Near-Drowning

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Definitions

- **Drowning** – suffocation by immersion, especially in water
  - Death resulting from suffocation within 24 hours of submersion in water

- **Near-drowning** – initial survival after submersion for at least 24 hours
You can’t talk about it you drowned!!!
# Near Drowning Classification

<table>
<thead>
<tr>
<th>Grade</th>
<th>Symptoms Respiratory</th>
<th>Symptoms Hemodynamic</th>
<th>Treatment</th>
<th>Destination</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No cough or dyspnea</td>
<td>Palpable radial pulses</td>
<td>Release at scene, Education</td>
<td>NO transport</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>Cough, normal auscultation</td>
<td>Palpable radial pulses</td>
<td>Rest, rewarm, reassure, and release</td>
<td>NO transport</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>Rales and small amount of foam</td>
<td>Palpable radial pulses</td>
<td>Oxygen via nasal cannula, observe for 6-24 hours</td>
<td>Transport for Observation: ED or overnight</td>
<td>0.6</td>
</tr>
<tr>
<td>3</td>
<td>Acute Pulmonary Edema</td>
<td>Palpable radial pulses</td>
<td>Oxygen non-rebreathing mask ACLS</td>
<td>Transport for Admission and observation</td>
<td>5.2</td>
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<tr>
<td>4</td>
<td>Acute pulmonary Edema</td>
<td>Hypotension</td>
<td>Possible ETT and fluids for blood pressure support ACLS</td>
<td>Rapid transport: ICU stay</td>
<td>19</td>
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<tr>
<td>5</td>
<td>Respiratory Arrest</td>
<td>Hypotension</td>
<td>Load and go. ACLS</td>
<td>Rapid transport ICU</td>
<td>44</td>
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<tr>
<td>6</td>
<td>Cardiopulmonary Arrest</td>
<td>ACLS</td>
<td>Rapid transport ICU</td>
<td>Rapid transport ICU</td>
<td>93</td>
</tr>
</tbody>
</table>

Where Do Drowning Occurs?
Epidemiology in Child/Teenagers

- Major cause of pediatric unintentional death worldwide with > 140,000 cases
- Second leading cause of unintentional death among U.S. children < 20 years old
- Leading cause of injury death and second leading cause of death overall in toddlers 12-23 months
- Increase occurrences secondary to the increased purchases of inflatable pools!!
- Highest rates in children < 5 years old and second among 15-19 year olds
Where incidents happened

- Private pool: 69 (includes 10 deaths)
- Condominium: 54
- Hotel: 11
- Private home: 3
- Public pool: 15
- Public recreational pool: 7
- Bathtub/jacuzzi: 5
- Pail/bucket: 3
- Open sea: 5

Source: KK WOMEN'S AND CHILDREN'S HOSPITAL

STRAITS TIMES GRAPHICS
Location

- 78% of infant drownings occur at home
  - 55% occur in bathtubs
  - 12% occur in buckets or other containers
- Swimming pools account for > 50% of drownings among children aged 1-4 years
- 37% of all drownings occur in other open bodies of water
Causes

- Inadequate supervision!!!
- Alcohol
  - Positive blood alcohol levels are present in 30-50% of adolescent drownings
- Neurological disorders
  - Seizures
  - Autism
- Clear evidence that drowning rates are higher among poor swimmers
- Child abuse
Prevention

- Adult supervision
- Fencing
- Swimming lessons
  - reduces chance of drowning by 85%
- Life jackets
- CPR training
Epidemiology of Drowning in Adults

- ETOH Consumption
  30-50% of 15-29 yrs. have ETOH level > 200
- Inability to swim
- Medical emergency occurrence in water
  - Stroke, MI, spinal injury
- Exhaustion
- Boating accidents
- Suicide attempt
- Animal bites
- Environmental conditions

- Vast majority of drowning (≈90%) are accidental
Where does Drowning Occur? In Adults
African Americans 6 times greater 5-19 age group


* Rates are per 100,000 population; rates of other races are unstable and are not presented.

Rates are suppressed because the state had between 0 and 9 deaths during the 1999-2007 timeframe.
Rates are suppressed because the state had between 10 and 20 deaths during the 1999-2007 timeframe.
* Age adjusted rate per 100,000
Pennsylvania Stats

390 kids under 14 die by accidental drowning
5,200 kids require E.R. treatment for drowning
#1 cause of DEATH for kids under age 5

CPSC
When Does Drowning Occur?

- Peak occurrences between May and August
- Occurrences greatest on weekends
- Rates vary inversely with per capita income
- Males are at greater risk than females
Types of Body of Water
Cold Water Drowning

- Water temperatures of $< 70^\circ F$
  - Water conducts body heat away 26x faster than air of the same temperature

- Hypothermia - Core body temperature of $< 95^\circ F$
  - $\downarrow$ Heart rate
  - $\downarrow$ Respiratory rate
  - $\downarrow$ Metabolic rate
  - $\downarrow$ Oxygen requirement
Cold-Water Drowning

- Patients fully resuscitated after submersions of up to 1 hour
- Diving reflex
  - ↓ Heart rate
  - ↓ or cessation of respirations
- Shunting of blood to core organs: heart, lungs, brain
Support/Rescue

- **Initial ABC’s** – assess and treat
  - Airway – intubate if cardiac arrest or no gag reflex present
  - Breathing – PPV with supplemental $O_2$
  - Circulation – CPR with ALS – ASAP!!!
- Immobilize if suspected c-spine injury
Survival Factors

- Age
- Length of submersion
- Water temperature
- Water type and quality
- Other injuries – e.g. C-spine
- CPR – time to onset and proper technique
Pathophysiology
Pulmonary Insult

- **Dry drowning**
  - 10-15% of all cases
  - No aspiration of fluid into lungs
  - Laryngeal spasm—leading to negative pressure pulmonary edema

- **Wet drowning**
  - 85-90% of all cases
  - Volume, type, and components of aspirated fluid determine the extent of injury
In negative pressure pulmonary edema, pulmonary mechanics and hemodynamics are stable. In fact, they work great. The problem is when something closes off the airway above the lungs and the person starts to asphyxiate.

The normal reaction is to try to suck in air as hard as possible. But, with the larynx blocked off, you can't. Pleural pressure can exceed -80 cmH2O.
• This literally creates a vacuum effect in the alveoli and the fluid gets PULLED into the alveoli!
• This is what happens when someone chokes on a piece of food, or a child swallows a marble or peanut down the airway.
• It can also occur during epiglottitis
Bilateral White Out!!!
Fresh Water Drowning

- Hypotonic
- Rapidly moves across alveolar-capillary membrane and absorbed into blood
- Destroys surfactant
- Creates alveolar instability
- End result is atelectasis
Salt Water Drowning

- Hypertonic
- Fluid leak into alveoli
- Surfactant washout
- Increase surface tension within alveoli
- Alveolar collapse
- Atelectasis
Common End Result

- Non-cardiogenic pulmonary edema
- Atelectasis
- V/Q mismatch
- Intrapulmonary (Qs/Qt) shunting
- ↓ FRC
- HYPOXEMIA → ARDS
Other End Results

- Hypovolemia
- Myocardial dysfunction
- Acidosis
- Cardiac Output
- Pulmonary hypertension
- Multi-organ system failure
Near Drowning: ARDS
Acute Respiratory Disease Syndrome
Historical Perspective

- ARDS Recognized by Ashbaugh/Petty-1967
  - Coined DaNang Lung
  - Shock lung/ventilator lung
  - Stiff lung
  - Re-perfusion lung

- Re-defined over past four decades and recognized now as a disease process
Berlin Criteria

- Acute Lung Injury (ALI) no longer exists
- Onset is now defined as <7 days
- No exclusion of heart failure in definition
Definition

• **Mild**
  – P/F <300>200
  – Mortality 24%

• **Moderate**
  – P/F <200>100
  – Mortality 34%

• **Severe**
  – P/F <100
  – Mortality 44%
3 Stages of ARDS
Pathophysiology

• The injured lung goes through 3 phases:

  Edema
  • Reparative
  Fibrotic
Edema Phase

- Occurs in the first week after onset of respiratory failure
- Inflammatory cells migrate into the lungs and release substances to cause capillary leakage
- Type I pneumocytes swell and detach from basement membrane
- Increased pulmonary vascular permeability
- Alveolar collapse
Homogeneous and Heterogeneous Alveolar Ventilation

Normal Lung

ARDS Lung

Reparative Phase

- May begin as early as 3\textsuperscript{rd} day, but usually prominent in 2\textsuperscript{nd} and 3\textsuperscript{rd} week after onset
- Type II cells proliferate and reline membrane
- Fibroblast infiltration – migration through breaks in membrane forming granulation tissue
- Surfactant abnormalities occur – damage to Type II and alveolar flooding destabilize the surfactant layer - Marked by poor gas diffusion
Fibrotic Phase

- May begin as early as two weeks after injury
- Extensive remodeling by collagenous tissue
- Alveolar duct fibrosis
- Elastic collagen replaced by rigid collagen – resulting in stiff lung
- Extent of fibrosis correlates with mortality
- $\text{VD/VT}>60\%$ Large amount of wasted ventilation
Structural Changes

- Damage to type I alveolar epithelial cells
- Increase edema influx
- Loss of surfactant
- Poor fluid clearance mechanism
- Development of a hyaline membrane
- Reduction in gas exchange
- Pulmonary Fibrosis development
- “Liver” lung appearance
Neurological Insult

- Cerebral edema
- Autonomic instability – hyperstimulation of sympathetic nervous system
- ? Seizures
- Hypotension
- Possible CNS infection
Neurological Insult

- **Anoxia/Hypoxia** – insufficient O\(_2\) supply
  - Loss of consciousness within 1-2 minutes
  - Brain damage after 4-6 minutes
    - Dilated pupils
    - Impaired sensorium

- **Ischemia** – blood flow or blood O\(_2\) content is markedly diminished
Neurological Insult

- Brain cells are strictly aerobic
  - If no O$_2$, transport mechanism slows or stops completely
  - K$^+$ leaks out of cell
  - Na$^+$ and Ca$^+$ flood into cell
Outcomes

- Victims usually survive neurologically intact or die
- Drowning is the second leading cause of death in ICU’s
- Worst neurological injury occurs from brain anoxia or ischemia
Predictors of Outcome

- Presence or absence of coma
  - Glasgow Coma Score
    - 3 is lowest possible score
    - 7 or less indicates coma
    - 14 is full consciousness
- Pupillary response
- Blood glucose levels
Table 1: THE GLASGOW COMA SCALE AND SCORE

<table>
<thead>
<tr>
<th>Feature</th>
<th>Scale Responses</th>
<th>Score Notation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Verbal response</td>
<td>Orientated</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Confused conversation</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Words (inappropriate)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Sounds (incomprehensible)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Best motor response</td>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localise pain</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion – Normal</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>– Abnormal</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Extend</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

TOTAL COMA ‘SCORE’ 3/15 – 15/15
Mortality and Morbidity

- ARDS accounts for 1-14% of patients admitted to PICU
  - 72% require intubation
  - Mortality rate 22% (higher among drowning)
- Factors affecting prognosis
  - Initial severity of hypoxemia
  - Presence of non-pulmonary organ system failure
  - Presence of CNS dysfunction
Management / Support

- Airway
- Breathing
- Ventilation
Management

- **Lung protective strategies – Pressure Control**
  - Lower $V_T$ of $< 6$ ml/kg (patients had lower incidence of end-organ complications including cardiac and renal failure and DIC)
  - Higher PEEP – “optimal” PEEP for alveolar recruitment
  - $\uparrow T_i$
  - Maintain peak airway and plateau pressures $< 30$ cmH$_2$O
  - Permissive Hypercapnia

**Optimize oxygen delivery**
- ? cardiac support
Ventilator Induced Injury

- Normal lung
- After 5 min at high pressure/high volume
- After 20 min at high pressure/high volume
PV Curve to Determine PEEP

- Upper Inflection Point
- Lower Inflection Point
- Tidal Volume
- PEEP
- PIP
- Alveolar Overdistention
- Alveolar Optimal Range
- Alveolar Collapse
Transpulmonary Monitoring

$\text{PIP} = \frac{\text{RAW}}{\text{total CLT}} \enspace 40\text{cm}$

$\text{PLT} = \frac{\text{Thoracic/pulmonary CLT}}{} \enspace 32\text{cm}$

$\text{TranspI} = \frac{\text{pulmonary CLT}}{} \enspace 22\text{cm}$

$\text{TranspI} = \text{what pressure the lung is receiving}$
Clinical Indications

• High ventilator settings:
  – PEEP > 12 cm
  – Airway pressures > 35 cm

• Patients with large abdomen and/or thoracic impedance
  – BMI > 40
  – Positive fluid status > 20 liters

• V-V ECMO patients

• Very difficult to wean patients
TranspE

TranspE< -2

TranspE> 2 cm
Post esophageal balloon placement.

Peak airway pressure minus esophageal pressure equals: Transpulmonary pressure (pressure the lung is receiving) Goal < 25 cm

Note peak airway pressure is 42 cm, Esophageal pressure 21 cm thus transpulmonary pressure (the lung receiving) only 19 cm

PEEP set at 14 cm
Based on PtransE Between -2 to +2 cm

Transpulmonary Monitoring

**Good esophageal deflections**

**PtransE around zero**
Ventilatory Techniques in the Management of Acute Lung Injury

- ARDS/net low volume ventilatory protocol
- High Frequency Percussive Ventilation (HFPV)
- Airway Pressure Release Ventilation (APRV)
- High Frequency Oscillatory Ventilation (HFOV)
Lung Protective Strategy

• Based on ARDSnet clinical trials
• Low tidal volume/plateau pressure
• Goal is not to re-inflate sick lung units but protect healthy lung units
• Marked by SpO$_2$ > 90% and no reduction in infiltrates via x-ray
• Maintain for two-three weeks
INCLUSION CRITERIA: Acute onset of
1. PaO₂/FiO₂ ≤ 300 (corrected for altitude)
2. Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema
3. No clinical evidence of left atrial hypertension

PART I: VENTILATOR SETUP AND ADJUSTMENT
1. Calculate predicted body weight (PBW)
   Males = 50 + 2.3 [height (inches) - 60]
   Females = 45.5 + 2.3 [height (inches) - 60]
2. Select Assist Control Mode
3. Set initial TV to 8 ml/kg PBW
4. Reduce TV by 1 ml/kg at intervals ≤ 2 hours until TV = 6 ml/kg PBW.
5. Set initial rate to approximate baseline VE (not > 35 bpm).
6. Adjust TV and RR to achieve pH and plateau pressure goals below.
7. Set inspiratory flow rate above patient demand (usually > 80L/min)

OXYGENATION GOAL: PaO₂ 55-80 mmHg or SpO₂ 88-95%
Use incremental FiO₂/PEEP combinations below to achieve goal

<table>
<thead>
<tr>
<th>FiO₂</th>
<th>0.3</th>
<th>0.4</th>
<th>0.4</th>
<th>0.5</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
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<tbody>
<tr>
<td>PEEP</td>
<td>5</td>
<td>5</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>12</td>
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</table>

<table>
<thead>
<tr>
<th>FiO₂</th>
<th>0.7</th>
<th>0.8</th>
<th>0.9</th>
<th>0.9</th>
<th>0.9</th>
<th>1.0</th>
<th>1.0</th>
<th>1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEEP</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>16</td>
<td>18</td>
<td>20</td>
<td>22</td>
<td>24</td>
</tr>
</tbody>
</table>

PLATEAU PRESSURE GOAL: ≤ 30 cm H₂O
Check Pplat (0.5 second inspiratory pause), SpO₂, Total RR, TV and pH (if available) at least q 4h and after each change in PEEP or TV.

If Pplat > 30 cm H₂O: decrease TV by 1 ml/kg steps (minimum = 4 ml/kg).
If Pplat < 25 cm H₂O: TV < 6 ml/kg, increase TV by 1 ml/kg until Pplat > 25 cm H₂O or TV = 6 ml/kg.

If Pplat < 30 and breath stacking occurs: may increase TV in 1 ml/kg increments (maximum = 8 ml/kg).

pH GOAL: 7.30-7.45
Acidosis Management: (pH < 7.30)
   If pH 7.15-7.30: Increase RR until pH > 7.30 or PaCO₂ < 25 (Maximum RR = 35).
   If RR = 35 and PaCO₂ < 25, may give NaHCO₃.
If pH < 7.15: Increase RR to 35.
   If pH remains < 7.15 and NaHCO₃ considered or infused, TV may be increased in 1 ml/kg steps until pH > 7.15 (Pplat target may be exceeded).
Alkalosis Management: (pH > 7.45) Decrease vent rate if possible.
ARDSnet

- Decreased mortality by 22\% when compared to an *injurious* ventilatory strategy!!
- Does the reduction in tidal volume make a difference if the Plt < 30cm H$_2$O?
- May not be applicable in all patient populations (Dicker, et al *Journal of Trauma*, Sept 2004)
- **Gold standard for ARDS??**
Lung Recruitment Strategy

• Best clinical results if implemented within forty-eight hours
• Improvement noted by increased P/F ratio and reduction in chest x-ray infiltrates
• Maintained for several days
What is APRV?

- Two levels of CPAP
- Long upper pressure level (> 5 seconds) with short expiratory releases (< 1 second)
- Marked by spontaneous breathing during the long upper pressure level
Airway Pressure Release Ventilation (APRV)

APRV is marked by a long inspiratory time and a short expiratory time.

Phigh

Release volume
Nurse reaction to VDR coming into her patient’s room!!!!
High Frequency Percussive Ventilation

• Classified as:
  - pneumatically driven
  - pressure limited
  - time cycled
  - high frequency flow interrupter
  - delivers high frequency in a range of 200-900 cycles
  - exhalation is passive
HFPV Waveform
HFPV
PiP 12 PEEP 0 (DELTA / P = 12)
Repetitive Alveolar Collapse & Re-expansion (R.A.C.E)
HFPV
PIP 24 PEEP 12 (DELTA / P = 12)
LUNG STABILIZED WITH O/CPAP
Development Of The Endobronchial Wedge

Bulk Gas Delivery

Oscillatory Sub-tidal Volumes
Enhanced muco-kinesis
Enhanced Muco-kinesis
Adjunctive Therapies
Nitric Oxide Administration

INo – studies in the 1990’s showed short-term improvement in outcomes

*↓* PA pressures
*↑* PaO₂/FiO₂ ratio
Adjunctive Therapies

- **Surfactant replacement**
  - Gregory et al – ARDS patients have altered surfactant composition
  - Hallman et al – surfactant is quantitatively deranged in ARDS patients
  - Appears to improve oxygenation acutely and lead to more rapid weaning from mechanical ventilation
Prone Positioning
Why Do ECMO?

• Veno-venous ECMO takes over the ventilation and oxygenation properties of the lung
• It allows the ventilator to be set on “rest” lung settings while the lung recovers from the underlying illness
• The goal is to oxygenate/ventilate the patient with ECMO while avoiding ventilator induced lung injury!
Single-site approach to venovenous ECMO cannulation:

A dual-lumen cannula is inserted in the internal jugular vein (extending through the right atrium and into the inferior vena cava). Venous blood is withdrawn through one “drainage” lumen with ports in both the superior and inferior vena cava. Reinfusion of oxygenated blood occurs through the second lumen, with a port situated in the right atrium. Inset: The two ports of the “drainage” lumen are situated in the superior and inferior vena cavae, distant from the reinfusion port. The reinfusion port is positioned so that oxygenated blood is directed across the tricuspid valve and directly into the right ventricle. This arrangement significantly reduces recirculation of blood when the cannula is properly positioned.
Single Catheter, Double lumen Catheter - Avalon
Case Presentation

- Seventeen year old male
- Playing basketball and drinking beer over a ten hour period
- Decide to go swimming to ‘clean up’ in order not to be hassled by parents
- Drove to George Washington Motel (Home Depot now)
- Jumped in pool
What Happens Next?

- Drunk, not an experienced swimmer, and very tried
- Abdominal cramps and went under
Now What???

- Prayed to God
- Somebody at pool pulled me out
- Woke up outside pool and heard loud crowning—me breathing
- Was ecstatic I was alive and drove home
What’s Next?

• Got home
• Nobody home
• Went into parents bed and developed fulgurating pulmonary edema
• Parents return home five hours later
• Dad goes next store and get Paramedic student-“Get him to the ER STAT!!!!
Clinical Course

- PaO2 < 50 on admission
- Placed on 100% Oxygen
- Spent five days in ICU
- 20-ABGs/60 IPPB txs/hrs of CPT therapy
- Discharged seven days after incident—minimal residual injury?
- Decides to do something positive and enrolls @LCCC in RTT program!!!
Conclusions

- Drowning is a major cause of mortality and morbidity in infants, child and adults.
- Morbidity is directly related to the degree of pulmonary and neurologic insult.
- Early intervention is key for optimal outcomes.
Conclusions

- Enjoy the water, but be cautious!!!