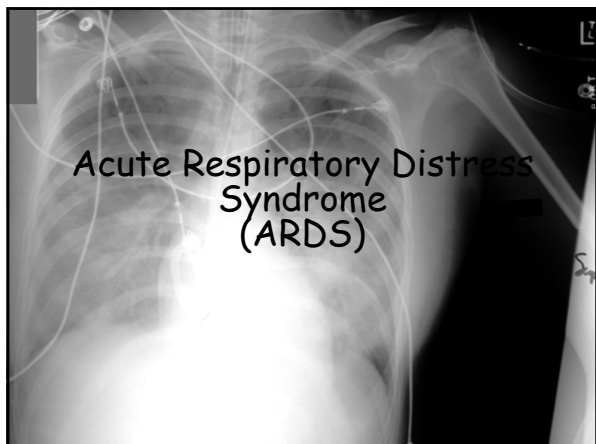
Ventilatory Failure



Inbalance between load on the lungs and the ability of bellows to compensate

Acute Hypoxemic Respiratory Failure

- Shunt disease - intracardiac or intrapulmonary
- Severe V/Q mismatch - asthma, PE
- Venous admixture due to low cardiac output states, severe anemia coupled with shunt and/or V/Q mismatch



Acute Respiratory Distress Syndrome (ARDS)

Leaky alveolar capillaries
 Plasma fluid and leukocytes leak into the airspace
 Shunt
 Hypoxemia

Acute Respiratory Distress Syndrome (ARDS)

American-European Consensus Definition:

- Refractory hypoxemia
 P_aO_2/F_iO_2 (P/F ratio)
 <300 for ALI
 <200 for ARDS
- A disease process likely to be associated with ARDS
- No evidence of elevated left atrial pressure elevation (by clinical exam, echo or PA catheter)
- Bilateral airspace filling disease on X-ray

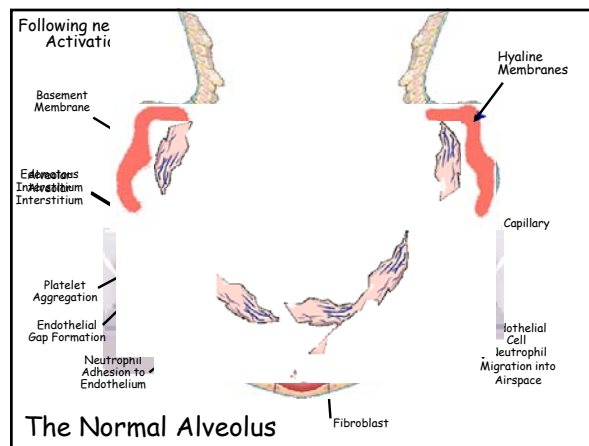
Report of the American-European Consensus conference on acute respiratory distress syndrome: definitions, mechanisms, relevant outcomes, and clinical trial coordination. Consensus Committee.

Acute Respiratory Distress Syndrome

Each year in the United States, *70,000-150,000 cases*

Causes of ARDS

<p>DIRECT LUNG INJURY</p> <ul style="list-style-type: none"> Pneumonia Aspiration of gastric contents Pulmonary contusion Near-drowning Inhalation injury (Cl⁻, smoke) Reperfusion pulmonary edema after lung transplantation or pulmonary embolectomy 	<p>INDIRECT LUNG INJURY</p> <ul style="list-style-type: none"> Non-pulmonary sepsis/SIRS Severe trauma with shock Cardiopulmonary bypass Drug overdose (Narcotics) Acute pancreatitis Transfusion (TRALI) Drug reaction (ARA-C, nitrofurantoin) fat/air/amniotic fluid embolism, bypass
--	--

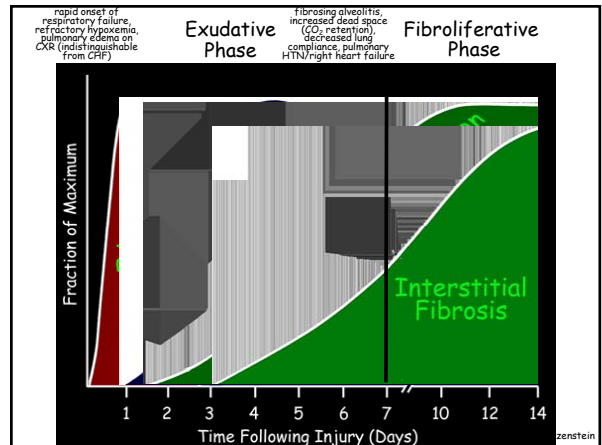
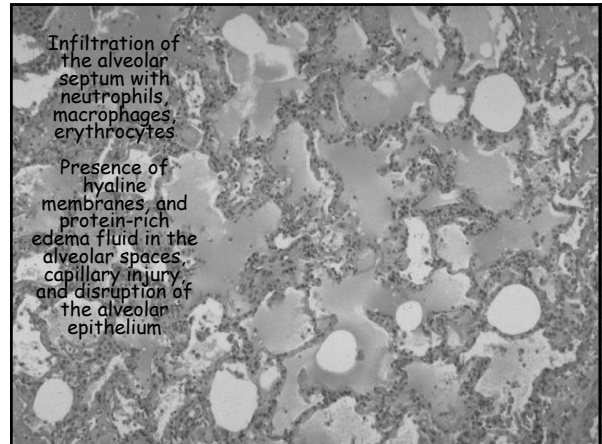


ARDS

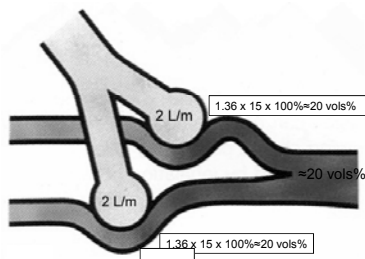
Fundamental Pathophysiology:

Increased alveolar permeability due to direct neutrophil-mediated injury to the alveolar epithelium

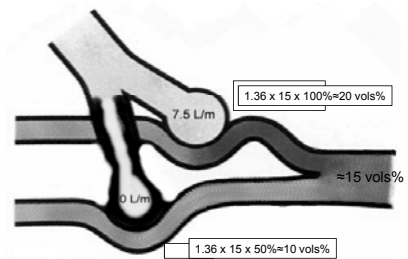
Not a distinct disease - rather a sequelae of activation of lung and systemic inflammatory pathways



Optimal V/Q matching



Shunt

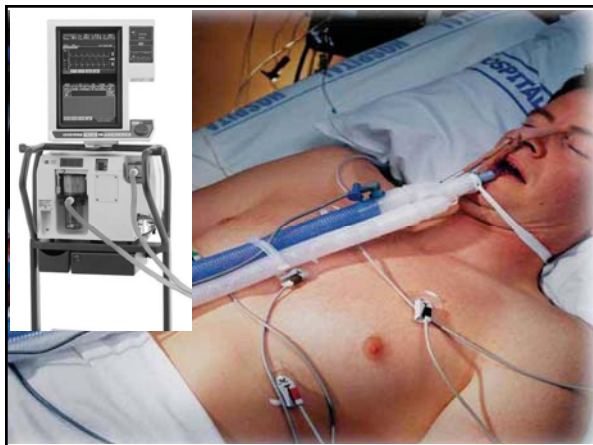


Severe Hypoxemia

Therapeutic Goals

Maintain reasonable oxygen delivery

Find & fix the primary cause



"Baby Lungs"

FRC can be reduced by 80% or more in ARDS

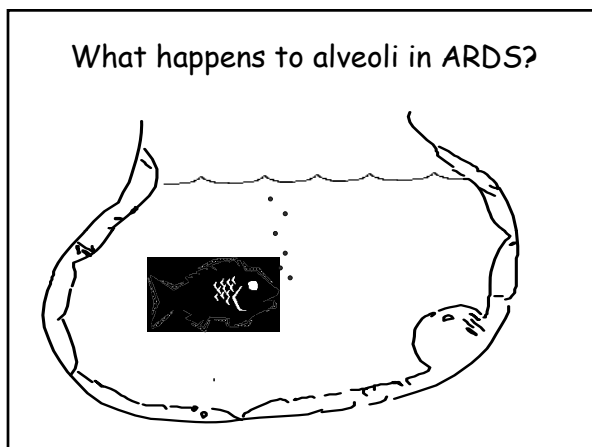
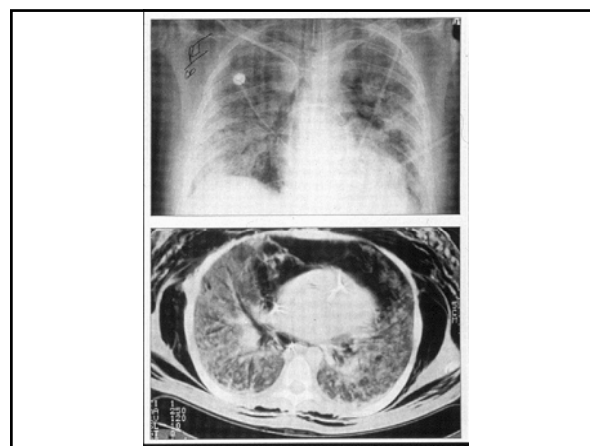
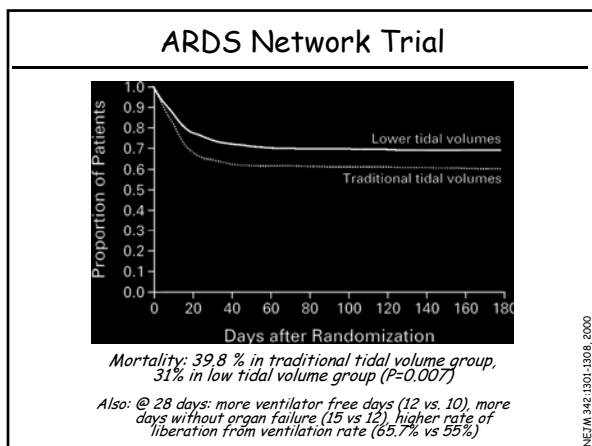
Gattinoni, et. al. Anesthesiology, 74:15-23, 1991.

ARDS Network Trial

Day 1 Ventilatory Characteristics

	Low V _t Group n=432	Traditional V _t Group n=429
V _t :	6.2 ± 0.9	11.8 ± 0.8
PEEP:	9.4 ± 3.6	8.6 ± 3.6
F _i O ₂ :	0.56 ± 0.19	0.51 ± 0.17
P _{plat} :	25.7 ± 7	33 ± 9
P _{peak} :	32.8 ± 8	39 ± 10
P _a O ₂ / F _i O ₂ :	158 ± 73	176 ± 76
P _a CO ₂ :	40 ± 10	35 ± 8
pH:	7.38 ± 0.08	7.41 ± 0.07

NEJM 342:1301-1308, 2000



What happens to alveoli in ARDS?

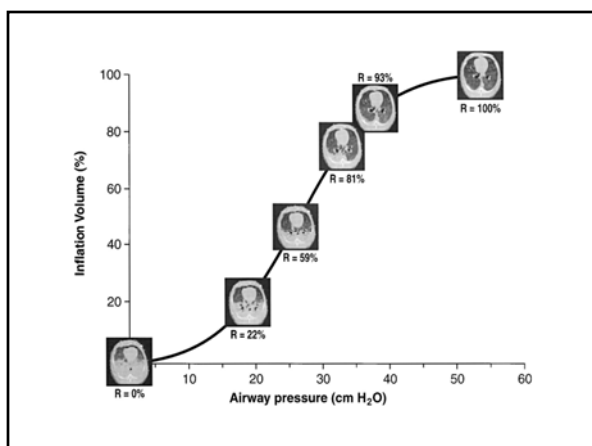
Edema accumulates in alveoli

Diluting & disaggregating surfactant

Surface tension increases

Alveoli collapse

Alveolar collapse decreases FRC and contributes to hypoxemia



Positive End-Expiratory Pressure (PEEP)

- **Beneficial Effects**
 - Increases FRC, CI, $P_{a}O_2$
 - Recruits Atelectatic Units
 - Decreases Q_s/Q_t
 - Allows Reduction in F_iO_2
- **Detrimental Effects**
 - Volutrauma
 - Alveolar Overdistention
 - Hemodynamic Derangements

PEEP

Oxygen is:

- A) good for you
- B) bad for you
- C) all of the above

F_IO₂ > 0.6 for 24 hours or more may cause lung injury

PEEP recruits collapsed alveoli,
improves FRC and
improves oxygenation

An essential therapy for patients with ARDS

ARDS Network Trial

The standard of care

Assist Control
V_T 6 cc/kg ideal body weight
PEEP of ≈8-10

Cause of Death in ARDS Patients?

Generally not due to respiratory failure

Does Mechanical Ventilation Contribute to MSOF?

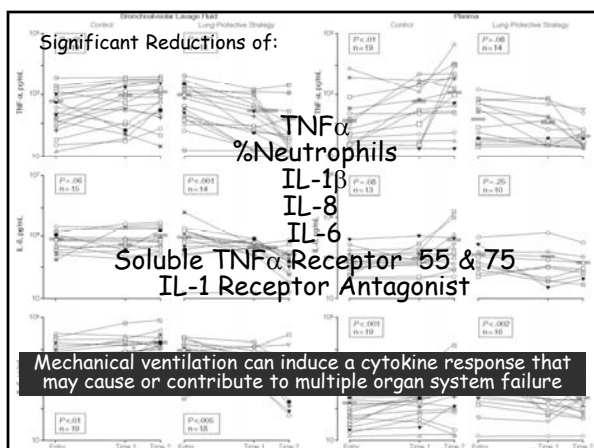
Ranieri, et al.*: randomized prospective study of the effects of mechanical ventilation on bronchoalveolar lavage fluid and plasma cytokines in patients with ARDS (primarily non-pulmonary causes).

Controls (n=19): Rate 10-15 bpm, V_T targeted to maintain PaCO₂ 35-40 mmHg (mean: 11 ml/kg), PEEP titrated to SaO₂ (mean: 6.5), P_{plat} maintained <35 cmH₂O

Lung protective ventilation (n=18): Rate 10-15 bpm, V_T targeted to keep P_{plat} less than upper inflexion point (mean: 7 ml/kg), PEEP 2-3 cmH₂O above LIP (mean: 14.8)

Plasma and BALF levels of IL-1β, IL-6, IL-8, TNFα, TNFα-sr 55, TNFα-sr 75, IL-1ra, measured within 8 hrs of intubation and again @24-30 hours & 36-40 hours after entry

*Ranieri, et al. Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: a randomized controlled trial. JAMA 282:94-61, 1999.



The lung is not just an innocent bystander - it functions as an immunomodulatory organ that may participate in the systemic inflammatory response that leads to multiple organ system dysfunction syndrome

Biotrauma

Goals for Management of ARDS

The American-European Consensus
Conference on ARDS, Part 2

- Ensure appropriate O₂ delivery to vital organs
- Minimize oxygen toxicity/tolerate mediocre ABG's
- Reduce edema accumulation
- Minimize airway pressures
- Prevent atelectasis/Recruit alveoli
- Use sedation and paralysis judiciously

Am J Resp Crit Care Med 157:1332-47, 1998.

Survival from "pure" ARDS

1979: 20-50%

2002: 50-90%