Hematology
GI, Renal, Neuro & Electrolyte Issues In Cardiac Patients

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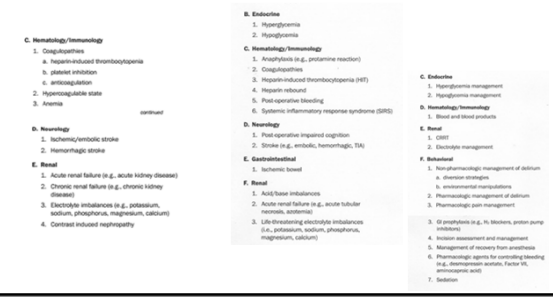
Test Blue print

CMC

CSC



Hematology Review

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

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

Severe

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

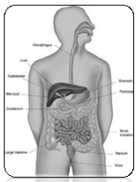
Protamine Reaction Risk Factors

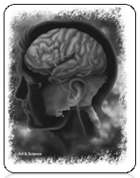
- Allergy to fish
- Use of NPH insulin

Protomine 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
- Combativeness
- Agitation

May be related to the development of microemboli, cerebral edema secondary to the inflammatory response, inadequate temperature regulation, or cerebral hypoperfusion

Delirium

AACN PracticeAlert™

Delirium Assessment and Management

Scope and Impact of the Problem

Delirium is an acute change in consciousness that is accompanied by inattention and either a change in cognition or perceptual disturbance.[1] Patients can have hyperactive delirium (agitation, restlessness, attempting to remove catheters, and/or emotional lability), hypoactive delirium (flat effect, withdrawal, apathy, lethargy, and/or decreased responsiveness), or a combination of both.[1] Delirium affects up to 80% of ICU patients, and it is estimated that ICU costs associated with delirium equal between $4 billion and $16 billion annually in the US.[2-4]

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 treament

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 Lanz, RN, BSN, CCRN
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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-therapuetic 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.

TYPES OF DELIRIUM

There are three types of delirium:

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

HYPOACTIVE DELIRIUM

- 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

HYPERACTIVE DELIRIUM

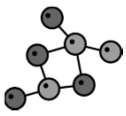
- This is the most recognized and the most difficult type of delirium to care for.
- It is much less common than the other two forms.
- Sings and Symptoms include:
  - Agitation
  - Increased non-purposeful activity
  - Confusion
  - Hallucinations and delusions
  - Paranoia
  - Aggressive and combative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

MIXED DELIRIUM

- 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**.

PATHOPHYSIOLOGY OF DELIRIUM

- 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.



CAUSATIVE FACTORS IN THE DEVELOPMENT OF DELIRIUM

- 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.

PHARMACOLOGICAL FACTORS

- Medications that have anticholinergic properties and other psychoactive medications are among the most likely to cause delirium.
- The next slide lists some commonly used home medications and ICU drugs that can lead to the development of delirium.

MEDICATIONS ASSOCIATED WITH DELIRIUM

- *Opioid Analgesics:*
  Fentanyl
  Morphine
  Demerol
- *Benzodiazepines:*
  Ativan
  Versed
  Valium
- *Antidepressants:*
  Pamelor
  Elavil
- *Antihistamines:*
  Benadryl
- *General Anesthetics:*
  Ketamine
  Inhaled anesthetics

- *Corticosteriods:*
  Solu- medrol
  Prednisone
  Decadron
- *H2- Blockers:*
  Tagament
  Zantac
  Pepcid
- *Antiarrhythmics:*
  Norpace
  Digoxin
- *Beta Blockers*
  Inderal
  Lopressor
- *Cardiovascular:*
  Diuretics

"THINK" MNEMONIC

To Identify Causes

- **T**oxic situations
  - CHF, shock, dehydration
  - Deliriogenic meds (tight titration of sedatives)
  - New organ failure (eg, liver, kidney)
- **H**ypoxemia
- **I**nfection/sepsis (nosocomial)
- **I**mmobilization
- **N**onpharmacologic interventions (Are these being neglected?)
  - Hearing aids, glasses, sleep protocols, music, noise control, ambulation
- **K**+ or electrolyte problems

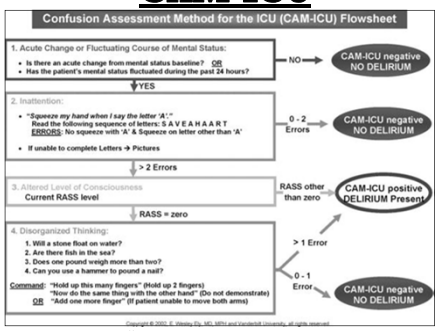
Source: Society of Critical Care Medicine & AACN Practice Alert

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



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 s/s 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.

Daytime interventions	Nighttime Interventions
☐ lights on/blinds open	☐ minimize noise
☐ up to chair	☐ bedtime routine
☐ adequate hydration	☐ limit TV hours
☐ hearing aids in place	☐ normal sleep/wake cycle
☐ plan to ambulate BID	☐ dim lights
☐ glasses on	
☐ orient to date/time	

ABCDEF BUNDLE

- **A**wakening and
- **B**reathing Trial Coordination (the Wake Up and Breathe Protocol)
- **C**hoice of Sedative
- **D**elirium Detection
- **E**arly Progressive Mobility and Exercise
- **F**amily Engagement

www.ICUdelirium.org

Improve patient care and reduce the impact of modifiable delirium risk factors.

ACTIONS FOR NURSING PRACTICE

- 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 (ie, 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 (eg, sedation scale, targeted sedation protocols, and daily awakening trials) or an alternative sedative (eg, 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.

Source: AACN Practice Alert

Creating a Standard Language

- Acute confusional state
- Confusion
- Acute brain syndrome
- Altered mental status
- Toxic or metabolic encephalopathy
- Sundowning

→ DELIRIUM

http://www.iculiberation.org/SiteCollectionDocuments/ICU-Liberation-ABCDEF-Delirium-Bundle-Implementation-Assess-Prevent-Manage.pdf

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

Ely E **JAMA.** 2001;286:2703-2710.
Dubois M. **Intensive Care Med.** 2001;27:1297-1304.
Ely E **JAMA.** 2003;289:2983-2991.
Ely E **JAMA.** 2004;291:1753-1762.
Thomason JW. **Crit Care**. 2005;9:R375-81.
Milbrandt EB **Crit Care Med.** 2004; 32:955-962.

Delirium: Long-Term Outcomes

- **Mortality**
  - Each day of delirium in the ICU increases the hazard of 1-year mortality by 10% [1]
- **Cognitive Impairment**
  - ICU delirium is an independent risk factor for long-term cognitive impairment [2,3]
    34% with scores similar to moderate TBI
    24% with scores similar to mild Alzheimer disease

[1]Pisani MA **Am J Respir Crit Care Med.** 2009;180:1092-1097.
[2]Girard T **Crit Care Med** .2010;38:1513-1520.
[3]Pandharipande PP **N Engl J Med.** 2013;369(14):1306-1316.

Causes of Delirium: THINK about Dr DRE

THINK	Dr DRE
T = Toxic Situations (CHF, shock, dehydration, deliriogenic meds, new organ failure)	D = Diseases (Sepsis, CHF)
H = Hypoxemia/hypercarbia	R = Removal of Drugs (Stop Benzodiazepines, antihistamines, opiods used for sedation)
I = Infection/inflammation, **immobility**	E = Environment • Remove restraints • Provide orientation items (clocks, calendars) • Reduce isolation/noise • Restore day/night light patterns • Mobility/promote sleep
N = Non-pharmacological interventions	
K = K+ and other electrolyte abnormalities	

Mnemonics from ICUDelirium.org

Brummel, et al. (2013) Implementing Delirium Screening in the ICU: Secrets to Success. CCMJournal Vol 41

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

Stroke

- Embolic/ischemic
  - Carotid disease
  - Aortic calcification
- Hemorrhagic -- rare

Post Pump Hepatic Failure

- Hepatic trauma occurs
  - Long pump runs
  - Inadvertently placing CTs
  - Manipulation of venous cannulas
- Hyperbilirubinemia is maximal at POD #2
- Watch for post op jaundice
- Hepatic trauma can rapidly lead to unexplained hypovolemia and require immediate abdominal exploration

Renal

Acute Renal Failure/insufficiency

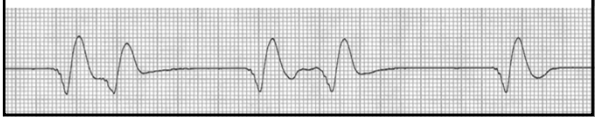
- Predominant cause of ARF is Acute tubular necrosis (ATN)
- Most common causes
  - Prolonged hypotension and hypovolemic shock
- Oliguria or anuria
- Elevated BUN and creatinine
- Isothenuria (urine osmolality = plasma osmolality)
- May need CRRT or hemodialysis

Protecting the Kidneys

- Renal perfusion must be maintained at all times
- U/O should be at least 0.5ml/kg/hr
  - Transient oligura may be present in the 1st 12 hours. Usually responds to volume or low dose dopamine
- Essential to have adequate CO and BP
- Need to replace the loss of the typical diuresising of 200 – 300 ml/hr post op

Life Threatening Electrolyte Imbalances

- Potassium
- Magnesium
- Sodium
- Phosphorus
- Calcium



Potassium 3.5 – 5.5 mEq/L

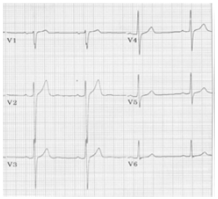
Hypokalemia – due to fluid shift from CBP, diuresis, correction of hyperglycemia with insulin

- 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

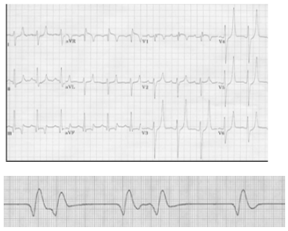
Hyperkalemia– due to over replacement, metabolic acidosis, ARF

- Tall, peaked tented T waves
- Flattened or absent p waves
- Widening QRS
- Asystole
- Nausea
- Muscle weakness
- Treat with kayexalate, insulin +glucose, sodium bicarb

Hypokalemia



Hyperkalemia



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

Hypermagnesemia due to deparessed renal function

- Peaked T waves
- Shortened QT interval
- Prolonged PR & QRS intervals
- Bradycardia, Heart Blocks
- Hyporelexia
- Letharthy – 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 phophorus

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
- Hyptension, 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 phosophate 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

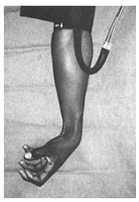
- An abnormal reaction to the stimulation of the facial nerve.
- When the facial nerve is tapped at the angle of the jaw (i.e. masseter muscle), the facial muscles on the same side of the face will contract momentarily
- Typically a twitch of the nose or lips

- Positive Chvostek signs

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 DIPand PIP joints extend, and the fingers adduct



- Positive Trousseau's Sign

Calcium 8.5 – 10.5 mg/dl

Hypocalcaemia due to CBP, hemodilution, low CO, or admin pRBC (citrate binds to CA++)

- Need ionized calcium to confirm hypocalcaemia
- ↓CO, ↓ contractility, hypotension
- Prolonged QT interval
- Low calcium and normal ionized calcium usually asymptomatic
- Numbeness/tingling of fingers and toes
- Muscle cramps, spasms/tremors, twitching
- Abd & intestinal cramping = hyperactive BS
- Treat with 10% calcium gluconate

Hypercalcemia due to ↑ intestinal absorption, ↑reabsorption, & ↓ elimination

- Altered mental status, fatigue, weakness, lethargy, anorexia, nausea, vomiting, constipation, decreased renal function or ARF
- Shortened QT interval, depressed T wave
- Treat with calcitonin

Electrolytes in a Nutshell

- 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

Electrolytes in a Nutshell (2)

- 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

Any Questions?

- Carol Rauen – has good handouts on electrolytes