Systemic Inflammatory Response to Cardiac Surgery

- Inflammation is the body’s response to disruption within the tissues
- Stimulates a series of controlled humoral and cellular reactions
- Activators
  - Trauma of surgery
  - Blood transfusion
  - Hypothermia
  - Cardiopulmonary Bypass (CPB)
- Hyperdynamic state without documented infection (SIRS)

Systemic Response of CPB

- Activation of immune system when blood comes in contact with the foreign substances of the circuit
- Aortic cross clamp causes reperfusion injury to the brain, kidneys, liver, heart, and lungs
- Characterized by the release of pro-inflammation factors
Post op effects

- Pulmonary dysfunction
  - Acute lung injury
- Cardiac dysfunction
  - Global myocardial dysfunction
  - Peripheral vasodilation

Treatment

- Supportive care until inflammatory response resolves

Protamine Reactions
(Give test dose)

- Type I
  - Systemic hypotension from rapid administration.
  - Give protamine over 10 – 15 minutes
- Type II
  - Anaphylactice or anaphylacotiod reaction: hypotension, bronchospasm, flushing, edema
- Type III
  - Catastrophic pulmonary vasoconstriction, systemic hypotension, myocardial depression

Protamine Reactions

Minor
- Hypotension
- Elevated PA pressures

Severe
- Massive systemic vasodilation
  - Hypotension
  - Decreased SVR
  - Increased CO
- Acute pulmonary vasoconstriction
  - Increased PAP
  - RV failure

Hemodynamic profile
- Bradycardia
- Decreased CO
- Elevated PAP
- Elevated SVR & PVR

Protamine Reaction Risk Factors

- Allergy to fish
- Use of NPH insulin

Protamine Reaction Treatment

- Calcium Chloride 500 mg to support systemic resistance and provide some inotropic support
- a-agents (phenylephrine, norepinephrine) to support systemic resistance
- B-agents for inotropic support that can also reduce pulmonary resistance (low dose epi, dobutamine)
- Aminophylline to manage wheezing
- Heparin to reverse protamine reaction
**Neurology/Gastrointestinal**

- Encephalopathy
  - Generic term referring to several types of brain dysfunction.
    - Confusion
    - Delirium
    - Lethargy
    - Depression
    - Disorientation
    - Hallucinations
    - TIAs
    - Mental status changes
    - Combative ness
    - Agitation
  - May be related to the development of microemboli, cerebral edema secondary to the inflammatory response, inadequate temperature regulation, or cerebral hypoperfusion

**Delirium**

- Most common neuro complication post cardiac surgery
  - Disturbance of consciousness with inattention
  - Accompanied by changes in cognition or perceptual disturbance
  - An acute onset with fluctuation course
  - Develops over the course of hours to days and may be life threatening
  - Usually reversible

**Etiologies of post op Delirium**

- Sleep deprivation
- Renal or hepatic failure
- Cardiogenic shock
- A-Fib
- Massive blood transfusions
- Bilirubin > 2 mg/dL
- Hypoalbuminemia
- Low hematocrit
- Acute infection
- Dehydration
- Thyroid disorders
- Electrolyte imbalances

**Symptoms of Delirium**

- Inability to maintain attention
- Disturbance of consciousness
- Cognitive deficits
- Memory impairment
- Disorientation
- Inappropriate speech
- Perceptual changes
- Fluctuations in level of alertness
- Agitation may accompany symptoms
Treatment of Delirium

1. Identify and treat underlying causes
2. Provide environmental and support measures
3. Administer drug therapy aimed at treating the symptoms
4. Conduct regular evaluations of the effectiveness of treatment

Treatment of Delirium

Identify and treat underlying causes
- Create an environment to promote sleep
- Treat electrolyte imbalances
- Treat drug or alcohol withdrawal
- Treat nutritional deficiencies
- Discontinue contributing medications
  - Avoid benzos and barbiturates
  - Treat with antipsychotics -- Haloperidol

ICU DELIRIUM

A PREVENTABLE COMPLICATION OF BEING HOSPITALIZED IN THE ICU

Jennifer Lanza, RN, BSN, CCRN
Rush University College of Nursing

DELIRIUM: HOW YOU ARE AFFECTED

- Intensive Care Unit (ICU) delirium needs to be important to every staff nurse that cares for ICU patients.
- It is a very extensive problem that affects as high as 80% of patients hospitalized in the ICU.
- It is undiagnosed in almost 60% of cases.
- ICU delirium is associated with a cost to the United States of 4-16 billion dollars annually.

DELIRIUM: HOW YOU ARE AFFECTED

- ICU delirium can contribute to increased ventilator days, long term neuropsychological dysfunction and increased morbidity and mortality.
- It has also been linked to an overall poorer quality of life in survivors.
- ICU delirium also affects the nursing unit leading to increased stress, job dissatisfaction, burn-out, and a non-therapeutic environment for other patients.

DEFINING DELIRIUM

The DSM IV defines delirium as a disturbance of the consciousness with inattention, accompanied by a change in cognition or perceptual disturbance that develops over a short period (hours to days) and fluctuates over time.
There are three types of delirium:

- **Hypoactive**
- **Hyperactive**
- **Mixed**

This is the second most common form of ICU delirium.
- It is often under-recognized and undiagnosed by ICU staff.
- Many times this form is mistaken for depression.
- Signs and Symptoms include:
  - Difficulty to arouse, somnolent
  - Slowed actions
  - Confusion
  - Withdrawn or distant
  - Flat affect

This is the most recognized and the most difficult type of delirium to care for.
- It is much less common than the other two forms.
- Signs and Symptoms include:
  - Agitation
  - Increased non-purposeful activity
  - Confusion
  - Hallucinations and delusions
  - Paranoia
  - Aggressive and combative behavior

This type of delirium is the most common of the three.
- It's presentation is a mixture of the other two types.
- The treatment for all three types of delirium is relatively the same.
- The most important aspect in treating delirium is detection.

The complete etiology of delirium is unknown, but it is thought to be due to a neurotransmitter imbalance.
- The neurotransmitters most likely affected are acetylcholine, dopamine, and y-aminobutyric acid.
- These imbalances lead to changes in mood, behavior, and cognition.

The neurotransmitters most likely affected are acetylcholine, dopamine, and y-aminobutyric acid.
- The causes leading to imbalance in these neurotransmitters are unknown.

- **Predisposing Factors**
- **Precipitating Factors**
- **Pharmacological Factors**
**Predisposing Factors in the Development of Delirium**

These are considered less modifiable risk factors for delirium development.

- Pre-existing dementia
- Alcoholism
- Age > 70 years
- Living in a SNF
- Smoking
- Hearing or visual loss
- History of CHF, CVA, epilepsy, or depression

**Precipitating Factors in the Development of Delirium**

These are considered more modifiable risk factors and also include environmental factors.

- Hypoxia
- Infection
- Organ failure
- Fever
- Dehydration
- Head trauma
- Noise
- Light
- Sensory overload
- Social isolation

**Pharmacological Factors in the Development of Delirium**

- Medications have consistently been proven the culprit in the development of ICU delirium, and can account for 12-39% of all cases.
- It is important for the nurse and physicians to review patient's medications daily to determine necessity and risk of delirium.

**Medications Associated with Delirium**

- **Opioid Analgesics:** Fentanyl, Morphine, Demerol
- **Benzodiazepines:** Ativan, Versed, Valium
- **Antidepressants:** Pamelor, Elavil
- **Antihistamines:** Benadryl
- **General Anesthetics:** Ketamine, inhalation anesthetics
- **Corticosteroids:** Solu-Medrol, Prednisone, Decadron
- **Beta Blockers:** Inderal, Lopressor
- **Antihypertensives:** Norpace, Digoxin
- **Antipsychotics:** Zantac, Pepcid
- **Antiarrhythmics:** Norpace
- **Diuretics:**

**“THINK” Mnemonic**

To identify causes:

- **T**oxic situations
  - CHF, shock, dehydration
- **H**ypoxemia
- **I**nfection/sepsis (nosocomial)
- **N**oimmobilization
- **G**eneral anesthesia interventions (Are these being neglected?)
  - Hearing aids, glasses, sleep protocols, music, noise control, ambulation
- **K**+ or electrolyte problems

Source: Society of Critical Care Medicine & AACN Practice Alert
**DETECTION OF DELIRIUM**

- The **MOST** important aspect of delirium care for a staff nurse is **DETECTION**.
- The most accurate way, that research has shown, to detect delirium is through assessment screening.
- The only assessment that can be used on non-verbal patients and has the most success in the ICU is the: **Confusion Assessment Method for the Intensive Care Unit (CAM-ICU)**

**CAM-ICU**

- The previous table outlined the components of the CAM-ICU bedside assessment.
- This assessment is very user friendly and easy to implement.
- The patient can participate in this assessment as long as they are able to squeeze a hand and nod their head.
- The Vanderbilt University has an amazing website on delirium with all the resources a practitioner would need to implement a bedside program in their hospital.
- The site is www.ICUDelirium.org

**TREATMENT OF ICU DELIRIUM**

- There is little known about the treatment of ICU delirium.
- There is currently not an FDA approved treatment for delirium.
- Based on clinical outcomes Haldol has been the drug of choice after a patient exhibits signs of delirium. However, there is little evidence to support this.
- Research shows that using alternative drugs for sedation may help. Such as Precedex in place of Propofol.

**ABCDEF BUNDLE**

- **Awakening and Breathing** Trial Coordination (the Wake Up and Breathe Protocol)
- **Choice of Sedative**
- **Delirium Detection**
- **Early Progressive Mobility and Exercise**
- **Family Engagement**

www.ICUDelirium.org

Improve patient care and reduce the impact of modifiable delirium risk factors.
Ensure that your unit has a policy for delirium assessment that includes a minimum of a once per shift assessment for all critically ill patients, utilizing a validated tool (e.g., CAM-ICU or ICDSC). Perform, document, and communicate delirium assessments at least once per shift.

Evaluate patients for potential risk factors for delirium, including a review of medications.

Consider strategies to decrease benzodiazepine usage, including titration strategies (e.g., sedation scale, targeted sedation protocol, and daily weaning trials) or an alternative sedative (e.g., dexmedetomidine or propofol).

Develop a protocol that incorporates early progressive mobility and exercise for all critically ill patients.

Evaluate patients for causes of delirium—including medications (especially benzodiazepines)—using the THINK pneumonic.

**Delirium: Epidemiology and Short-Term Outcomes**

- **Prevalence**
  - 50% to 80% of mechanically ventilated patients
  - 20% to 50% of lower severity patients

- **Associated outcomes**
  - Prolonged hospitalization
  - Increased mortality
  - Increased cost

**Delirium: Long-Term Outcomes**

- **Mortality**
  - Each day of delirium in the ICU increases the hazard of 1-year mortality by 10%.

- **Cognitive Impairment**
  - ICU delirium is an independent risk factor for long-term cognitive impairment. 34% with scores similar to moderate TBI.

**Causes of Delirium: THINK about Dr DRE**

- **T** = Toxic Situations
  - (CHF, shock, dehydration, delirigenic meds, new organ failure)

- **H** = Hypoxemia/hypocarbia

- **I** = Infection/Inflammation, immobility

- **N** = Non-pharmacological interventions

- **K** = Kidney and other electrolyte abnormalities

- **D** = Diseases
  - (Sepsis, CHF)

- **R** = Removal of Drugs
  - (Stop Benzodiazepines, antihistamines, opioids used for sedation)

- **E** = Environment
  - Remove restraints
  - Provide orientation items (clocks, calendars)
  - Reduce isolation/noise
  - Restore day/night light patterns
  - Mobility/promote sleep

**Postop Impaired Cognition**

- Probably due to CPB – OPCAB less impaired cognition
- Mild difficulty with memory, problem solving, attention, and ability to learn
- Typically symptoms improve in 1 – 2 months but may take up to 6 months

Sources:
- Source: AACN Practice Alert
<table>
<thead>
<tr>
<th>Stroke</th>
<th>Post Pump Hepatic Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Embolic/ischemic</td>
<td>• Hepatic trauma occurs</td>
</tr>
<tr>
<td>– Carotid disease</td>
<td>– Long pump runs</td>
</tr>
<tr>
<td>– Aortic calcification</td>
<td>– Inadvertently placing CTs</td>
</tr>
<tr>
<td>• Hemorrhagic -- rare</td>
<td>– Manipulation of venous cannulas</td>
</tr>
<tr>
<td></td>
<td>• Hyperbilirubinemia is maximal at POD #2</td>
</tr>
<tr>
<td></td>
<td>• Watch for post op jaundice</td>
</tr>
<tr>
<td></td>
<td>• Hepatic trauma can rapidly lead to</td>
</tr>
<tr>
<td></td>
<td>unexplained hypovolemia and require</td>
</tr>
<tr>
<td></td>
<td>immediate abdominal exploration</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Renal</th>
<th>Acute Renal Failure/insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Predominant cause of ARF is Acute tubular</td>
</tr>
<tr>
<td></td>
<td>necrosis (ATN)</td>
</tr>
<tr>
<td></td>
<td>• Most common causes</td>
</tr>
<tr>
<td></td>
<td>– Prolonged hypotension and hypovolemic shock</td>
</tr>
<tr>
<td></td>
<td>• Oliguria or anuria</td>
</tr>
<tr>
<td></td>
<td>• Elevated BUN and creatinine</td>
</tr>
<tr>
<td></td>
<td>• Isothenuria (urine osmolality = plasma</td>
</tr>
<tr>
<td></td>
<td>osmolality)</td>
</tr>
<tr>
<td></td>
<td>• May need CRRT or hemodialysis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protecting the Kidneys</th>
<th>Life Threatening Electrolyte Imbalances</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Renal perfusion must be maintained at all times</td>
<td></td>
</tr>
<tr>
<td>• U/O should be at least 0.5ml/kg/hr</td>
<td></td>
</tr>
<tr>
<td>– Transient oligura may be present in the 1st 12 hours. Usually responds to volume or low dose dopamine</td>
<td></td>
</tr>
<tr>
<td>• Essential to have adequate CO and BP</td>
<td></td>
</tr>
<tr>
<td>• Need to replace the loss of the typical diuresising of 200 – 300 ml/hr post op</td>
<td></td>
</tr>
<tr>
<td>• Potassium</td>
<td></td>
</tr>
<tr>
<td>• Magnesium</td>
<td></td>
</tr>
<tr>
<td>• Sodium</td>
<td></td>
</tr>
<tr>
<td>• Phosphorus</td>
<td></td>
</tr>
<tr>
<td>• Calcium</td>
<td></td>
</tr>
</tbody>
</table>
### Potassium 3.5 – 5.5 mEq/L

**Hypokalemia** — due to fluid shift from CPB, diuresis, correction of hyperglycemia with insulin
- Depressed ST segments
- Flat or inverted T waves
- Presence of U waves
- Dysrhythmias — PVCs, VT
- Accompanied by metabolic alkalosis and hypomagnesemia
- Treat with potassium replacements

**Hyperkalemia** — due to over replacement, metabolic acidosis, ARF
- Tall, peaked tented T waves
- Flattened or absent p waves
- Widening QRS
- Asystole
- Nausea
- Muscle weakness
- Treat with kayexalate, insulin + glucose, sodium bicarb

### Magnesium 1.5 – 2.5 mEq/L

**Hypomagnesemia** due to fluid shifts from CPB and diuresis
- Flat or inverted T waves
- ST segment depression
- Prolonged QT interval
- PVCs, SVT, VT, Torsades
- Chvostek’s sign
- Trousseau’s signs
- Hyperreflexia

**Hypomagnesemia** due to depressed renal function
- Peaked T waves
- Shortened QT interval
- Prolonged PR & QRS intervals
- Bradycardia, Heart Blocks
- Hyporelexia
- Lethargy – coma
- Treat with insulin + glucose

### Sodium 135 – 146 mEq/L

**Hyponatremia:** Fluid excess — Sodium Deficit
- Causes: Thiazide diuretics, vomiting, diarrhea, CHF, NSAIDs
- Mental changes – confusion to coma, muscle weakness, irritability

**Hypernatremia:** Fluid Deficit — Sodium Excess
- Rare
- Associated with hyperventilation

### Phosphorus 2.5 – 4.5 mg/dl

**Hypophosphatemia** due to increased renal elimination from resp alkalosis or from postop stress
- Paresthesias
- Severe, profound and progressive muscle weakness
- Tremors
- Muscle pain/tenderness
- Lethargy, confusion, anxiety and apprehension
- If untreated: hypoxia, bradycardia, hypotension
- Resp rates ↓ as Phos ↓
- Treat with IV phosphorus

**Hyperphosphatemia** due to decreased renal function
- Relationship between resp acidosis and hyperphosphatemia
  - ↑ CO2 leads to ↑ phosphorus
  - Resp Acidosis causes phos to move from IC to EC fluid
- Metabolic acidosis is also assoc with hyperphosphatemia
- Altered mental state, delirium, seizures, paresthesia
- Positive Trousseau’s and Chvostek’s signs
- Hypotension, cardiac arrhythmias
- Watch for heart block or flaccid paralysis with infusion of phosphorus as these are signs of rebound hyperphosphatemia. Stop infusion immediately!

### Hypophosphatemia

- Causes myocardiac dysfuntion
  - Check phosphate level if increasing inotropes to keep hemodynamics stable
- Prolonged mechanical ventilation
  - From muscle weakness

### Hypophosphatemia

**Hypophosphatemia**
- Causes myocardiac dysfuntion
  - Check phosphate level if increasing inotropes to keep hemodynamics stable
- Prolonged mechanical ventilation
  - From muscle weakness
The Chvostek sign (Weiss sign)

- low magnesium - low calcium - high phosphorus

- An abnormal reaction to the stimulation of the facial nerve.
- When the facial nerve is tapped at the angle of the jaw (i.e. masseter muscle), the facial muscles on the same side of the face will contract momentarily
- Typically a twitch of the nose or lips

Trousseau’s signs

- low magnesium - low calcium - high phosphorus

- To elicit the sign, a blood pressure cuff is placed around the arm and inflated to a pressure greater than the SBP and held in place for 3 minutes.
- With the brachial artery occluded, the patient’s hypocalcemia and subsequent neuromuscular irritability will induce spasm of the muscles of the hand and forearm.
- The wrist and metacarpophalangeal joints flex, the DIP and PIP joints extend, and the fingers adduct

Calcium 8.5 – 10.5 mg/dl

- Hypocalcemia due to CBP, hemodilution, low CO, or admin prBC (citrate binds to Ca++)
- Need ionized calcium to confirm hypocalcemia
- ↓CO, ↓contractility, hypotension
- Prolonged QT interval
- Low calcium and normal ionized calcium usually asymptomatic
- Numbness/tingling of fingers and toes
- Muscle cramps, spasms/tremors, twitching
- Abd & intestinal cramping = hyperactive BS

- Treat with 10% calcium gluconate

Electrolytes in a Nutshell

- Low Phosphorus
  - Associated with resp alkalosis
  - Prolonged mechanical ventilation
  - Myocardial dysfunction
- High Phosphorus
  - Associated with resp acidosis, Positive Trousseau's and Chvostek's signs
  - Watch for heart block or flaccid paralysis with infusion of phosphorus as these are signs of rebound hyperphosphatemia. Stop infusion immediately!
- Low Calcium
  - ↓CO, ↓contractility, hypotension, Prolonged QT interval, hyperactive BS
  - Positive Trousseau's and Chvostek's signs
- High Calcium
  - Shortened QT interval, depressed T wave. Mental changes

Electrolytes in a Nutshell (2)

- Low Potassium
  - Flattened T waves, u waves, PVCs, V-tach
- High Potassium
  - Tall, peaked T waves, absent p waves, wide QRS, ventricular escape/Asystole
- Low Magnesium (Potassium’s little brother)
  - Flattened T waves, prolonged QT, PVCs, V Tach,
  - Positive Trousseau's and Chvostek's
- High Magnesium
  - Peaked T waves, bradycardia, hyporelexia
- Low Sodium
  - Fluid excess – Sodium Deficit/mental changes/Confusion

Any Questions?

- Carol Rauen – has good handouts on electrolytes