

Join the Club! Strategies for Successful Journal Club Development

Wendy Anne Cook

Level: Beginner

CONTENT DESCRIPTION

Participation in an active, well managed nursing journal club is a rewarding and enjoyable professional experience. Journal clubs promote nursing collegiality and offer participants an opportunity for scholarly discussion, as well as development and improvement of presentation and literature review skills. Journal clubs foster a culture of research appreciation and enhance ability to implement evidence based practice, often identifying new issues pertinent to individual practice. Strategies to develop a successful journal club include identifying purpose and goals, establishing leadership and administrative responsibilities, creating a discussion format, and promoting participation. Tactics for improving existing journal clubs and addressing common problems are essential for prolonged sustainment.

At the completion of this session participants will have a clear understanding of the benefits of journal club participation, will possess knowledge of strategies for establishing a new journal club, and an awareness of methods to improve an existing journal club and address common problems and challenges.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Discuss professional benefits of journal club participation
2. Discuss strategies to develop a successful journal club
3. Discuss strategies Improve an existing journal club and address common problems and challenges

SUMMARY OF KEY POINTS

- I. Professional benefits of journal clubs
 - A. Enhance critical appraisal skills and research interpretation skills
 - B. Enhance familiarity with current literature
 - C. Enhance appreciation and enthusiasm for research
 - D. Promotes nursing collegiality
 - E. Development of presentation and communication skills
 - F. Maintain continuing education requirements
 - G. Potential improved patient care
- II. Journal Club Formats
 - A. Unit-based
 - B. Hospital-based
 - C. Multidisciplinary
 - D. Online/Internet based
 - E. Roving journal club
 - F. Multi-institutional
 - G. Formal vs. informal

III. Journal Club Development

- A. Identify purpose
- B. Identify goals
- C. Identify format to meet purpose and goals
- D. Establish support from nursing leadership and nurse managers
- E. Determine structure
- F. Identify meeting length, schedule, location, frequency
- G. Determine participation requirements
- H. Identify leader/administrator and administrative details
- I. Determine discussion leader requirements
- J. Establish ground rules/policies
- K. Consider formal charter
- L. Consider multiple journal clubs to meet multiple needs

IV. Journal Club Improvement

- A. Develop standard discussion template for consistency of presentations and ease of preparation
- B. Develop discussion questions in advance and distribute to participants for preparation
- C. Incorporate short sessions regarding elements of literature review and study design as part of the meeting, especially during the early meetings
- D. Survey current and potential participants
 1. Recommendations for topics
 2. Ideal meeting time
 3. Barriers to participation
- E. Participants evaluate each meeting
- F. Journal club activities
 1. Writing letter to the editor- recent articles
 2. Contacting author to provide feedback
 3. Consider debate team format
 4. Consider replicating an interesting study
 5. Document cost savings related to the journal club
 6. Consider a brief discussion of related evidence in addition to the featured article
- G. Offer continuing education units to document participation

V. Common Problems and Challenges

- A. Ongoing Sustainment
- B. Identifying discussion leaders
- C. Supporting attendance/ability to leave bedside
 1. Maximize attendance without impacting patient care
 2. Make it as easy as possible to encourage attendance, encourage participants to bring lunch, provide food/snacks

3. Considering offering multiple meeting sessions for same article
4. Nurse managers encourage participation
5. Consider holding meetings following staff meetings for unit-based journal clubs
6. Record meetings for staff unable to attend

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Journey to Iraq and Afghanistan

Paul R. Dickinson
Wesley H. Pierce

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this class is to give nurses an understanding of being deployed as a military nurse to a combat environment. This includes the pre-deployment & train-up phase, actual experiences while being deployed, and the re-deployment phase. These experiences are based on two nurses who each spent a year deployed as the head nurse of an ICU; one to Afghanistan, and one to Iraq.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

- I. Gain insight and knowledge of pre-deployment preparation experiences and requirements

SUMMARY OF KEY POINTS

- I. Gain insight and knowledge of pre-deployment preparation experiences and requirements.
 - A. Initial Pre-Deployment training at current duty station (Briefings, updates, physicals, immunizations, labs, etc.).
 - B. Pre-Deployment & training (about a month long - weekends & holidays off) with unit we'd be deploying with (ABLS, TNCC, more lectures & briefings, Lessons learned "Do's & Don'ts", etc. equipment issue, PT and PT tests "Hanging Out" learning who you'll be working with & getting to know them, weapons issue and training, what to take and not to take (have stuff mailed to you later).
- II. Gain insight and knowledge of actually deploying to another country and getting settled in.
 - A. The flight to Theater (Stop-Over's, comfort on plane [with weapons]) layovers, arriving to destination point.
 - B. Getting settled in theater (Living arrangements, Food, Weapon, body armor [always with you or within reach], entertainment, mail/phone/computer access).
 - C. Getting to work ("hand-off", work schedules, patient load & types of patients seen, taking leave, experiences gained)
- III. Describe types of patients and wounds received in theater, and patient flow through a Combat Support Hospital system of operations.
 - A. Combat trauma patients (usually young males with combat trauma (air-evac'd in) & with non-battle injuries.

- B. View video "Another kind of battle" (approx. 20 min.) and view slides from Afghanistan (approx. 15 min.).
 - C. Discuss: Cultural barriers, interpreters, language, Canadian nurses, hospital layout, challenging patients, (etc.), skill levels, equipment challenges, supply challenges.
 - D. After viewing the video: Discuss what you saw, "is it what you expected?" Can you picture yourself there? "How can you explain what you saw "over there" when you get back home"?
- IV. Gain understanding of the post-deployment process.
 - A. The trip back "home" (People left theater in stages; Some left behind briefly to make sure new unit was running smoothly).
 - B. Customs (They're strict; stuff is searched - specific on what you can bring back to the states (no ammo, non-issued weapons, antiques, explosives, Cuban cigars, bongos, camel saddles).
 - C. Flight back home (Uneventful with stop-over's [i.e.: Ireland]).
 - D. "Back in the USA!" (Family waiting at tarmac for some brief ceremony, turn in weapons, get rooms with families, no driving for 24 hours, No ETOH.)
 - E. SRP, Post deployment health assessments and screens done before, during, and after deployment, classes, equipment turn-in.
 - F. Heading home (Saying good-bye to friends, drive safely...).
 - G. Really back home (Post-deployment leave, returning back to work).
 - H. Looking back now that we've had some time to reflect on: experiences gained, memories, meaning of it all, loss, comparing "here" to "there".
 - I. Protect your nursing license (Things you did independently there you can't do here).
 - J. Would you go back?

Keeping the Balance: Achieving Desired Outcomes With CRRT

Leslie Swadener-Culpepper

Level: Intermediate

CONTENT DESCRIPTION

This session is designed for the experienced ICU RN performing bedside CRRT for critically ill patients. Discussion will focus on maintaining and evaluating intravascular volume as well as promoting appropriate electrolyte balance. The role of replacement and dialysate solutions in achieving appropriate clearance will also be discussed. A portion of the lecture will cover CRRT as an intervention for Acute Renal Failure in Sepsis, and new uses for CHF will be explored. Finally, the role of anticoagulation methods will be reviewed.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Assess and intervene for the CRRT patient experiencing intravascular volume and electrolyte imbalance issues.
2. Describe appropriate changes to dialysate and replacement fluid admixtures to achieve appropriate patient chemistry values.
3. Discuss potential uses for CRRT in Sepsis and CHF

SUMMARY OF KEY POINTS

- I. Today's CRRT
 - A. The basic components of CRRT therapy
 - B. The REAL differences between CRRT therapies
 1. SCUF
 2. CVVH
 3. CVVHD
 4. CVVHDF
 - C. The role of Replacement and Dialysate Solutions
 - D. Blood flow rates
- II. Caring for the patient... beyond the machine!
 - A. Critical patient assessment parameters
 1. Hemodynamics / BP / Cardiac Output
 - a. Cardiac Function
 - b. Vascular Tone
 - c. Determining Intravascular Volume
 - d. Retaining / replacing intravascular Volume
 2. Electrolyte adjustment
 - a. The role of "replacement" solutions
 - b. Dialysate composition and rates
 - c. Lab values
 - B. CRRT in treatment of other disease processes
 1. Sepsis
 - a. Septic Mediators and SIRS response
 - b. Avoiding hemodynamic fluctuations in organ dysfunction.
 2. CHF
 - C. Anticoagulation methods
 1. Heparin based protocols
 2. Citrate based protocols
- III. CRRT outcomes
 - A. Early vs Late
 1. The Futility debate.

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Keys to an Effective Board: Starting off on the Right Foot

Mary Bylone

Level: New and experienced chapter leaders

CONTENT DESCRIPTION

Every chapter leader wants to start the year off right. Why doesn't this happen? Sometimes, expectations are not articulated. Members volunteer for board positions without really understanding what it entails, or their role is not well defined. Sometimes meetings fall short of getting the business done, yet go on for hours. This course is targeted for the chapter leader who is looking for direction on setting clear expectations for board members, establishing healthy meeting structure, utilizing the strategic plan to evaluate chapter progress and setting goals.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Write a job description for the board positions
2. Define appropriate meeting norms
3. Identify required reports

SUMMARY OF KEY POINTS

- I. It starts with the transition meeting
- II. Officer transition - key points to cover
- III. Written Job Descriptions for Board Positions
- IV. Setting up a timeline for the year

- V. Ideas about Budgets
- VI. How to run the meeting
 - A. Setting agendas
 - B. The importance of meeting minutes
 - C. Keeping to a time limit
 - D. Communication norms for meetings
 - E. What happens when there is conflict - is a vote really necessary?
- VII. Financial Management
- VIII. How many committees do you need?
- IX. What should you be doing at every meeting?
- X. What should you be doing at least quarterly?
- XI. What needs to be done annually?
- XII. Nomination/election items
- XIII. Audits
- XIV. Planning officer transition
- XV. Evaluate board's work for the previous year

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Kids Will Eat Anything! Case Studies In Pediatric Poisoning

Maureen Madden

Level: Beginner

CONTENT DESCRIPTION

Over one million cases of exposure to toxins involving children younger than 6 years of age occur annually, making poisoning a major and persistent cause of injury-related morbidity in children in the United States. There have been significant advances made in the prevention and treatment of pediatric poisonings over the past several decades. This session will discuss evolving trends in pediatric poisoning by focusing on epidemiological, prevention, management principles and treatment modalities. The early recognition and management of pediatric poisonings will help to improve patient outcomes and help to decrease the incidence of preventable injuries. A discussion about the principles of management, the identification of signs and symptoms that comprise a toxidrome and treatment modalities will allow the participant to develop a rational approach to the patient with a known or unknown ingestion. The individuals attending this session must possess sound decision making skills and be able to apply critical thinking. The participant must have prior knowledge of critical care in order to identify abnormal versus normal clinical states and apply critical thinking to identify appropriate management and treatment. This session is not exclusively for pediatric critical care nurses but the focus will be on principles relevant to the pediatric population. At the conclusion of this session, the participant will have an understanding of the magnitude of the problem of pediatric poisonings and an appreciation for the changes in management and treatment of pediatric poisonings.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Identify the epidemiological factors for pediatric poisoning including the toxic agents and risk factors by age.
2. Describe the principles of management related to a suspected poisoning.
3. Identify toxidromes and apply them to clinical scenarios.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Principles of Pediatric Toxicology
 - A. Definition of Toxicology
 - B. Cornerstone of Treatment
 - C. Goal of Treatment
 - D. Poison Control Centers

- E. TESS Annual Report- 2005 Data
- F. Common Agents
- G. Toxic Agents
- H. Developmental Risk Factors
 1. Toddlers
 2. School Age
 3. Adolescents
- III. Principles of Management
 - A. Primary Management Principles- Stabilization
 1. Airway
 2. Breathing
 3. Circulation
 4. Disability
 - B. General Management Principles- Secondary Phase
 1. History of Poisoning
 2. Decontamination
 3. Drugs/Antidotes
- IV. Toxidromes
 - A. Definition
 - B. Categories
 1. Sympathomimetic
 - a. Symptoms
 - b. Causative Agent
 2. Theophylline
 - a. Symptoms
 - b. Causative Agent
 3. Sedative/Hypnotic
 - a. Symptoms
 - b. Causative Agents
 4. Opiate
 - a. Symptoms
 - b. Causative Agents
 5. Anticholinergic
 - a. Symptoms
 - b. Causative Agents
 6. Cholinergic or SLUDGE
 - a. Symptoms
 - b. Causative Agents
- V. Management
 - A. Laboratory Studies
 1. Electrolytes
 - a. Metabolic Acidosis or MUDPILES
 - M-methanol
 - U-uremia
 - D-diabetic ketoacidosis
 - P-paraldehyde and phenformin

- I-iron, INH
- L-lactic acidosis- hypoxia, shock, CO, cyanide
- E-ethanol, Ethylene glycol
- S-salicylates
- 2. ABG
- 3. Urinalysis
- 4. EKG
- 5. Toxicology Screening- indications and utility
 - a. Urine
 - b. Serum
 - c. Quantitative screening
- 6. Drug levels
 - a. Radiologic Studies
 - (1) Radio-opaque substances or CHIPS
 - C-chloral Hydrate, cocaine packets
 - H-heavy Metals- lead, arsenic, mercury
 - I-iron
 - P-phenothiazines
 - S-slow- release, enteric coated tablets
 - b. Gastrointestinal Decontamination
 - (1) Decreasing Absorption
 - (a) Gastric Emptying- Gastric Lavage
 - (b) Forced Emesis- Syrup of Ipecac
 - (c) Activated charcoal- ineffective with CHAMP
 - i. C-camphor and caustics
 - ii. H-halogenated hydrocarbons
 - iii. A-aromatic hydrocarbons and alcohols
 - iv. M-metal containing compounds
 - v. P-pesticides

- (2) Altering Metabolism
- (3) Increasing Elimination
 - (a) Multiple dosed charcoal
 - (b) Forced Diuresis
 - (c) Alkalinization or acidification of urine

VI. Case Studies

VII. Conclusions

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Liver Failure, Comorbidity, and Managing Portal Hypertension!

Richard Arbour

Level: Expert

CONTENT DESCRIPTION

Primary purpose is analyzing physiological causes and consequences of liver failure in context of effects on hepatic blood flow, bleeding risk and incidence of encephalopathy. Secondary purpose is correlating multi-system consequences of liver cirrhosis and portal hypertension with management strategies. These strategies include aggressive crystalloid and blood product resuscitation as well as invasive measures including vascular shunting procedures, sclerotherapy and banding of esophageal varices to control hemorrhage. Session scope includes clinical, laboratory and radiographic findings of cirrhosis and hepatic failure. Multi-system consequences of hepatic failure including hepatopulmonary syndrome and encephalopathic states will be explored by case study. Lactulose administration for hepatic encephalopathy is reviewed. Interventions for massive upper gastrointestinal hemorrhage including aggressive resuscitation with blood/blood products, colloid and crystalloid are examined. Managing hematological complications including coagulopathies due to impaired liver synthesis of clotting factors with interventions such as plasma volume replacement and blood product resuscitation are analyzed. Drug therapies including octreotide, beta blockade and midrodine are appraised as part of a comprehensive plan of care. Optimal critical care management is appraised as integral to decreasing patient ICU length of stay and providing a successful bridge to orthotopic liver transplantation. The target audience includes clinicians, educators and advanced practitioners managing hepatic dysfunction. Content application optimizes management of systemic consequences of liver dysfunction, improves outcomes, cardiopulmonary stability and decreases ICU length of stay. Participants possessing basic knowledge of systemic consequences of liver failure benefit most from session content. Session contains 50 % pharmacology content.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Analyze physiological consequences of hepatic failure/cirrhosis in context of consequences affecting multiple body systems.
2. Appraise multi-system consequences of portal hypertension and related clinical findings.
3. Analyze plan of care for optimal clinical management of patient with hepatic failure complicated by portal hypertension.

SUMMARY OF KEY POINTS

- I. Introduction-Definition of terms:
 - A. Fulminant hepatic failure: Rapid development of

severe acute liver injury in a patient with previously normal liver function.

1. Impaired synthetic function.
2. Encephalopathy.

B. Etiologies:

1. Viral: Hepatitis
2. Drug/toxin causes: Dose-dependent (Acetaminophen)/adverse drug reactions.

II. Clinical consequences: Toxin exposure.

A. Primary cellular level injury: Biochemical toxicity.

1. Liver metabolism/first-pass exposure.
2. Final fraction metabolized (cytochrome pathways) to toxic, reactive intermediates (NAPQI).
3. NAPQI reacts with cell components, causing widespread cellular level hepatic injury.
4. Oxidative injury:
 - a. Free radical generation.
 - b. Cytokine release.

B. Spread of hepatic injury, cytokine release.

1. Secondary inflammatory response, additional involvement of inflammatory cells.
2. Vasodilated state, decreased vascular resistance, increased vessel capacitance.
3. Inflammatory mediators released from necrotic/injured liver tissue.

C. Clinical consequences:

1. Peak elevations in LFT's, jaundice, hepatic encephalopathy, elevated ammonia levels, severe coagulopathy, bleeding, acidosis, hypoglycemia.
 - a. Renal failure (ATN).
 - b. Final common pathway to fulminant hepatic failure:
 - c. Global/multisystem consequences.

III. Global physiologic consequences: Systemic hemodynamic dysfunction:

- A. Generalized vasodilatation
- B. Increased cardiac output.
 1. Decreased SVR/MAP.
- C. Systemic inflammatory response syndrome.
 1. Cytokine release
 2. Inflammatory response.

IV. Portal hypertension physiology: Common pathway for venous drainage from the gut.

- A. Delivers blood/substrates toward liver for metabolic processing.
- B. Low pressure system joins higher-pressure system post-hepatic artery.
 1. Hepatic vein: Venous drainage from liver.

- C. Portal hypertension: Multiple sources.
 1. Pre-hepatic/presinusoidal: Portal vein thrombosis, splenic vein thrombosis.
 2. Intrahepatic/sinusoidal: Hepatitis, toxin exposures, liver hyperplasia.
 3. Post-hepatic/post-sinusoidal: Cardiac disease, IVC obstruction.
 - a. Measurement: Invasive measurement, wedged hepatic venous pressure.
 - b. Wedged hepatic venous pressure ≥ 12 mmHg \gg increased risk of variceal bleed.
 - (1) Caution in pre-hepatic flow obstruction.
 4. Contributing factors: Interaction between splanchnic vascular bed and circulating factors.
 - a. Role of nitric oxide.
 - b. Vasoconstrictors: Norepinephrine, angiotensin 2, ADH, Endothelin 1.
 - c. Vasodilators: Glucagon, prostaglandins, substance P.
 - d. Mediators mediators: Hypoxemia, adenosine, acidosis,
 - e. Endothelium-derived factors.
 5. Increased hepatic blood inflow: Vasodilated state, hyperdynamic cardiovascular state.
 6. Alterations in hepatic microcirculation, fibrosis, shear stress $\gg \gg$ Decreased hepatic blood outflow.
 7. Total increase in blood volume through gut and portal flow.
- D. Role of food ingestion:
 1. GI hormonal increase: Rapid transient arteriolar dilatation/increased portal blood flow $\gg \gg$ increased risk of variceal bleed.
- V. Global clinical consequences:
 - A. Prolonged hyperdynamic, vasodilated state.
 - B. Volume retention.
 - C. Secondary involvement of other organ systems.
 - D. Collateral vessel formation $\gg \gg$ varices.
 1. Catastrophic hemorrhage.
 - E. Initial clinical management:
 1. Airway management: Intubation/controlled ventilation.
 2. Temporizing: Balloon tamponade: Short-term.
 3. Goal: Direct management of varices.
 - F. Invasive intervention:
 1. Endoscopic band ligation: Direct management of bleed, obliteration of varix.
 2. Sclerotherapy: Injection of sclerosing agent, rapid hemostasis.
 3. Transjugular intrahepatic portosystemic shunt (TIPS).
 - a. Direct stenting between hepatic/portal blood flow.
 - b. Decompression of varices.
 - c. Long-term follow up/adjunctive therapy: Risks.
 - G. Aggressive hemodynamic support/circulating blood volume: crystalloid/blood/blood product resuscitation $\gg \gg$ large-bore access.
 1. Target INR-1.5.
 2. Platelets 75k
 3. Factor VII
- H. Modulate mesenteric, portal and hepatic blood flow.
 1. Vasoactive agents: Somatostatin/octreotide
 2. Coordinate bolus/infusion dosing.
 3. Vasopressin: Limited use re: ischemic risk.
 4. Beta blockade: Decrease cardiac output/portal blood flow.
- VI. Neurologic consequences:
 - A. Hepatic encephalopathy: Reversible impairment of brain function in hepatic failure.
 1. Serum ammonia: Direct neurotoxicity (primary event).
 - a. GI sources: Bleeding, consequent to liver impairment.
 2. Secondary consequences: Neuronal transport/ neurotransmitter dysfunction.
 3. Increased intracellular osmolality in astrocytes: Functional changes.
 - a. Impaired brain energy metabolism/ Neurotransmission:
 - b. Altered blood-brain barrier/brain edema.
- VII. Management of hepatic encephalopathy: Context of overall management priorities.
 - A. Aggressive management of causes.
 1. Reduction of ammonia levels.
 2. GI elimination, increased stool volume.
 3. Lactulose, increased fecal nitrogen elimination.
 4. Oral antibiotics: Rifaximin.
- VIII. Pulmonary consequences: Hepatopulmonary syndrome.
 - A. Clinical triad
 1. Liver disease.
 2. Increased alveolar-arterial gradient.
 3. Intrapulmonary vascular abnormalities (intrapulmonary vascular dilations-IPVD's): Shunt physiology.
 - B. Clinical manifestations:
 1. Dyspnea
 2. Spider nevi: Marker for IPVD's
 3. Refractory hypoxemia, progressive with liver function decline.
 - C. Hyperdynamic cardiovascular state:
 1. Elevated cardiac output.
 2. Decreased SVR/PVR.
 3. Narrow AVO₂ difference.
 4. Compromised tissue oxygen delivery.
 - D. Hypoxemia:
 1. V/Q mismatch
 2. Shunt physiology
 3. Limited oxygen diffusion.
 - E. IPVD: Potential causes.
 1. Circulating pulmonary vasodilators.
 2. Inhibition of normal vasoconstriction.

- IX. Portopulmonary hypertension: Pulmonary hypertension associated with portal hypertension.
- A. Vasoactive agent produced in splanchnic circulation, normally metabolized in liver.
 - B. Vasoactive agent ultimately causing vascular changes in pulmonary vasculature.
 - C. Clinical manifestations:
 1. Elevated mean PA pressure (> 25 mmHg at rest).
 2. PCWP < 15 mmHg/PVR > 120 dynes/sec/cm⁵
 3. Elimination of secondary causes of pulmonary hypertension.
 - D. Management of pulmonary consequences: Intubation/controlled ventilation.
 1. Titration of oxygen, flow rates, ventilation modes.
 2. Vasoactive agents.
 3. Vascular shunting: TIPS procedure.
- X. Hepatorenal syndrome: Acute renal failure consequent to advanced liver disease.
- A. End-stage of progressive process of reduced renal perfusion.
 - B. Pathophysiology:
 1. Splanchnic/generalized vasodilatation.
 2. Increased cardiac output.
 3. Decreased SVR: Refractory to renin-angiotensin and sympathetic nervous system activation.
 4. Reduced glomerular filtration rate (GFR).
 5. Reduced sodium excretion.
 - C. Management:
 1. Vasoactive agents:
 - a. Midrodine: Systemic vasoconstrictor.
 - b. Octreotide: Inhibits endogenous vasodilator release.
 2. Renal replacement therapies:
 - a. Dialysis.
 - b. CRRT.
- XI. Hepatoadrenal syndrome: Adrenal failure in context of acute and critical illness.
- A. Structural issues.
 1. Hypothalamus
 2. Pituitary
 3. Adrenal gland.
 - B. Physiologic stress adaptation: Hypothalamic/pituitary/adrenal axis activation.
 1. Cortisol release basic component of cellular/organ homeostasis.
 2. Basis for stress dosing of glucocorticoids.
 - C. Consequences of acute illness: Decreased cortisol bioavailability and receptor binding.
 - D. Issues within setting of hepatic failure:
 1. Sepsis/hepatic failure common pathophysiology.
 - a. Endotoxin
 - b. Proinflammatory mediators
 - c. Decreased apolipoprotein A.
 - d. Decreased HDL cholesterol.
 - e. High incidence of adrenal failure in hepatic disease.
 - f. Poor hemodynamic reserve.
- XII. Case study:
- A. Mrs. A. B. 46 y/o patient: ICU admission for evaluation/management of liver disease.
 1. Liver disease consequent to hepatitis C/ Progressive decline in hepatic function.
 2. Upper GI hemorrhage: Variceal bleed.
 - B. Clinical issues: ABC's
 1. Airway management.
 2. Hemodynamic instability.
 3. Coagulopathy.
 4. Hemodynamic instability.
 5. Decline in LOC.
 - C. Clinical management: Mechanism-based/goal-directed.
 1. Intubation/controlled ventilation.
 2. Large-bore accesses (multiple): Aggressive volume resuscitation.
 3. IV bolus/infusion octreotide.
 4. Stress dosing: Hydrocortisone.
 5. Esophageal balloon tamponade/temporize.
 6. IR consult: TIPS procedure.
 7. Dialysis catheter insertion:
 - a. Large volume plasmapheresis.
 - b. Replacement with FFP/clotting factors.
 - D. Pulmonary artery catheterization: Low SVR/PVR, high cardiac output state.
 - E. TIPS successful: Portal/systemic pressure gradients (pre/post procedure).
 1. Hemostasis achieved, varices decompressed.
 - F. Progression to stage 4 encephalopathy/probable ICP elevation: Priority listing for transplant.
 1. ICP monitoring >>>>> fiberoptic catheter: Intracranial hypertension.
 2. Urgent head CT: Optimal device placement, no bleeding.
 3. Serial lab monitoring: CBC/electrolytes/Pt/Ptt/INR, fibrinogen/FSP.
 - G. Further measures for ICP management: Mechanism-based.
 1. Drug-induced coma.
 2. Therapeutic hypothermia.
 3. EEG-based monitoring: Barbiturate titration in real-time.
 - a. Effective cerebral metabolic control.
 - H. ICU day 4- "Yellow alert" followed by "red alert": OR for liver transplantation.
 1. Good clinical outcome: Extubation POD 3, ICU discharge POD 5.

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Long QT or Wrong QT?

Barbara Drew

David Pickham

Julie Shinn

Sponsored by Philips

Level: Intermediate

CONTENT DESCRIPTION

Recently published American Heart Association (AHA) Practice Standards for ECG Monitoring in Hospital Settings recommends nurses consider 3 goals of monitoring when they attach a patient to a cardiac monitor: 1.) arrhythmia, 2.) ischemia and, 3.) QT interval monitoring. Currently, few nurses consider this third goal of monitoring. The purpose of this session is to raise awareness of the importance of QT interval monitoring to prevent drug-induced torsades de pointes. Content will include 3 lectures: 1.) "State of the Science" presentation, 2.) report on the QT In Practice (QTIP) Study, and 3.) account of how an Advanced Practice Nurse can improve ECG monitoring practices.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Explain why the QT interval must be corrected for heart rate and what QTc threshold indicates a patient is at risk for developing torsades de pointes.
2. List ECG signs of impending torsades de pointes and nursing responsibilities when these signs are observed.
3. Explain what continuous QT interval monitoring is and how it might improve nurses' adherence to the AHA's Practice Standards.
4. Identify 2 strategies for implementing institution wide changes in practice.
5. Identify strategies for confronting resistance to QT interval monitoring on non-cardiac units.

SUMMARY OF KEY POINTS

- I. State of the Science
 - A. Definition of torsades de pointes (TdP) and ECG characteristics
 - B. Distinguishing TdP from ventricular fibrillation
 - C. QT-prolonging drugs
 - D. Risk factors for developing TdP
 - E. Things to know about QT measurement
 1. Correction for heart rate; QTc >500 ms (0.50 sec) is considered dangerous prolongation that puts the patient at risk for TdP
 2. AHA Practice Standards indications for QT interval monitoring

3. Methods to measure QTc in hospital units; pros/cons for each method

- a. Standard 12-Lead ECG
- b. Manually with hand-held calipers
- c. e-calipers from the central monitor station
- d. new continuous QTc monitoring software

4. ECG signs of impending TdP and nursing responsibilities

II. Continuous QT Interval Monitoring: The QTIP Study

- A. Study aims and hypotheses
- B. Continuous QTc monitoring software; frequency of measurement, alarms, trends
- C. Study setting, sample, research design
- D. Preliminary findings

III. Quality Improvement Related to ECG Monitoring

- A. Building consensus for standardizing practice
- B. The CNS role in advancing initiatives
- C. Planning education
 1. Time constraints
 2. Staff availability
 3. Creative solutions
- D. Lessons learned
 1. Dealing with resistance to change
 2. Moving from "it's another task I have to do" to "it's of value to my patient"

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Looking for Changes, Listening for Sounds: Cardiac Tamponade

Susanne C. Wheeler

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this presentation is to discuss nursing care of the patient with pericarditis, pericardial effusion and cardiac tamponade. Case studies will be used to illustrate the common clinical presentations, physical assessment findings, and electrocardiogram (ECG) changes that often accompany these three conditions.

The pericardial layer of the heart can be affected by many different adverse events and disease pathologies. There are many pathological processes, either directly or indirectly, that can lead to pericardial disease. Although these changes to the pericardium are often benign and self-resolving some patients experience not only discomfort and an altered functional status, but a life threatening consequence such as cardiac tamponade. In fact, approximately 25-30% of large pericardial effusions lead to cardiac tamponade. Through the use of physical assessment skills, knowledge of pathophysiology, and awareness of patients who are at risk, nurses can play a crucial role in the early recognition and subsequent treatment of patients experiencing pericardial alterations. The objectives of this session include: understanding the pathophysiology of pericarditis, pericardial effusion, and cardiac tamponade; identification of patients at risk for pericarditis, pericardial effusion, and cardiac tamponade and the nursing and medical care related to these patients; and the recognition of signs, symptoms, and ECG changes often associated with these conditions. A basic understanding of cardiac anatomy and physiology, basic ECG, and physical assessment skills are the prerequisite knowledge suggested to participants attending this session.

LEARNING OUTCOMES

At the end of the session the participant will be able to:

1. Describe the pathophysiology of pericarditis, pericardial effusion, and cardiac tamponade.
2. Identify patients at risk for the pericardial disease.
3. List nursing and medical care of patients with pericardial disease.
4. Recognize the signs, symptoms, and electrocardiogram changes most commonly associated with pericardial disease.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Anatomy and Physiology of the Pericardium
 - A. Functions of the Pericardium
 - B. Normal Intrapericardial Pressure
- III. Pericardial Disease
 - A. Pericarditis
 1. Definition

- a. Inflammation, thickening, or fibrosis of the pericardium
- b. Pathophysiology
2. Etiologies
3. Types
4. Clinical Signs & Symptoms
5. Assessment
6. Diagnostics
7. Management
8. Nursing Care
9. Case Study
- B. Pericardial Effusion
 1. Definition
 - a. Excess or abnormal fluid, blood, or pus in the pericardial sac or space
 - b. Pathophysiology
 2. Etiologies
 3. Types
 4. Clinical Signs & Symptoms
 5. Assessment
 6. Diagnostics
 7. Management
 8. Nursing Care
 9. Case Study
- C. Pericardial or Cardiac Tamponade
 1. Definition
 - a. Decompensated phase of cardiac compression caused by effusion accumulation and increasing cardiac pressure
 - b. Pathophysiology
 2. Etiologies
 3. Clinical Signs & Symptoms
 4. Hemodynamic Changes
 5. Assessment
 6. Diagnostics
 7. Management
 8. Nursing Care
 9. Case Study

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Looking in 3-D: Dementia, Delirium, and Depression

M. Lynn Rodgers

Level: Beginner

CONTENT DESCRIPTION

Often patients during acute or critical illness exhibit behaviors that are difficult to manage. Are these behaviors part of their existing condition or are they part of their preexisting comorbidities? This session will examine three common causes of behavior disorders seen in hospitalized and non-hospitalized patients: dementia, delirium, and depression. Each of these conditions will be defined and signs and symptoms of each will be reviewed. Assessment tools will be presented that can assist the practitioner in evaluating for these conditions. Management of these disorders will be outlined including nursing care and pharmacological interventions. Specific drugs and drug classifications for dementia, delirium, and depression will be discussed in this session, whose content will be at least 50% pharmacology. Of particular interest will be an analysis of the consequences of discontinuation of drug therapy for these disorders when a patient goes from an outpatient to an acute inpatient environment, i.e., serotonin syndrome and withdrawal phenomenon. This session is appropriate for any novice or expert nurse that cares for patients that exhibit signs of dementia, delirium or depression in any inpatient or outpatient practice setting. Basic knowledge of dementia, delirium, and depression is desired. By the end of this presentation, participants will have a better understanding of these three conditions that are frequently encountered in many patient situations. With an enhanced knowledge of dementia, delirium and depression, recognition, assessment, care, and management of these patients can be improved and complications may be decreased.

LEARNING OUTCOMES

1. Define dementia, delirium, and depression and their contributing factors.

2. Identify impact of dementia, delirium, and depression upon behavior disorders seen in hospitalized and non-hospitalized patients.
3. Discuss nursing and pharmacological interventions that can improve management of dementia, delirium, and depression.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Dementia
 - A. Definition
 - B. Contributing Factors
 - C. Assessment
- III. Delirium
 - A. Definition
 - B. Contributing Factors
 - C. Assessment
- IV. Depression
 - A. Definition
 - B. Contributing Factors
 - C. Assessment
- V. Impact of Dementia, Delirium, Depression
 - A. Hospitalized Patients
 - B. Non-Hospitalized Patients
- VI. Nursing/Pharmacological Interventions
 - A. Dementia
 - B. Delirium
 - C. Depression
- VI. Summary and Discussion
 - A. Case Studies
 - B. Questions