

Safe Practice, Safe Patients, Safe Systems: A Non-punitive Model

Robert Clark
Fred Danforth
Debra Simmons

Level Intermediate

CONTENT DESCRIPTION

This session is intended for registered nurses both in day-to-day practice and in management and leadership roles. The focus will be on the key issues of safety in today's healthcare, the current perceptions and practices regarding error and error management in healthcare today, and the importance of current theories about error. Current error theories are transforming the work environment of nurses in order to minimize errors and provide additional systems for preventing errors besides the memory and intervention of a nurse at the point-of-care. Learning will be enhanced by real world examples of errors and the systems and environments they occurred in, included will be a framework for creating a "Just Culture" which focuses on improving problem solving and not increasing blame.

A new pilot program: the Healthcare Alliance Safety Partnership, HASP, is modeled after the highly successful approach used by the airline industry, which utilizes an increased understanding of systems and human factors in the occurrence of medical errors that involve nurse actions and seeks solutions rather than blame.

HASP offers an innovative approach that examines human performance and system factors as the root cause of medical errors, stimulates a larger intervention based upon solutions to system factors and provides protection to the public by addressing error-prone healthcare circumstances; all while protecting the nurse's practice.

The HASP project is consistent with the mission of the Texas State Board of Nurses and the systems focus of the recent institute of medicine reports. HASP seeks to provide protection to the public while also documenting the role systems and human performance factors contribute to errors.

LEARNING OUTCOMES

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

1. Describe the role of nurses in medical errors
2. Identify the steps involved in establishing a non-punitive model for resolving medical errors
3. Express an understanding of the importance of developing a program to help minimize the occurrence of medical errors

SUMMARY OF KEY POINTS

- I. Concerns about medical errors
 - A. Headline news events regarding recent medical errors
 - B. Case studies of medical errors
- II. The need for a non-punitive program that minimizes errors
- III. The HASP program:
 - A. How it was created
 - B. How HASP works
 - C. How to develop similar programs across the country

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Web Sites:

Healthcare Alliance Safety Partnership (HASP): www.texashasp.org

Institute of Medicine: www.iom.edu

Texas Board of Nurse Examiners: www.bne.state.tx.us

Saving Face: Do You Want to Look More Vibrant?

Mary C. Dugan

Level: Beginner

CONTENT DESCRIPTION

A healthy, glowing complexion and rested appearance are an asset to our personal and professional image. Nurses are health promotion experts, so we can model and provide an evidenced-based education about skin care and medical aesthetics. This session will present the physiology of aging aesthetics. This session will present the physiology of aging skin, including extrinsic and intrinsic factors, and an evidenced based description of the most popular “lunch time” procedures available today in physician offices and medical spas across the country. The list of procedures includes: glycolic acid peels; salicylic acid peels; microdermabrasion; botulinum toxin injections; injection of various fillers to treat deeper facial lines; and the use of non-ablative (laser and radio frequency) for photo damaged skin and wrinkles. Common prescription and over-the-counter medications such as tretinoin, hydroquinone, kojic acid, azelaic acid, hyaluronic acid, and alpha hydroxy acids are presented from both the prescribers’ and consumers’ point of view. The effective use of sunscreens and sun blocks, as well as the effective ingredients necessary to protect our skin from photo damage and skin cancer will be included. There are two outcomes for nurses and their patients. The first being that nurses will be versed in medical and aesthetic language. Secondly, the nurse will be familiar with common minimally invasive aesthetic procedures and will be equipped to educate patients, family and friends about these procedures and products. This information will assist the nurse in choosing appropriate procedures, medications, and over-the-counter preparations that have been proved to enhance the health and appearance of the skin and reduce the effects of aging.

LEARNING OUTCOMES

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

1. Describe the effects of intrinsic and extrinsic aging on the skin.
2. Identify 3 treatments or therapies for aging skin.
3. Formulate at least 3 critical questions to ask prior to purchasing products and procedures.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Anatomy and physiology of youthful skin
 - A. Collagen and elastin
 - B. Epidermis, papillary, and reticular dermis
 - C. Blood supply
 - D. Cellular regeneration
- III. Anatomy and physiology of aging skin
 - A. Collagen and elastin
 - B. Epidermis, papillary, and reticular dermis
 - C. Blood supply
 - D. Turn-over of cells

- IV. Intrinsic factors contributing to skin aging
 - A. Genetics
 - B. Hormones
- V. Extrinsic factors contributing to aging skin
 - A. Hydration
 - B. Exercise
 - C. Environment
 - D. Sun exposure
 - E. Smoking
 - F. Diet
 - G. Alcohol
- VI. Superficial Treatments
 - A. Glycolic acid peels
 - B. Salicylic acid peels
 - C. Microdermabrasion
 - D. Nonablative treatments
- VII. Pharmacological Therapies
 - A. Alpha hydroxy acids
 - B. Kojic acid
 - C. Hyaluronic acid
 - D. Hydroquinone
 - E. Retinoids
 - F. Salicylic acid
- VIII. Treatments for larger lines
 - A. Botulinum toxin injections
 - B. Line fillers
- IX. Prevention
 - A. Lifestyle
 - B. Sunscreen
 - C. Sunblock
- X. Questions to ask about products and procedures
- XI. Conclusions

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Sedation in the ICU: What is New?

Brenda Pun

Level: Intermediate

CONTENT DESCRIPTION

Increased scrutiny has recently been placed on appropriate titration of sedative and analgesic medications in critically ill patients, especially those being treated with mechanical ventilation. Patient comfort should be a primary goal in the ICU, including adequate pain control, anxiolysis, and prevention/treatment of delirium. However, achieving the appropriate balance of sedation and analgesia is challenging. There are grave dangers associated with both undersedation and oversedation. Without rational and agreed upon “target levels” of sedation, it is likely that different members of the healthcare team will have disparate treatment goals, increasing the chance for iatrogenic complications and potentially impeding recovery. The 2002 SCCM clinical practice guidelines (CPG) outline recommendations for sedation assessment, sedation therapy, and sedation selection. These recommendations include the routine use of goal-directed sedation delivery and daily awakening trials. In addition to providing an overview of the guidelines, this session will provide a presentation of the results of recent studies (ie ABC trial, MENDS trial, MIND trial, etc). The session will also outline an example of the implementation process of the guideline recommendations including monitoring scales, protocols and evaluations. This session is for critical care staff nurses, educators, and advance practice nurses.

LEARNING OUTCOMES

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

1. Identify the indications and dangers of sedation
2. Identify differences in drugs currently used for Sedation
3. Compare and contrast the current recommendation for sedation delivery
4. Identify key features of successful implementation of sedation protocols

SUMMARY OF KEY POINTS

- I. Why do we give sedation?
 - A. Overview of the indication for sedation
 - B. Dangers of Undersedation and Oversedation
 1. Pain
 2. Delirium
 3. PTSD
 4. Long-Term Cognitive Impairment
- II. What is new in Sedation Assessment?
 - A. Objective Monitoring – BIS monitoring
 - B. Subjective Monitoring –
 1. Sedation Scales (RASS, SAS, Ramsay, MAAS)
 2. Multi-Domain Scales
- III. What is new regarding the drugs we use for sedation?
 - A. Overview of Drugs

1. Benzodiazepines
2. Propofol
3. Alpha 2 Agonists
4. Antipsychotics
- B. MENDS Study
- C. MIND Study
- D. Other new studies
- IV. What are the recommendations (and controversies) for sedation delivery techniques?
 - A. Nurse Managed Protocols
 - B. Targeted Sedation Delivery (titration to a sedation scale/objective monitor)
 - C. Daily Awakening Trials
 1. Introduction
 2. Pro’s and Con’s
 3. ABC Study
 - D. Narcotic Driven Protocols
- V. What are the features of successful implementation of sedation protocols?
 - A. Barriers to Implementation
 - B. Implementation Framework
 - C. Example of Sedation Protocol

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Self-Study Pavilion

Sponsored by GE Healthcare

CONTENT DESCRIPTION

The Self Study Pavilion offers a change of pace for NTI participants. For those who are interested in exploring alternative avenues of continuing education, the Pavilion offers a variety of learning opportunities in a self-paced format.

In addition to general preconferences, concurrent and mastery sessions, the popular Self-Study

Pavilion offers a unique way to experience NTI and earn continuing nursing education credit through independent self-study. The Pavilion is comprised of two areas:

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Relax in the library while studying over 100 AACN educational catalog resources, including books, audio CD's and clinical simulation CD-ROMs.

LEARNING OUTCOMES

1. Acquire relevant content, to be applied to clinical practice through independent learning activities.
2. Identify and experience alternative learning methods.

Sepsis Code: ABC's of Change to Ensure Rapid Reliable Care

Joya Pickett
June Altaras

Level: Beginner

CONTENT DESCRIPTION

In the US more than 500 patients die daily of severe sepsis. This presentation describes the interrelationships of the sepsis spectrum, early identification, and research-based goal directed management of sepsis. It also highlights how to effectively and consistently implement these processes at the bedside through the utilization of reliability science. Clinicians have identified the problem, science has discovered some of the solutions and yet we struggle with implementation of appropriate care. Nurses will learn phase approach steps to ensure reliable care for their patients. Using an integrated case-study approach, the pathophysiology of the body's response to infection, SIRS, and progression to severe sepsis and septic shock will be presented. Key principles of supply and demand as measured by the ScvO₂ catheter will be discussed. AACN's Practice Alert Severe Sepsis will be highlighted. A fundamental question facing each hospital "I know the research behind the science, but how can I bring this change in practice to the patient"? The participant will learn how a focus on reliable processes, based on principles of change theory and reliability science, will result in decreased sepsis mortality. Methods and strategies for the implementation of evidence-based practice changes will be presented. Nurses will learn how the use of simple tools such as bundles, automated order sets, and small-tests-of-change can be utilized to integrate scientific principles into nursing practice. Participants will have an interactive volunteer opportunity to perform a hands-on test-of change. Target audience is adult critical care, intermediate care, progressive care/telemetry nurses, and nurse managers. No pre-requisite knowledge required.

LEARNING OUTCOMES

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

1. Differentiate between SIRS, sepsis, severe sepsis, septic shock.
2. Describe current evidence-based therapies utilized in the management of sepsis.
3. Discuss three strategies that facilitate reliable processes.

SUMMARY OF KEY POINTS

- I. The septic patient
 - A. Our case study
 - B. Cement the purpose
- II. Introduction
 - A. Why study sepsis?
 1. Incidence
 2. Outcomes

3. Financial burden
 - B. Surviving Sepsis Campaign
 1. Major healthcare challenge
 - C. AACN Practice Alert
 1. Practice recommendations
- III. Key terms & concepts
- A. SIRS
 - B. Sepsis
 1. Sepsis Spectrum
 - C. Severe Sepsis
 - D. Septic Shock
 1. Bacteremia – only 30 – 50 % present with positive blood cultures
- IV. Pathophysiology of Sepsis
- A. Antigen
 - B. Inflammation
 - C. Laboratory signs of inflammation
 1. Elevated neutrophils
 2. Increased bands
 3. Elevated WBCs
 - D. Immune system responds
 - E. Sepsis cascade
 1. Cytokine cascade
 - a. Tumor Necrosis Factor (TNF)
 - b. Interleukin 1 (IL-1)
 - c. Interleukin 6 (IL-6)
 2. Coagulation cascade response
 - a. Platelet activation
 - b. Microvascular thrombus
 - c. Impaired fibrinolysis
 - F. Endothelial cell dysfunction
 - G. Hemostasis alterations
 - H. Endogenous activated protein-C
 - I. Cardiovascular effects
 1. Vasodilatation
 2. Maldistribution of blood flow
 3. Myocardial depression
 - J. MODS
 1. ARDS
- V. Tissue oxygenation
- A. Supply and demand
 - B. Indicators of tissue hypoxia
 1. Lactate levels
 2. ScvO₂
 - C. ScvO₂ measurement
- VI. Evidence-based management of sepsis
- A. Early goal-directed therapy

1. What does it mean?
 - a. Sepsis screen
 - b. Vigilant nursing assessment
 2. Sepsis code concept
 3. Volume resuscitation - CVP
 4. Vasopressors
 - B. Antibiotic administration
 1. Antibiotics STAT
 - C. Lung protective ventilation
 - D. Low-dose steroid therapy
 - E. Tight glycemic control
 1. Patient outcomes
 2. What's all the fuss?
 - F. Activated protein-C (drotrecogin alpha activated, Xigris)
 1. How does it work?
 2. When indicated?
 3. Administration guidelines
- VII. Background
- A. Reliability in health care
 - B. Health care goals and aims
 1. Focus on patient safety
 2. Reliably implement patient care
 3. Mortality
 4. 100K Lives Campaign
- VIII. Basic concepts and how terminology relates
- A. Reliability
 1. The science of reliability
 2. What does it really mean?
 3. Focus on the process
 - B. Failure rate
 - C. Human factor science
 - D. Outcomes
 - E. Process measures
- IX. Process Bundles
- A. What's a bundle?
 1. Collection of research-based practices
 2. Bundle elements
 3. Minimum numbers
 - B. Reliability measurement
 1. Comparative reliability between industries
 - D. Reporting data
 1. Which patients
- X. Change
- A. Principles of change
 1. Plan, Do, Study, Act: PDSA
 2. Small test-of-change cycles
 - a. Phase approach
 3. How long will it take?
 4. Barriers to change and combat strategies

- XI. Phase I
- A. Gather the troops
 1. Strong administrative support
 2. Collaborate, collaborate, collaborate
 - B. Do the leg work
 1. Protocol and algorithm development
 2. Order set development
 3. Audit tools
 4. ScvO₂ catheter implementation
 - C. Pilot unit
 - D. Staff feedback
 - E. Evaluation
- XII. Phase II
- A. Partial Implementation
 1. Parts of the whole
 - B. Complete implementation
 - C. Measure
 - D. Data evaluation
 - E. Staff feedback
 - F. Re-measure, re-work, re-do
- XIII. Phase III
- A. Time to spread
 1. Rapid Response Team (RRT), Emergency department
- XIV. Let's practice reliability
- A. Form small voluntary team groups to practice hands-on test-of change

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Sepsis: Identification and Management of Sepsis

Tom Ahrens

Mike Ackerman

Sponsored by Eli Lilly and Company

Level: Intermediate

CONTENT DESCRIPTION

One of the most complex and clinically dangerous conditions in acute and critical care is sepsis. This program is designed to help the bedside clinician successfully intervene in sepsis to improve survival in this leading cause of critical care deaths. This educational program on sepsis serves two key functions, i.e. providing a sense of clarity to the complicated clinical area of sepsis identification and management and providing innovative educational strategies to enable a superior learning experience. In this program, the pathogenesis of sepsis is discussed, along with new theories from genetics to immune system modulators. The program, while addressing complex topics, is centered on helping the bedside clinician easily and accurately identify sepsis. The program also helps the clinician simply treatment options while including the latest evidenced based guidelines from the Surviving Sepsis Campaign. At the end of the program, the clinician will be better prepared to recognize and successfully treat the patient with sepsis.

LEARNING OUTCOMES

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

1. Identify how sepsis can appear to be subtle until life threatening organ dysfunctions occur
2. Describe the key treatments for sepsis, including limitations and strengths
3. Discuss the essential concepts of how to implement sepsis management guidelines

SUMMARY OF KEY POINTS

- I. Importance of managing sepsis
 - A. the subtlety of sepsis presentation
 - B. sepsis beyond the ICU
 - C. Implications in resource consumption
 - D. Mortality
 - E. Patient wishes
- II. Pathophysiology of sepsis
 - A. Role of immune system and the emerging role of genetics
 - B. Septic cascade
 1. inflammation at tissue level
 2. endothelial cell dysfunction
 3. altered hemostasis
 4. impact on tissue oxygenation
 5. altered SvO₂ levels
- III. Identification of the patient with sepsis
 - A. Definition of sepsis
 - B. Definition of organ dysfunction
 - C. Need for early identification and intervention

IV. Treatment of sepsis - Issues and Controversies

- A. Surviving sepsis campaign - 2007 revisions from 2004
- B. Current therapies - what works and what does not - controversies with clarity
 1. Volume expansion, vasopressors and inotropes with goal directed therapy
 2. Antibiotics
 3. Lung protective strategy
 4. Replacement steroids
 5. Glucose regulation
 6. Activated protein C (drotrecogin alpha activated or Xigris)
 - a. Mechanism of action
 - b. Research regarding use
 - c. When to give and who should receive this therapy

V. Implementation of bundles and Clinical Case studies

VI. Summary

- A. Management of sepsis in first part of 21st century
- B. Role of nursing at improving patient outcomes

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Severe Acute Pancreatitis (SAP): Managing a Sticky Situation!

Linda DeStefano

Level: Advanced Practice

CONTENT DESCRIPTION

There are approximately 210,000 cases of pancreatitis yearly in the United States, and approximately 20% progress to Severe Acute Pancreatitis (SAP) with a mortality of 30-50%. This presentation provides an evidence-based overview of key concepts related to the care of the SAP patient including: review of anatomy, physiology, pathophysiology, etiology, assessment, monitoring, laboratory and diagnostic findings, differential diagnosis, invasive and non-invasive treatment strategies in accordance with the latest Society of Critical Care Medicine 2004 International Consensus Conference Statement.

This concurrent session discusses the interpretation of scoring systems to identify severity and those at increased risk of adverse outcome, evaluation of serum biomarkers, and radiographic imaging results necessary to formulate a diagnosis and treatment plan. The evidence-based guidelines direct appropriate use of fluid resuscitation, prophylactic antimicrobials, and other pertinent therapies. Techniques to assess for complications such as intestinal infarction, perforation, hemorrhage, acute renal failure, biliary tract disease, and abdominal compartment syndrome will be discussed, as well as procedures to differentiate between sterile and infected pancreatic necrosis. Optimization of nutrition including feeding methods, formulas, and post-pyloric small bowel feeding tube placement techniques will also be presented.

Providing appropriate interventions to abort progression of SAP to sepsis, multi-organ dysfunction and death requires a skilled multidisciplinary team approach. Critical-care and Advanced Practice Nurses who have knowledge of the 23 recommendations related to patient management as outlined in the latest evidence-based consensus statement for SAP will be able to greatly assist the healthcare team in providing optimal care and facilitate improved patient outcomes.

LEARNING OUTCOMES

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

1. Evaluate clinical symptoms, patient presentation, and diagnostic findings for SAP patients.
2. Discuss treatment strategies according to the latest consensus statements for the management of SAP.
3. Describe optimal methods and timing of providing nutritional support utilizing an evidence based approach.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Definition of Severe Acute Pancreatitis (SAP)
 - B. Epidemiology
 1. Morbidity

2. Mortality
3. Associated costs in United States
- C. Etiology
 1. Toxins/alcohol
 2. Biliary tract disease
 3. Medications
 4. Trauma- blunt, procedural or surgical
 5. Tumors, cancer
 6. Infections: bacterial, viral, parasitic
 7. Hyperlipidemia/vascular disease
 8. Hypercalcemia/hyperparathyroidism
 9. Pregnancy
 10. Hypothermia
 11. Idiopathic (10-20%)
 12. Other
- II. Anatomy, Physiology, and Pathophysiology
- III. Assessment and Diagnosis
 - A. Clinical manifestations
 - B. Prognostic and Severity Assessment Tools
 1. Ranson's criteria
 2. Glasgow criteria
 3. APACHE Score
 4. Sequential Organ Failure Assessment (SOFA)
 5. Atlanta criteria
 6. Balthazar score (CT severity index)
 7. Others
 - C. Classification by timing
 - D. Laboratory Tests
 - E. Diagnostic Tests and procedures
 - F. Patient History
- IV. Patient Management: Evidence Based recommendations/interventions using a multidisciplinary approach
 - A. Initial resuscitation
 1. Fluids: restore circulating volume
 2. Respiratory support
 - B. Treatment based on extent of injury
 - C. Treatment of systemic complications
 - D. Treatment of local complications
 - E. Patient Placement
 - F. Nutrition
 - G. Prevention of iatrogenic complications
 - H. Long-term complications
- V. Case Studies

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Shake, Rattle and Roll: Snake Bite Management

Scott C. Thigpen

Level: Intermediate

CONTENT DESCRIPTION

Twenty-five species of poisonous snakes make North America their home. Worldwide, more than 3000 species of snakes are considered dangerous to humans. Snake bite management is a medical emergency and health care professionals must act quickly to determine the type of snake bite and institute appropriate care to promote healing and prevent disability. Appropriate field care and hospital management will be discussed. This session will examine the myths of snake bite management and evidenced based research findings. Treatment is based on the severity of envenomation. Participants will explore the use of a grading scale to determine the degree of envenomation and appropriate treatment of poisonous snake bites. The effects of envenomation produce local and systemic disruption and may contribute to multiple organ system failure. Pulmonary mechanics may be altered significantly with local edema, increases capillary leak and interstitial fluid in the lungs. Cardiac failure can result from hypotension and acidosis. Myonecrosis raises concerns about myoglobinuria and renal damage. Management of local and life-threatening systemic reactions will be discussed as well as hemotoxic and neurotoxic symptoms. Pharmacologic therapies, including antivenin administration, and wound management will be explored. All attendees will learn methods to prevent snake bites through the creation of healthy environments. The goal of this session is to provide nurses with the knowledge to deliver appropriate care to clients with a snake bite injury.

LEARNING OUTCOMES

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

1. Differentiate between poisonous and non poisonous snakes.
2. Utilize the Severity of Snake Bite Management Scale to determine the degree of the envenomation of poisonous snakes.
3. Discuss appropriate pharmacologic and wound management for snake bites.

SUMMARY OF KEY POINTS

- I. Life in the Okefenokee Swamp
- II. Snake Bite Management – A Medical Emergency!
- III. First Aid for Snake Bites – Myths Exposed
- IV. Venomous vs. Non-venomous Snakes
- V. Snake Envenomation
- VI. Local Reactions
- VII. Life-Threatening Systemic Reactions
- VIII. Snake Bite Severity Scale
- IX. Hemotoxic vs. Neurotoxic Symptoms
- X. Diagnostic Laboratory Testing
- XI. Antivenin Administration
- XII. Pharmacotherapeutic Management: Ovine crotalidae polyvalent immune fab-purified (Crofab), Ceftriaxone (Rocephin), Diphtheria-tetanus toxoid
- XIII. Wound Management: Surgical vs. Non-Surgical Intervention
- XIV. Healthy Environments – Education and Prevention of Snake Bites

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- Centers for Disease Control and Prevention. How to Prevent or Respond to a Snake Bite <http://www.bt.cdc.gov/disasters/snakebite.asp>
- Crotalid Envenomation <http://www.krpc.com/proffed/snake%5Csnakebite.cfm>
- Emedicine Instant Access to the Minds of Medicine: Snakebite <http://www.emedicine.com/med/topic2143.htm>
- Snake Bite Critical Care <http://www.emedicine.com/med/TOPIC2143.HTM>
- Snakes of North America <http://www.pitt.edu/~mcs2/herp/SoNA.html>

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She's a Good Nurse But...

Lisa Pettrey
Denise Thornby

Level: Beginner

CONTENT DESCRIPTION

How often in your career have you heard someone say, She's a good nurse, but or He's a good nurse, but...? Whatever description follows the work "but" is never good nor positive. In fact most often the words that follow describe behaviors that threaten teams, learning and growing and all that we define as healthy work environments. Often the descriptors indicate behaviors found in the literature describing lateral violence or nurse-to-nurse hostility. These behaviors disrupt team relationships and effective communication. They negatively impact our ability to safely and effectively care for patients. This presentation will talk about the behaviors of horizontal violence and strategies to confront and eradicate these destructive behaviors. Case studies, humor and interactive discussion will be utilized. Any staff nurse, manager, advance practice nurse, or leader with a desire to better communicate with others would benefit from this session.

LEARNING OUTCOMES

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

1. Define lateral violence and workplace bullying and the workplace conditions that allow it to exist.
2. Describe strategies to manage or prevent lateral violence.

SUMMARY OF KEY POINTS

- I. How big is this issue?
 - A. Crabs and the Crab bucket
 - B. Difficult People
 - C. Silence Kills – painted part of the picture
- II. Good Nurses
 - A. Great clinical knowledge
 - B. Great judgment and ability to use their knowledge well
 - C. Great interpersonal and team skills
- III. Nurse-to-Nurse Hostility or Lateral Violence
 - A. Lateral violence occurs when nurses covertly or overtly direct their dissatisfaction inwards towards each other, towards themselves and towards those less powerful than themselves
 - B. Oppression theory: oppression elicits negative behaviors: silence, a lack of voice, poor self-esteem and the experience of powerlessness and lateral violence is the natural expression of the resulting anger.
 - C. Who might be considered less powerful in your unit?
- IV. Workplace Bullying
 - A. Psychological violence – the intentional use of

power, including threat of force, against another person or group that can result in harm to physical safety, mental, spiritual, moral or social development.

- B. Reasons for bullying in the workplace:
 1. Failure to achieve a goal
 2. Thwarting of ambitions and wishes
 3. Feeling threatened
 4. An alteration in one's physical or mental state
 5. Substance Abuse
 6. An aggressive personality type
 7. Learned behaviors
- V. Most common behaviors seen in lateral violence in nursing practice
 - A. Nonverbal innuendo
 - B. Verbal affront
 - C. Undermining activities
 - D. Withholding information
 - E. Sabotage
 - F. Infighting
 - G. Scapegoating
 - H. Backstabbing
 - I. Failure to respect privacy
 - J. Broken confidences
- VI. Why does it still continue?
 - A. Denial
 - B. Subtle or not visible
 - C. Lack of Confrontation
 - D. Acceptance of the behavior
 - E. Managers not dealing with the behaviors
- VII. So, what do we do?
 - A. Confront disrespectful behavior and unprofessional behavior
 - B. Don't tolerate malicious gossip and disrespectful behavior towards others
 - C. Manage our boundaries
 - D. Develop a zero tolerance
 - E. Work to create a healthy work environment to support empowered nurses and team members.
- VIII. Be Prepared:
 - A. Think of what you would say
 - B. Think of how you would respond
 - C. Practice
- IX. Stop the Cycle
 - A. Standing up to the bullies
 - B. Unflinching attention
 - C. Higher expectations
 - D. Commit to nothing less than professional, respectful behavior.

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- www.silencekills.com

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The Silent Epidemic: Obesity's Impact on Critical Illness

Sonia M. Astle

Level: Beginner

CONTENT DESCRIPTION

The scope of this presentation is to link research and evidence based guidelines to practice in order to predict, prevent and manage complications associated with obesity. Case analyses will uncover hidden complications affecting morbidly obese critically ill patients experiencing increased intraabdominal pressure, rhabdomyolysis, acidosis and respiratory failure. Key concepts include advanced pharmacotherapy and medication calculation for the obese patient, fluid resuscitation, mobility, bariatric equipment and injury prevention techniques for health care providers. There are no prerequisites for this presentation. The target audience includes progressive care, critical care and advance practice nurses.

LEARNING OUTCOMES

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

1. Analyze the prevalence of obesity in the United States.
2. Identify the pathophysiology associated with morbid obesity.
3. Predict, prevent and manage complications associated with morbid obesity.
4. Discuss specific competencies required to care for the morbidly obese patient through case presentation analysis.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Major health epidemic in United States
 - B. 50% population overweight - body mass index (BMI) greater than 25 kg/m²
 - C. 30% population obese BMI greater than 30 kg/m²
 - D. 5% population morbidly obese BMI greater than 40 kg/m²
 - E. Leading cause of preventable death, excess deaths over 300,000 annually
 - F. Cancer mortality – 52% increase in men and 62% women with BMI greater than 40
 - G. Annual cost of \$100 billion, 10% of national health care expense
 - H. Require both hospitalization and intensive care (6%-24% bariatric surgical patients require ICU care
 - I. Prejudice and discrimination
- II. Body Mass Index (BMI)
 - A. Assesses weight relative to height
 - B. Ratio of body weight in kilograms (kg) to the square of height in meters

- C. Expressed as kg/m²
- D. BMI for age is the calculation used for pediatrics (most treatment is weight based)
- III. Comorbidities associated with obesity
 - A. Cancer (esophagus, colorectal, liver, gallbladder, pancreas, kidney, non-hodgkins' lymphoma, multiple myeloma)
 - B. Cholelithiasis
 - C. Coronary heart disease
 - D. Dyslipidemia/hypercholesterolemia
 - E. Diabetes (Type 2)
 - F. Hypertension
 - G. Hypertrophic cardiomyopathy
 - H. Hypoventilation
 - I. Psychosocial impairments
 - J. Sleep apnea
 - K. Stroke
 - L. Thrombophlebitis
- IV. Physiology of Obesity
 - A. Imbalance of energy intake and expenditure
 - B. Adipose tissue stores fat – serves as an energy reservoir
 1. Excess fat accumulation increases diameter and number of adipose cells
 2. Increased size of normal structures and fat interfere with body mechanics
 3. Requires additional need for oxygenation and alveolar ventilation to maintain tissue perfusion
 4. Increased blood flow requirements to adipose tissue
- V. Pharmacokinetics
 - A. Pharmacodynamic alterations are due to a greater percent of adipose tissue and a lower percentage of water and lean body mass
 - B. Medications high in lipid content (absorbed in adipose tissue) require dose calculation based on actual weight
 - C. Medication with minimal lipid (absorbed mainly in lean tissue) require dose calculation based on patient ideal weight
 - D. Lower gastric pH affects drug absorption
 - E. IV route preferred, subcutaneous and dermal patches poorly absorbed
 - F. Medication calculation: actual versus ideal body weight
 - G. Fluid deficit calculation: Modified formula – not well researched!
 1. Part I Weight = IBW+(0.4 x [ABW-IBW])

a. Example:

ABW = 200 kgs IBW = 74 kgs

(200 - 74 = 126 kgs)

Difference ABW and IBW x 0.4 =

(126 x 0.4 = 50)

IBW + 40% difference of ABW and IBW

(74 + 50 = 124)

2. Part II Dosing factor x weight (kg) x ((serum Na/140) - 1)

a. Example:

(Dosing factor = 0.6 male and 0.5 female)

0.6 x 124 x ((160/140) - 1) = 10.4 Liters

3. Adrogué HJ et al. Hyponatremia. N Engl J Med 2000 May 8;342(20):1493-9

VI. Pathophysiology of Obesity

A. Immunology

1. Creates a chronic inflammatory state
2. Adipose tissue source of tumor necrosis factor and interleukin-6
3. Impaired neutrophil function

B. Metabolic

1. Increased resting energy expenditure
2. Insulin resistance
3. Increased proteolysis
4. Elevated circulating concentrations of catecholamines, mineralocorticoids, renin and aldosterone

C. Respiratory

1. Increased work of breathing caused by
 - a. Increased blood volume and chest wall mass
 - b. Abnormal diaphragm position
 - c. Upper airway resistance
 - d. Increased CO₂ production
2. Complications
 - a. Decreased compliance
 - b. Decreased functional residual capacity (FRC), total lung capacity (TLC), vital capacity (VC), inspiratory capacity (IC), expiratory reserve volume (ERV) and reserve volume (RV)
 - c. Increased ratio of forced expiratory volume (FEV₁) in one second to forced vital capacity (FVC)
 - d. Increased work of breathing
 - e. Atelectasis
3. Ventilator Management
 - a. Tidal volume calculated on ideal body weight (IBW) – adjust by plateau pressure and arterial blood gas (ABG) analysis
 - b. Mode of ventilation is not different for the obese patient – full support given for first 48 hours to unload the fatigued diaphragm
4. Airway Management
 - a. Supine = Decreased FRC
 - b. Desaturation
 - c. BVM difficult
 - d. End-tidal CO₂ unreliable – ABG better indicator of minute ventilation
 - e. Limited neck mobility

f. Short sternomental distance

g. Need longer trach tube

h. Modified trach removes cervical fat

i. Percutaneous tracheostomy contraindicated

j. Cormack and Lehane Score for intubation

(1) Grade I: most of glottis can be seen

(2) Grade II: only posterior portion of glottis is seen

(3) Grade III: only epiglottis (none of glottis seen)

(4) Grade IV: neither epiglottis nor glottis can be seen

D. Cardiovascular system

1. Increased blood volume to support adipose tissue (3mL blood per 100 Gm of adipose tissue), increases preload, stroke volume, cardiac output and myocardial work
2. Catecholamines, renin and aldosterone increase afterload
3. Complications
 - a. Hyperkinesis
 - b. Myocardial hypertrophy
 - c. Decrease compliance
 - d. Diastolic dysfunction
 - e. Ventricular failure
 - f. Cardiac arrhythmias

E. Gastrointestinal system

1. Hiatal hernia
2. Decreased gastric pH
3. Increased gastric secretion volume
4. Nutrition
 - a. Critically ill patient cannot mobilize fat stores for energy – use protein
 - b. Early enteral feeding recommended
 - c. Enteral feeding recommendations 30kcal/kg and 2 g/kg protein/day
 - d. Research, Dickerson et al – hypocaloric enteral feedings (less than 20 kcal/kg/day based on IBW) had decrease ICU length of stay, fewer antibiotics and decrease length of ventilator days

F. Hematological system

1. Increased viscosity, decreased concentration of antithrombin-III
2. Increased fibrinogen and plasminogen activator inhibitor 1 (produced by adipose tissue).
3. Sedentary life style – venous stasis
4. Endothelial damage (post operative)
5. Management
 - a. Thromboprophylaxis
 - b. Prophylactic inferior vena cava filter

G. Renal system

1. Increased clearance of renally excreted drugs
2. Hypertensive and diabetic nephropathy

H. Integumentary system

1. High risk for skin breakdown, use of specialized bariatric equipment

2. Hyperglycemia increases prevalence of infection and delays wound healing
 3. Skin folds become moist harboring bacteria and yeast and are prone to breakdown due to poor vascular supply to adipose tissue
 4. Prevention – scheduled turning and prevention of shearing
 5. Position off drainage tubes
- VII. Pressure-Induced Rhabdomyolysis
- A. Overview
 1. Syndrome characterized by muscle necrosis, the release of intracellular muscle constituents into the circulation, pigmenturia is due to myoglobinuria in association with elevated serum muscle enzymes
 2. Elevation of serum muscle CK elevated to above 100,000 IU/L almost all in the form of CK – MM (skeletal muscle fraction)
 - B. Etiology:
 1. Prolonged, unrelieved pressure involving lower limbs, gluteal or lumbar regions
 2. Abdominal compartment syndrome following abdominal surgical procedures
 - C. Management
 1. Prevent (padding and positioning during OR)
 2. Aggressive hydration
 3. Prevent or manage acute renal failure due to myoglobinuria
 4. Forced diuresis (for creatinine greater than 5,000 IU/L) with mannitol (mobilizes muscular interstitial fluid and increase renal tubular flow)
 5. Alkalinization of the urine - sodium bicarb (increases the solubility of myoglobin)
 6. Target goal for urine output - 1.5mL/kg/hr
 7. Hemofiltration
 8. Fluids and electrolyte replacement
 9. Treat compartment syndromes
- VIII. Added Considerations
- A. Vascular Access
 1. Altered landmarks makes insertions difficult
 2. Increased depth of insertion to gain venipuncture
 3. Distorted angle of insertion
 4. Fewer complications for internal jugular access
 5. Skin folds predispose to increase catheter related blood stream infections
 - B. Bariatric equipment
 1. Hill-Rom, Inc (<http://www.hill-rom.com>)
 2. Kinetic Concepts, Inc (<http://www.kci1.com>)
 3. SIZEwise Rentals (<http://www.sizewise.net>)
 4. Wheelchairs of Kansas (<http://www.wheelchair-sofkansas.com>)
 - C. Injury Prevention

- IX. Outcome Measures
- X. Bundle Compliance
 - A. Positioning - reverse trendelenburg 45 degrees
 - B. DVT prophylaxis
 - C. Glucose management
 - D. Wound prevention
 - E. Early mobilization

XI. Case Presentations

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Skilled Communication and Patient Safety: Making the Connection

Vicki Good
Sonya Flanders
Web Cast Sponsored by GE Healthcare

Level: Beginner

CONTENT DESCRIPTION

Skilled communication is the first standard in the AACN Standards for Achieving and Sustaining a Healthy Work Environment due to the importance communication has on teamwork and patient safety. This session will explore tools and techniques the bedside RN can utilize to enhance the communication on his/her unit. Tools and Techniques will include: Structured communication, critical language, and briefing and debriefing.

LEARNING OUTCOMES

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

1. Define the key elements of skilled communication needed to sustain a healthy work environment.
2. Articulate the impact of skilled communication on teamwork in the critical care environment.
3. Demonstrate utilization of a variety of communication techniques based on the needs of the clinical situation.

SUMMARY OF KEY POINTS

- I. Key elements of skilled communication needed to sustain a healthy work environment.
 - A. Regulatory importance of skilled communication and the implications of lack of communication on adverse events.
 1. Joint Commission requirements for effective hand-off communication (nurse to nurse, nurse to physician or other care delivery personnel, physician to physician).
 2. Case studies demonstrating the impact of communication on adverse events and ultimately lawsuits.
 - B. Types of communication present in the critical care work environment.
 1. Informal lines of communication
 2. Formal lines of communication
 - a. Shift to shift
 - b. Nurse to physician
 - c. Nurse to mid-level practitioner
 - d. Physician to physician
 - C. Communication challenges present in the critical care work environment.
 1. Characteristics of communication utilized by physicians versus nurses.
 2. Timeliness of communication
 3. Critical nature of critical care communication.

- D. Characteristics of skilled communication
 1. STEEEP
 - a. Safe
 - b. Timely
 - c. Efficient
 - d. Effective
 - e. Equitable
 - f. Patient centered
- II. Articulate the impact of skilled communication on teamwork in the critical care environment.
 - A. Critical care team and function.
 1. Team definition
 2. Key characteristics and strategies of a team.
 - B. Interaction of key skills (communication, situational awareness, leadership, mutual support) on the functionality of a team.
 - C. Crucial points of communication within a critical care team.
 - D. Relationship and rapport
- III. Demonstrate utilization of a variety of communication techniques based on the needs of the clinical situation.
 - A. Structured communication methods
 1. SBAR
 - a. SBAR Communication Template
 - S SITUATION
Pertinent information about what is going on with the patient now
 - B BACKGROUND
Frame the pertinent clinical background / situational context
 - A ASSESSMENT
(The Problem)
 - R RECOMMENDATION
(What is needed to address the problem?)
 - (a) Situation
 - (b) Background
 - (c) Assessment
 - (d) Recommendation(s)
 - B. Case Example
 1. Critical language/Assertion
 - a. "Stop the Line"
 - (1) Keyword examples
 - (2) Chain of Command
 - b. Case examples
 - C. Team Activities
 1. Briefing
 2. Huddle

3. Debriefing
 - a. Guidelines for feedback

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Skilled Communication: Do You Hear What I Hear?

John F. Dixon

Level: Beginner

CONTENT DESCRIPTION

The AACN Standards for Establishing and Sustaining a Healthy Work Environment include the standard of Skilled Communication. In acute and critical care work environments, communication is a complex issue at the best of times and everyone encounters situations where communication challenges exist. The purpose of this session is to examine communication focusing on evidence that defines its importance and issues impacting successful communication. Considerations for viewpoints, context, and settings will be presented. Feedback from staff related to communication will be described along with possible solutions based on these identified needs. Guidelines and methods for handling both structured and unstructured communication situations will be outlined. Methods for integrating communication skills in a variety of roles will be examined. This presentation is appropriate for all nurses seeking Skilled Communication knowledge and tools to positively contribute to healthy work environments. No pre-requisite knowledge is required for this session.

LEARNING OUTCOMES

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

1. Describe the impact and importance of communication in the clinical setting.
2. Identify communication issues and pathways.
3. Discuss potential strategies and considerations to improve communication.

SUMMARY OF KEY POINTS

- I. AACN's Healthy Work Environment Standards
 - A. Rationale for Need, Development, & the 6 Standards
 - B Linkage with other references:
 1. AACN Synergy Model for Patient Care, Forces of Magnetism, & Quint Studer 9 Principles
 - C. Focusing on Skilled Communication
- II. Making a Case for Communication
 - A. Silence Kills
 - B. Sentinel Events & JCAHO
 - C. ANA Code of Ethics
 - D. Your Own State's Nurse Practice Act
 - E. Your Own Institution – Mission, Vision, & Philosophy
 - F. What's In It For Me (WIIFM). = 2080, 8760, 23.7%
 - G. Goals of Effective Respectful Dialogue
- III. Learnings from Other Contexts
 - A. Aviation - Crew Resource Management
 - B. High Reliability Organizations - 5 Hallmarks
- IV. The Complexity of Communication

- A. Traditional issues
 - B. Educational Backgrounds & Socialization Processes
 - C. The Language of Chest Pain
 - D. "Iffers" vs. "Untillers"
- V. Assessing Your Environment
 - A. Types of Handoffs - Structured vs. Unstructured
 - B. Outcomes of Interactions
 - C. Gaps vs. Doing Well
 - D. Reporting & Tracking Systems
 - E. Zero Tolerance Policies
 - VI. Communication Strategies
 - A. Templates - the Alphabet Soup
 1. Uni-directional information vs. Bi-directional dialogue
 2. 3 Phase Model
 - B. Universally Recognized Phrases - Your CPR Class
 - C. Functioning Under Stress
 - D. Sending Mail to the Right Address – The Deadly Triangle
 - VII. Integrating Communication Development into All Roles
 - A. New Graduates, Incumbent Staff Nurses, Educators, Preceptors, Advanced Practice Nurses, & Nurse Managers
 - VIII. Closing Thoughts
 - A. Culture, Commitment, & Vigilance

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Sleep Smart: Understanding Why Patients Don't Sleep

Christine Hedges

Level: Intermediate

CONTENT DESCRIPTION

Disturbed sleep may be responsible for serious decrements in critical care patients' conditions and can herald daytime dysfunction, delayed healing, cognitive disturbances, agitation and delirium. Critically ill patients have few opportunities to receive undisturbed periods of sleep. Yet sleep promotion, as an intervention, is afforded less importance alongside other highly demanding and technical activities. Although the purpose of sleep remains unknown, research indicates that sleep plays an important role in the maintenance of overall health, including healing, physical restoration, and mental acuity. However, in order for sleep to be restorative, it needs to be continuous for 7-8 hours. The problem of disturbed sleep is complex, as many patients report pre-existing sleep disorders prior to hospitalization. If critical care nurses are to create a healing, humane environment and make their optimal contribution to patients' needs, then evidence-based strategies to promote sleep should be employed. In this presentation, the speaker will discuss the circadian, ultradian and homeostatic sleep processes, and review sleep stages and their distribution. Correlates of disturbed sleep in critical care, such as age, pain, mood, environmental disturbances, cardiac and respiratory conditions will be discussed as well as common sleep disorders and pharmacological treatment for insomnia. The presenter will include techniques for nurses to use to obtain a sleep history and will discuss valid and reliable methods of sleep measurement, including polysomnography, actigraphy, questionnaires and diaries. The speaker will address the scope of the problem of disturbed sleep in critically ill and acute care patients by presenting findings from current sleep research and will incorporate examples from her own program of research on sleep and cognition in cardiac patients.

LEARNING OUTCOMES

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

1. Discuss the current theories about the sleep process including sleep stages and the physiology associated with the REM and NREM sleep.
2. Identify intrinsic and extrinsic factors associated with poor sleep in the critical care setting.
3. Identify methods of assessing sleep and obtaining a sleep history in critically ill patients.
4. Discuss current research findings related to sleep disturbances in critical and acute care.

SUMMARY OF KEY POINTS

- I. Human Sleep
 - A. Define sleep and discuss the current theories about the purpose of sleep

- B. Theories of sleep
 1. Sleep homeostasis theory
 2. The two-process model of sleep
 - a. Interaction of Process "S" and Process "C" alongside circadian process
 - b. Opponent model of sleep-inducing process
 3. Circadian, ultradian and homeostatic perspectives
 - a. 24-hour biologic clock aligned with other body processes (temperature, hormonal regulation)
 - b. Brain regulation of circadian processes in suprachiasmatic nucleus and anterior hypothalamus
 - c. Importance of environmental cues, zeitgebers
 - d. Sleep-wake cycles
- II. Sleep Cycles
 - A. Stage 1-4 Non-rapid Eye Movement (NREM) and Rapid Eye Movement (REM) sleep
 - B. NREM-REM cycles
 - C. Distribution of sleep stages throughout the night
 - D. Characteristics of body and brain activity throughout the stages
 - E. Polysomnography
- III. Factors affecting sleep
 - A. Intrinsic factors
 1. Aging - changes in sleep associated with aging
 - a. Increased fragmentation
 - b. Insomnia
 - c. Excessive daytime sleepiness
 - d. Nocturia and falls
 2. Mood disorders
 - a. Depression, anxiety and mood disturbances
 - b. Impact of antidepressants, sleeping pills, OTC medications, polypharmacy
 3. Pain
 - a. Acute Pain and Sleep
 - b. Chronic Pain and Sleep
 - c. Effect of opioids on sleep
 4. Cardiac and respiratory disease
 - a. Sleep disorders and hypertension
 - b. Changes in vascular tone
 - c. Higher SNS activation
 - d. Sleep disordered breathing
 5. Sleep disorders
 - a. central sleep apnea
 - b. apnea-hypopnea syndromes
 - c. Obstructive sleep apnea syndrome
 - d. Insomnia and shift work

- 6. Pharmacology for Insomnia
 - a. Benzodiazepines
 - b. Benzodiazepine-receptor agonists
 - c. Sedating antidepressants
 - d. Commonly used OTCs
- B. Extrinsic Factors
 - 1. Noise, light and temperature
 - a. Hospital beds, unfamiliar environment
 - b. US EPA recommended dBA noise level
 - c. Measurement of noise
 - d. Sources of noise in ICUs
 - 2. The hospital and ICU environment
 - a. Lack of privacy in critical care
 - b. Inability to adhere to bedtime routines
 - c. Hospital design features, healing environments
- IV. Sleep assessment and obtaining a sleep history
 - A. Obtaining a sleep history
 - B. Methods of measuring sleep
 - 1. Polysomnography
 - 2. Actigraphy and motion loggers
 - 3. Sleep diaries
 - 4. Self-report instruments
- V. Current Sleep Research in acute care settings
 - A. Sleep in cardiac surgery patients
 - 1. Mood and cognition
 - 2. On-pump vs off-pump surgery

- B. Sleep in heart failure patients
- C. Sleep and Noise Disturbances
- VI. Future directions

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So You want to be a Flight/Transport Nurse

Katie Schatz

Level: Beginner

CONTENT DESCRIPTION

The purpose of this session is to provide the participant with the knowledge base to be appropriately prepared when selecting and applying for a flight/transport position and if already doing transport informally, to be educated as to the basics of liability within the transport role. Many nurses are doing transport without the proper training, competencies and support needed to provide optimal patient care. This session will introduce the specialty of flight/transport nursing; the prerequisites, skills and knowledge needed to be effective in the role of flight/transport nurse. Case studies will be presented to reinforce the skill and knowledge requirements.

This course is for any nurse who is interested in adult and / or pediatric flight/transport, from the novice RN to the advanced practice nurse.

There are many different types of transport teams and many differing roles (skill-mix) within the transport specialty. This session will introduce these roles along with a basic overview of ground and flight transport including the Commission on Accreditation of Medical Transport Services (CAMTS) accreditation. Review of certifications and requirements needed to apply for a position, as well as the legalities and liabilities related to the autonomous role will be discussed. Case studies involving “on scene” medical and trauma patients as well as interfacility transport by ground, helicopter (rotor wing) and airplane (fixed wing) will be used to demonstrate the unique complexities of the role. A question/answer period will allow the opportunity for participants to seek clarification of the content/ role.

Background

As the number of air and ground transport companies increase in the US the shortage of qualified nurses applying for these positions and the lack of support and standards of non-accredited companies lead nurses to potentially be placed in high risk situations both for patient safety and for their personal safety. Through an overview of the transport specialty and review of the role and the autonomous decision making necessary to provide competent patient care, the nurse attending this course will be better prepared to advocate for proper training, and support of both patient safety and personal safety.

LEARNING OUTCOMES

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

1. The participant will be able to discuss the requirements for competent flight/transport nursing.
2. The participant will understand the autonomous role, including the liability of flight/transport nurse.
3. The participant will be able to compare and contrast the different roles between the hospital based RNs and the flight/transport RNs.

SUMMARY OF KEY POINTS

Objective: Requirements for competent flight/transport nursing

- I. Introduction to Flight/Transport Nursing
 - A. Accreditation of transport teams
 - B. Types and skill-mix within transport teams
 - C. Standard basic requirements for applying for a position

Objective: Autonomous role, including the liability of flight/transport nurse

- II. The Legalities and Liability Specific to the Role of Transport Nurse
 - A. Training requirements
 - B. Protocol guidelines
 - C. Competency definitions

Objective: Different roles between the hospital based RNs and the flight/transport RNs

- III. Case Studies
 - A. On scene case study involving an adult patient
 - B. Special considerations for pediatric patients
 - C. Interfacility transport of adult patients
- IV. Question and Answer time

BIBLIOGRAPHY/WEBLIOGRAPHY

www.camts.org
www.flightweb.com
www.astna.org

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Speeders, Slowers, Squeezers, Unloaders, Pee-makers and Clot-busters

Lorraine Micheletti

Level: Beginner

CONTENT DESCRIPTION

This session will discuss the pharmacokinetics of major cardiac and related medications utilized in the critical care area. The discussion will start with a review of the concepts of cardiac output; namely, preload, heart rate, contractility and afterload. This will set up the discussion on how cardiac medications affect cardiac output.

The second part of the discussion will center on the cellular effects of these medications and how they manipulate cardiac muscle function. Diuretics will also be discussed to review major concepts that affect renal function in relation to cardiac disease. And lastly, this session will discuss the bleeding/clotting cascade and anticoagulation agents that influence these processes.

LEARNING OUTCOMES

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

1. Discuss the anatomy and physiology of the cardiac muscle in relation to how anti-arrhythmics and vasoactive medications affect its function.
2. Discuss the mechanism by which the renal system functions in relation to diuretic therapy.
3. Discuss the bleeding-clotting cascade in relation to anticoagulant medications.

SUMMARY OF KEY POINTS

- I. Review of anatomy and physiology of the cardiac system
 - A. Review of the determinants of cardiac output
 1. Preload
 2. Heart rate
 3. Contractility
 4. Afterload
 - B. Cellular physiology: How medications affect the cell's action potential
 1. Resting state
 - a. Na⁺ more concentrated extracellularly
 - b. K⁺ more concentrated intracellularly
 - c. Cell membrane highly permeable to K⁺
 2. Depolarization
 - a. When K⁺ diffuses out of cell, Na⁺ rushes into cell muscle
 - b. To maintain contraction, Ca⁺ rushes inside cell and extends depolarization
 3. Repolarization
 - a. When Ca⁺ stops rushing into cell, muscle relaxation begins
 4. Action potential

- a. Cell properties
 - (1) automaticity
 - (2) refractory periods
 - b. Types of cells
 - (1) Mechanical
 - (2) Electrical
 - (3) Concept of automaticity
 - (4) Refractory periods
 - c. Arrhythmias
 - (1) Causes
 - (2) Pathogenesis
 - (3) Therapy and drug classifications
 - d. Nursing concerns/issues
- II. Hemodynamics and vasoactive medications
 - A. Stroke volume and heart rate
 1. Slowers – decreased chronotropy; decreased conduction velocity
 - a. Class I – slows down movement of Na⁺ into cell
 - b. Class II – inhibits Phase 4 depolarization (beta-blockers)
 - c. Class III – prolongs repolarization
 - d. Class IV – blocks Ca⁺ channels
 - e. Digoxin – inhibits Na⁺/K⁺ pump allowing the influx of Ca⁺ into cells; Ca⁺ prolongs depolarization
 - f. Adenosine – slows down AV node conduction and inhibits reentry pathways
 2. Speeders - increased chronotropy; increased conduction velocity
 - a. Adrenergic reception – release and use of epi/norepi: cells will contract/constrict or relax/dilate
 - b. Sub types:
 - (1) Alpha 1 – constriction of blood vessels vasodilation of skin and GI
 - (b) Alpha 2 - manipulation of cyclic AMP inside cell
 - (c) Beta 1 – acts on cardiac pacemaker = increased HR
 - (d) Beta 2 - vasodilation of skeletal/bronchial muscles
 - c. Atropine Sulfate – blocks vagal effects on SA and AV nodes = in turn enhances AV node conduction and increases HR
 - d. Isoproterenol – Beta 1 and Beta 2 effects
 3. Squeezers - Inotropic drugs – increases ventricular contractility
 - a. Digoxin

- b. Dobutamine
- c. Amrinone/Milrinone
- d. Norepinephrine
- e. Dopamine
- f. Epinephrine
- 4. Unloaders – preload reducer, vasodilator, anti-hypertensive, ACE inhibitor, ARB
 - a. NTG
 - b. Nipride
 - c. Alpha/beta adrenergic blockers
 - d. ACE inhibitors
 - e. Angiotensin receptor blockers
- III. Diuretic therapy
 - A. Physiologic and hormonal controls on the renal system.
 - 1. Thirst mechanism
 - 2. ADH release
 - 3. Renin-angiotensin system activation
 - B. Classification of diuretics
 - 1. Carbonic anhydrase blockers – promotes renal excretion of Na⁺, K⁺, bicarb and water
 - 2. Thiazide diuretics – inhibits Na⁺ and Cl⁺ reabsorption, increases Na⁺ and water excretion
 - 3. Loop diuretics – inhibits Na⁺ and Cl⁺ reabsorption
 - 4. Osmotic diuretics – inhibits reabsorption of water and lytes
 - 5. Potassium-sparing diuretics – increases Na⁺ and water excretion
 - C. Nursing concerns/issues

- IV. Thrombolytic therapy
 - A. Review of clotting cascade
 - 1. Intrinsic pathway
 - 2. Extrinsic pathway
 - B. Thrombolytics
 - 1. Common agents used
 - 2. Reperfusion phenomena
 - C. Thrombolysis in CVA due to embolic phenomena
 - 1. Common agents used
 - D. Nursing issues/concerns

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Street Drugs and Overdose: Do Your Kids Know More Than You?

Andrea Efre

Level: Beginner

CONTENT DESCRIPTION

The purpose of this presentation is to provide up to date information on street drugs and discuss treatment options in the management of the patients who use them. We will also discuss overdoses of both street drugs, prescription medications and over the counter medications and the treatment options in managing the overdose.

For centuries drugs have been a part of history. Each cultural group found their own way of making and using recreational drugs, and each generation believed they were unique in their drug use. Many great musical accomplishments were created while the artists enjoyed hallucinogenic drugs of the decade. However, if you are practicing currently, you need knowledge of the drugs used today. For example, the assessment of an unconscious teenager not only involves the ABC's, but may also include a rectal exam. Why you ask? Because a new method of use for Gamma hydroxybutyrate (GHB) is to soak tampons in the drug and insert rectally or vaginally, allowing for a longer absorption period.

This presentation will review the most commonly used street drugs including cocaine, heroine, methamphetamine, opioids, hallucinogenic agents and designer drugs such as ecstasy and GHB. Participants will have the opportunity to examine and handle drug paraphernalia and to see how everyday items can become containers for drug storage. We will discuss signs and symptoms, assessment and treatment options of suspected drug use, withdrawal or overdose. Street drugs affect multiple patient populations and therefore this presentation is a must for all health care professionals including students, nurses, advanced practice nurses and physicians. The only prerequisite for this program is an open mind, as some of the information can be shocking. Knowledge is power. You must be armed with as much information as possible on the multiple drugs available and how they are used, so that you are prepared for all possibilities.

LEARNING OUTCOMES

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

1. Identify the most commonly used street drugs, and the culture surrounding each drug.
2. Recognize types of paraphernalia needed to use the drugs and identify types of drug storage containers
3. List signs and symptoms of suspected drug use, withdrawal or overdose and discuss treatment options for each.

SUMMARY OF KEY POINTS

- I. Cocaine
 - A. Cutting: Often mixed/cut to make the drug less potent

- B. History of Cocaine
 - C. Crack Cocaine
 - D. Freebasing - Cocaine into Crack: All you need is:
 - E. Crack Pipes
 - F. Signs of a Long Term Cocaine or Crack User
 - G. "Speedball" or "Moonrock"
 - H. Cocaine with Cannabis - Combined in a "joint" or "blunt"
 1. Body Packers (mules)
 2. Body Stuffers
 - I. Signs of Cocaine Overdose
 - J. Cocaine and its Cardiac Implications
 - K. Diagnosis
 - L. Treatment/Management
 - M. Medications
 - N. Medications to Avoid
 - O. Consultations
- II. Heroin
 - A. Cutting of Heroin - Other substances commonly used to increase the bulk:
 - B. Short Term Effects
 - C. Long Term Effects
 - D. Signs to look for: Signs of Overdose
 - E. Diagnosis
 - F. Treatment and Management
 - G. Withdrawal
 - H. Methadone
 - III. OxyContin
 - A. Signs of Overdose
 - B. Treatment and Management
 - C. Opioid Withdrawal look for:
 - IV. Club Drugs" - a general term for a number of illicit drugs, primarily synthetic, that are most commonly encountered at nightclubs and "raves."
 - V. Metamphetamine
 - A. Short Term Effects
 - B. Long Term Effects
 - C. Signs of use or Overdose
 - D. Treatment and Management
 - VI. Ketamine
 - A. Date Rape Drug
 - B. Signs of use or Overdose
 - VII. LSD
 - A. Signs of use or Overdose
 - VIII. Ecstasy (MDMA).
 - A. Symptoms of use
 - B. Symptoms of Overdose
 - C. Treatment and Management

- IX. Rohypnol (Flunitrazepam)
 - A. Signs of use or Overdose
- X. GHB - Gamma hydroxybutyrate
 - A. There are more than 80 known names for GHB
 - B. Signs of use or Overdose
 - C. Treatment and Management for Overdose of Club Drugs
- XI. PCP (Phencyclidine)
 - A. Signs of use or Overdose
- XII. Wetstick
 - A. Are marijuana mixed with embalming fluid and laced with PCP
- XIII. Psilocybin Mushrooms
- XIV. Peyote and Mescaline
- XV. Inhalants
 - A. Four Categories:
 - 1. Volatile Solvents
 - 2. Aerosols
 - 3. Gases
 - 4. Nitrites
 - B. Uses:
 - 1. Sniffed
 - 2. Snorted
 - 3. Huffed
 - 4. Bagged
 - C. Aerosols: (Propellants and solvents)
 - D. Gases: (Medical, Household and Commercial)
 - E. Nitrites: often considered a special class of inhalants.
 - F. Short Term Effects of Using Inhalants:
 - G. Long Term Effects of Using Inhalants:
 - H. "Sudden Sniffing Death"
 - 1. Withdrawal
- XVI. KHAT (also known as Catha Edulis)
 - A. Khat has over 40 street names:
 - B. Signs of Use or Overdose
- XVII. Other medications used for Overdosing
 - A. Prescription medications often used
 - 1. Tricyclics
 - 2. Any antidepressants
 - 3. Analgesics
 - 4. Sedatives and sleep aids
 - 5. Beta-blockers
 - 6. Calcium channel blockers
 - B. Non prescription medications often used
 - 1. Tylenol
 - 2. Aspirin
 - 3. Ibuprofen
 - 4. Cold medicines
 - 5. Coricidin (skittling)

- XVIII. Determining Use and Overdose
 - A. Look for clues –
 - 1. Paraphernalia
 - a. Shake a bottle of water to test for GHB
 - b. Look for empty soda cans (with holes in)
 - c. Does anything look like a pipe?
 - d. Candy, Bubbles, glow-sticks
 - 2. Get the whole story
 - 3. Talk to everyone involved
 - 4. Friends may help if they understand the seriousness
- XIX. Smuggling: The new smuggling trick: Aquafina water bottles with a hidden compartment for drugs.
- XX. Overdose on Street Drugs
- XXI. Current Trends in the Management of Overdose
- XXII. Overdose General Management
- XXIII. Summary

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Stress-Induced Cardiomyopathy: Mending a Broken Heart

Kiersten Henry

Level: Beginner

CONTENT DESCRIPTION

The purpose of this session is to explore the pathophysiology and management of Stress-Induced Cardiomyopathy (also known as transient left ventricular apical ballooning or “Broken Heart Syndrome”). The diagnosis and management of stress-induced cardiomyopathy will be differentiated from that of acute myocardial infarction.

LEARNING OUTCOMES

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

1. Differentiate between stress-induced cardiomyopathy and acute myocardial infarction.
2. Discuss possible causes of stress-induced cardiomyopathy and strategies to manage patients with stress-induced cardiomyopathy.
3. Identify medications used to manage stress-induced cardiomyopathy.

SUMMARY OF KEY POINTS

- I. Patient Presentation- Case Study
- II. Stress-Induced Cardiomyopathy vs. Acute Myocardial Infarction (Table 1)
- III. Management of Stress-Induced Cardiomyopathy

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Students in Critical Care: Transforming Students into Colleagues

Terry L. Tucker

Level: Expert

CONTENT DESCRIPTION

Senior nursing students can be crafted into successful critical care nurses if skillfully introduced into the domain of critical care and then mentored in learning and developing critical skills that tap into their talents, curiosity and enthusiasm. A career-defining experience in a critical care clinical rotation can be enhanced by the partnering of the goals of the course curriculum with the available resources of the hospital and clinical unit. Should the student decide to join the critical care unit as a full-fledged staff member after graduation, the learning and development begun as a student can continue and grow in a familiar and comfortable environment? The purpose of this session is to profile and illuminate how a student in critical care can be transformed into a skilled colleague. A review of strategies to engage the student in clinical development activities as well as strengthen cognitive and psychomotor skills will be presented. Strategies for resource people such as the preceptor, Critical Care CNS and Nurse Manager will be provided to guide the resource team in providing effective clinical learning experiences and in evaluating student/colleague skill performance and knowledge retention. Finally, a review of the importance of socialization of the student in the critical care environment will be reviewed. Knowledge of the roles of preceptor, critical care educator and/or Critical Care CNS and Nurse Manager will be beneficial to appreciate the nuances of transforming students into colleagues.

LEARNING OUTCOMES

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

1. Discuss areas of critical care knowledge and skills that correlate with the student's school course curriculum, the student's interests, current knowledge and areas needing development.
2. Describe activities that can actualize a student's potential in the critical care setting and encourage the student to move into the full professional role of critical care nurse.
3. Discuss orientation and development activities that will transform the student into a skilled and competent colleague.

SUMMARY OF KEY POINTS

- I. Looking at the student, the curriculum and the clinical rotation
 - A. Why students in critical care? How did we get to this point?
 1. Trends in student development and placement
 - a. Acknowledging the diversity among today's students

- b. Exploring expectations
 - c. Interviewing the student to make the best match
 2. Critical care: a buyer's market for the student
 - a. The facts and myths about loyalty
 - b. Developing transportable skills sets
 - B. Looking at the school course curriculum
 1. What it is and what it isn't
 2. Correlating the student's course objectives with the critical care unit's mission
 - a. Reviewing curriculum goals
 - b. Evaluating evaluation tools
 - c. Examining the school's clinical resources
 - C Enhancing the clinical rotation
 1. Selecting the right preceptor for the best fit
 2. Transferring the cognitive domain into psychomotor skills
 - a. Developing and honing critical thinking skills
 - b. Correlating lecture content with unit clinical activities
 - c. Enhancing the clinical learning environment
 - II. Actualizing the student's potential utilizing the best unit resources
 - A. Precepting, mentoring and coaching: influential relationships
 1. Key components of each influence
 2. Examining the roles and influence of the preceptor, Critical Care CNS and Nurse Manager
 - B. Scheduling student clinical time to optimize opportunities
 1. Selecting clinical situations to strengthen clinical skills
 - a. Tapping into technology
 - b. Clinical observation and simulation activities
 - c. Looking at patient load and care requirements
 - d. Planning patient assignments
 - e. Integrating required unit activities
 2. Promoting participation in other unit and organizationally-based activities
 - a. Importance and influence of unit activities
 - b. Involvement in organizational events
 3. Acknowledging and working with the affective domain of critical care nursing
 - a. Dealing with the stress of critical care
 - b. Decompression and restorative activities
 - III. Facilitating the transition from student to colleague
 - A. Beginning the journey from Novice to Expert
 1. The influence of fellow staff

- a. The socialization process in the critical care setting
- b. Securing and capitalizing on mentoring relationships
- 2. Initiation into the “professionalism” of nursing
 - a. Importance of professional association involvement
 - b. Beginning professional activities
- 3. Establishing a culture of learning: setting a course for career success
 - a. The value of journal-keeping
 - b. Re-examining values, career goals and dreams
- B. Looking to the future: “Re-timing” your timeline
 - 1. The paradigm shift with graduate education
 - 2. Contemplating academic involvement: writing, teaching and publishing
 - 3. On the horizon: reversal and expansion of roles
- IV. One unit’s success story

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Successful Precepting-Communication and Emotional Intelligence

Mitzie Trammel
Sandy Hunter

Level: Beginner

CONTENT DESCRIPTION

Communication between two people is an important component to successful orientation, employment, and work relationships. Communication involves sending a message and receiving a message. The effectiveness of communication may be influenced by the communication method used, and the emotional state of the individuals involved. Emotional intelligence represents personal competence, which is the ability to manage oneself, and social competence, which is the capacity to relate to others. Emotionally intelligent preceptors help others to succeed by sharing information, support and resources.

Communication strategies such as feed forward and feedback will provide clear direction for the orientee and optimize teachable moments. Written communication methods include documentation of daily performance and periodic evaluation by the educator or unit nursing leader. The purpose of this session is to provide effective communication strategies for nurses who precept employees. It will also provide the nurse with information about emotional intelligence, and its effect on communication. This session is intended for all critical care nurses who precept employees on their units. No prerequisite knowledge is required for the participants. Participants will be able to utilize successful communication strategies when precepting employees.

LEARNING OUTCOMES

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

1. Describe the factors influencing communication
2. Identify and apply effective communication strategies to Precepting
3. Define and apply Emotional Intelligence to Precepting

SUMMARY OF KEY POINTS

- I. Factors Influencing Communication
 - A. Purpose of communication
 - B. Methods of communication
 - C. Emotional State
- II. Effective Communication Strategies
 - A. Feed back- focus on past events
 - B. Feed forward- focus on future events
 - C. Teachable moments
 - D. Documentation of daily performance
 - E. Written evaluation
- III. Emotional Intelligence and how it impacts communication
 - A. Self-Awareness
 - B. Self-Regulation
 - C. Self-Motivation
 - D. Social Awareness
 - E. Social Skills

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The Sugar High: Caring for the Child with Diabetic Ketoacidosis

Ruth M. Lebet
Patricia A. Moloney-Harmon

Level: Intermediate

CONTENT DESCRIPTION

This session, geared toward nurses caring for critically ill children in an Emergency Department or ICU setting, will discuss the changing epidemiology, initial presentation, key concepts in treatment based on current evidence, and potential complications of Diabetic Ketoacidosis in the child. Case study format will be used to review the presentation and treatment course of several patients, providing an opportunity for discussion of patient management and family process.

LEARNING OUTCOMES

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

1. Review the pathophysiology of DKA, including potential complications and identify ways in which DKA presentation and illness trajectory differs in the child
2. Identify laboratory findings most helpful in assessing the effectiveness of DKA treatment
3. Discuss the most current research regarding effective treatment of DKA

SUMMARY OF KEY POINTS

- I. Epidemiology
 - A. Initial diagnosis
 1. Age
 2. Type 1 vs Type 2
 - B. Incidence
 - C. Causes
 - D. Mortality
- II. Pathophysiology
 - A. Early evolving DKA
 1. Type 1 vs Type 2
 - B. Progressive DKA
 1. Hormonal interactions
 - a. stress hormones
 - b. growth hormone
 - c. cortisol
 2. Insulin secretion: Type I vs Type II
 3. Physiologic effects of progressive DKA
 - a. hyperglycemia
 - b. osmotic diuresis
 - c. ketoacidosis
 - d. peripheral insulin resistance
 - e. acidosis
- III. Clinical Presentation
 - A. Differential diagnoses
 - B. Classic presentation
 1. Three Ps

2. Abdominal symptoms
 3. Respiratory exam
 4. Mental status
- C. Clinical issues
 1. Dehydration
 2. Acidosis
 3. Ketosis
 4. Electrolyte abnormalities
 - a. Glucose
 - b. Potassium
 - c. Sodium
 - d. Chloride
 - e. Calcium
 - f. Phosphate
 - g. Magnesium
 - D. Management
 1. Fluid administration
 - a. Resuscitation
 - b. Maintenance
 2. Insulin administration
 3. Dextrose administration
 4. Electrolyte replacement
 5. Laboratory studies
 - a. Blood glucose monitoring
 - b. pH monitoring
 - c. Osmolarity
 - d. Anion gap
 - E. Complications
 1. Hypoglycemia
 2. Hypokalemia
 3. Cerebral edema
 - a. Pathophysiology
 - b. Treatment
 - (1) Medications
 - (2) Ventilation control
 4. Pulmonary edema
 5. CNS thrombosis
 6. Cardiac arrhythmia
 - a. prolonged QTc
 7. Renal failure
 8. Mucormycosis
- IV. Goals of management
 - A. Initial phase
 - B. Transition phase
 - V. New technologies
 - A. Home Insulin pumps
 - B. Home blood glucose monitoring

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Surviving Sepsis: No Missed Opportunities

Barbara "Bobbi" Leeper

Level: Intermediate

CONTENT DESCRIPTION

Sepsis is a process of acute inflammation out of control resulting in severe morbidities and increased mortality rates. Severe sepsis and septic shock have been identified at the most common cause of death in a critical care unit. Experts estimate the incidence of severe sepsis will continue to increase contributing increased morbidity and mortality rates. The purpose of this presentation is to describe the disease continuum of sepsis and the sometimes subtle indicators of SIRS progressing on to sepsis and severe sepsis. This presentation will include a review of the impact of sepsis on patients, the healthcare economy as well as hospitals. The pathophysiology of normal responses to infection as well as sepsis progressing to SIRS, severe sepsis and subsequently septic shock will be discussed. The clinical presentation will be reviewed. The management of severe sepsis including important medical and nursing interventions including the importance of early goal directed therapy will be reviewed. The Institute for Healthcare Improvement (IHI) bundles for sepsis as well as AACN's Practice Alert on Sepsis will be presented. A case study demonstrating the impact of early goal directed therapy will be used to exemplify severe sepsis. Nursing has a unique opportunity to identify patients with signs of infection and/or signs of organ dysfunction for the purpose of implementing sepsis protocols early on, and thereby improving patient outcomes. The target audience for this presentation is nurses working in progressive care / telemetry and critical care. There is no pre-requisite knowledge needed for this session.

LEARNING OUTCOMES

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

1. Differentiate between SIRS, sepsis and severe sepsis.
2. Discuss the important nursing aspects when caring for a patient with severe sepsis and septic shock.
3. Analyze a case study demonstrating Early Goal Directed Therapy.

SUMMARY OF KEY POINTS

I. Introduction

A. Sepsis: Defining a Disease Continuum

1. Infection / Trauma
2. Systemic Inflammatory Response Syndrome (SIRS)
 - a. A clinical response arising from a nonspecific insult, including 2 or more of the following:
 1. Temperature $\geq 38^{\circ}\text{C}$ or $\leq 36^{\circ}\text{C}$
 2. HR of 90 or more beats/min
 3. Respirations of 20 or more/min
 4. WBC count of 12,000/mm³ or greater OR less than 4,000/mm³

5. 10% immature neutrophils
3. Sepsis: SIRS with a presumed or confirmed infectious process
4. Severe Sepsis
 - a. Sepsis with ≥ 1 sign of organ failure
 1. Cardiovascular (refractory hypotension)
 2. Renal
 3. Respiratory
 4. Hepatic
 5. Hematologic
 6. CNS
 7. Unexplained metabolic acidosis

B. The Problem

1. More than 750,000 cases occur annually
 - a. Challenges:
 - (1) Spreads quickly
 - (2) Often difficult to recognize
 - (3) Mortality rate 28% to 50%
 - b. Onset of multiple organ failure & mortality rates:
 - (1) 1 organ = 20%
 - (2) 2 organs = 40%
 - (3) 3 organs = 65% - 70%
 - (4) 4 organs = 75% - 80%
2. Incidence projected to increase!
3. Financial Impact
 - a. Severe sepsis costs: average \$22,000 / pt
 - b. Total costs to US hospitals: \$16.7 billion
 - c. Cost of treating sepsis is 6 times greater than a non-septic patient

C. Surviving Sepsis Campaign

1. Phase I: October 2002
 - a. Increase education
 - b. Establish practice guidelines
2. Phase II: 2003
 - a. International healthcare groups
 - b. Establish consensus opinion based on the evidence
 - (1) Improve survival rates by 25% within 5 years
 - (2) Reduce costs
3. Phase III: Sepsis Bundle – Implement
 - a. Re-evaluate in 2008

II. Pathophysiology of Sepsis

- A. Sepsis is overwhelming inflammation activation of the stress response
 1. 4 Hormones are produced to increase gluconeogenesis
 - a. Cortisol
 - b. Epinephrine

- c. Growth Hormone
 - d. Glucagon
 - 2. Laboratory signs of inflammation
 - a. Elevated neutrophils
 - b. Increased bands
 - c. Elevated WBC's
 - B. Pathophysiology – Body's normal response to infection:
 1. Antigen enters the blood stream
 2. Immune system releases proinflammatory mediators to clear the antigen and promote recovery of affected tissue
 - a. TNF alpha
 - (1) injures the endothelial lining
 - (2) prevents utilization of glucose
 - b. Interleukin 1: causes vasodilation, hypotension, fever, increased vascular permeability, sleepiness, anorexia, myocardial depression, hypercoagulability
 - c. Interleukin 6: causes fever, increased cortisol production
 3. Proinflammatory mediators attract neutrophils which engulf the antigen
 4. Initiation of coagulation cascade leading to clot formation to isolate the antigen
 - a. Platelet activation
 - b. Thrombus formation
 5. Fibrinolysis is inhibited to provide time for the antigen to be destroyed
 6. Anti-inflammatory mediators are released to:
 - a. Protect the normal surrounding tissue
 - b. Restrict the inflammatory response to the local site of infection
 - C. Sepsis
 1. Balance is upset
 2. Immune response not localized becomes systemic
 3. Results:
 - a. Excess coagulation
 - b. Exaggerated inflammation
 - c. Impaired fibrinolysis
 4. Endothelial disruption
 - a. Platelet activation, deposition & aggregation
 - b. Platelets bind to fibrinogen
 - c. Fibrin is trapped
 - d. Final result is the thrombus
 5. Immune system imbalance supports coagulation
 - a. Platelet activation
 - b. Increased generation of thrombin
 - c. Increased deposition of fibrin
 - d. Progresses to:
 - (1) Microvascular hypoperfusion
 - (2) Diminished oxygen delivery
 - (3) Tissue necrosis
 6. Problem is not in the arteries but in the capillaries where cellular gas exchange occurs
 7. Process similar to ACS, but with ACS, clotting is localized...with sepsis it is everywhere!
 - 8. Look for early coagulation signals
 - a. Thrombocytopenia
 - b. Elevated fibrin degradation products or d-Dimers
 - c. Decreased fibrinogen levels
 - 9. Thrombin is pro-coagulatory
 - a. Usually controlled by thrombomodulin
 - (1) Released from endothelium
 - (2) Binds to thrombin
 - (3) Expresses protein C
 - (a) Profibrinolytic
 - (b) Antineutrophil
 - (c) Anti-inflammatory
 - (d) Antithrombotic
 - b. Severe Sepsis: thrombomodulin levels decrease leading to lesser amounts of protein C
 - (1) Protein C deficiency is a constant throughout sepsis
 - (2) End point is pro-coagulation
 - 10. Subsequently:
 - a. Organ failure occurs
 - b. Progression to severe sepsis
 - c. Procoagulant state causes more endothelial damage
 - d. Vascular damage causes release of more neutrophils and inflammatory cytokines
 - e. Self-perpetuating chain of events
 - D. Signs of Inflammation out of control
 1. Hemodynamics
 - a. Arterial hypotension
 2. General Signs / Symptoms
 - a. Fever / hypothermia
 - b. Heart rate > 90/min
 - c. Tachypnea
 - d. Altered mental status
 - e. Significant fluid imbalance
 - f. HYPERGLYCEMIA
 3. Looking for signs of organ dysfunction
 - a. Arterial hypoxemia
 - b. Acute oliguria
 - c. Increased creatinine
 - d. Coagulation abnormalities
 - e. Ileus
 - f. Thrombocytopenia
 - g. Hyperbilirubinemia
 4. Looking at the Inflammatory variables
 - a. Leukocytosis
 - b. Leukopenia
 - c. Normal WBC > 10% bands
 - d. Elevated C reactive protein
 - e. Elevated procalcitonin
 - f. Decreased PROTEIN C levels
 - g. Decreased Platelets
- III. Who is at Risk?
- A. Causes not completely understood
 - B. Increased risk associated with:

1. Trauma to GI tract
 2. Perforation of small bowel / Intra-abdominal sepsis
 3. Splenectomy
 4. IV drug abuse
 5. Surgery
 6. Chronic conditions (ex: diabetes)
 7. Use of invasive procedures and devices in the ICU
 8. Immunosuppression therapy
- IV. Lessons Learned
- A. Improved survival rates from early identification and early intervention
 - B. Key Nursing Aspects
 1. Astute clinical assessment for:
 - a. Clinical signs of infection
 - b. Onset of organ dysfunction
 2. Temp > 38°C reported promptly
 3. Monitor:
 - a. Wound drainage
 - b. Catheter insertion sites
 - c. Mouth
 - d. Skin
 4. Monitor labs:
 - a. Leukocytosis or leukopenia
 - b. Increased immature neutrophils (“bandemia”)
 - C. A Question of Timing
 1. Early recognition of patients at high risk
 2. Two or more signs of SIRS
 - a. Increased or decreased body temperature
 - b. Increased resp rate
 - c. Decreased PaCO₂
 - d. Increased Heart Rate
 - e. Elevated WBC
 3. Signs of occult tissue hypoxia
 - a. Elevated serum lactate
 - b. Decreased systolic BP
 4. Think in terms of “global tissue hypoxia,” i.e., who may have it. Any patient identified with these, should be treated as likely to have sepsis
 - D. Other Facts
 1. Septic shock present if:
 - a. Failure to respond to fluid resuscitation
 - b. Demonstrates perfusion abnormalities
 - c. Caveat: more chemical mediators are being released and causing more damage
 2. Remember: severe sepsis present if:
 - a. Dysfunction of one organ
 3. Patient being treated for pneumonia suddenly becomes worse or develops:
 - a. Abrupt drop in urine output
 - b. Requires mechanical ventilation
 4. Patient receiving antibiotics requires a vasopressor
 - a. Should be a signal for evaluation of sepsis
 - b. Vasopressor therapy associated with higher mortality rates

- V. Management of Sepsis
- A. Standard care
 1. Source control
 2. Antibiotics
 3. Hemodynamic support
 4. Mechanical ventilation
 5. Renal replacement therapy
 6. Sedation/analgesia
 7. Ensure adequate nutrition
 8. Provide hematological support
 9. Other supportive measures
 10. Xigris®: Decreases inflammation and coagulation; promotes fibrinolysis
 - B. It’s all about the tissues!
 1. Consider monitoring SvO₂ or ScvO₂
 - C. Monitoring ScvO₂
 1. What is ScvO₂?
 - a. Fiber optics have been incorporated into central venous catheter providing ability to monitor the O₂ saturation in the superior vena cava.
 - b. ScvO₂ vs SvO₂: Difference between the two:
 - (1) ScvO₂ is consistently higher by 5-13% with an average of 7.5%
 - (2) A ScvO₂ of 70% implies that SvO₂ is likely to be 60 – 65%
 - (3) ScvO₂ has been shown to trend with SvO₂
 2. Early Goal Directed Therapy: Rivers E.
 - a. Randomly assigned pts arriving in the ED with sepsis or septic shock to one of 2 groups:
 - (1) 6 hours of EGDT or
 - (2) Standard therapy
 - b. ICU clinicians were blinded
 - c. 263 patients enrolled
 - (1) 130 assigned to EGDT
 - (2) 133 to standard therapy
 - d. Results
 - (1) EGDT group in-hospital mortality = 30.5% as compared to 46.5% in the standard therapy group
 - (2) EGDT group had:
 - (a) Significantly higher ScvO₂
 - (b) Lower se lactate level
 - (c) Lower base deficit
 - (3) EGDT group had lower APACHE II scores & shorter hospital LOS
- VI. Case Study: Early Goal Directed Therapy
- 85 y/o male with Hx hypertension, ASHD and CHF. Presents with a cough, SOB that started while making breakfast
- He became ill while visiting wife in the hospital.... went to the ED from the hospital floor
- Physical Exam:
- Temp: 34.5°C; HR 84; BP 150/98; RR 28
- Mild JVD at 30°, dry mucous membranes
- Crackles in left lung base
- CV: regular rate / rhythm, no murmurs
- ABD: mildly distended; tympanic below umbilicus

Rectal: very enlarged prostate
“Up to 50% of patients resuscitated from shock may have continued global tissue hypoxia” E. Rivers, MD

A. Other management considerations

1. Serum Glucose control
2. CRRT to assist with volume to assist with fluid management
3. Bicarbonate therapy has not been proven to be beneficial.
4. CI/O_2ER Ratio
 - a. Normal = 12
 $CI = 3; O_2ER @ 25\%$
 - b. Ratio less than 10 indicates inadequate CV response, i.e., increased oxygen extraction
 - c. In septic shock, a ratio less than 10 may indicate myocardial depression

VII. Sepsis Bundles: www.ihl.org

A. 6 Hour Sepsis Resuscitation Bundle -

1. Tasks that should begin immediately
 - a. Serum lactate measured
 - b. Blood cultures prior to antibiotic therapy
 - c. Broad spectrum antibiotic started within:
 - (1) 3 hours if ED admission
 - (2) hour if in-house
 - d. Treat hypotension or se lactate > 4 mmol/L with fluids
 - (1) minimum vol 20mL/Kg
 - e. Apply vasopressors for ongoing hypotension not responsive to volume to maintain MAP > 65mmHg
 - f. Maintain adequate CVP: administer volume to achieve CVP > 8
 - g. Maintain adequate $ScvO_2$: ($ScvO_2 > 70\%$)
2. 24 Hour Sepsis Management Bundle
 - a. Begin immediately
 - b. Can take 24 hours to accomplish
 - (1) Administer low dose steroids by a standard policy
 - (2) Administer drotrecogin alfa (activated) by a standard policy
 - (3) Maintain adequate glycemic control (< 150 mg/dL)
 - (4) Prevent excessive inspiratory plateau pressures (less than 30 cm)

VIII. AACN Practice Alert - Severe Sepsis

IX. Conclusions

- A. Sepsis is a significant healthcare challenge with major morbidity, mortality, and health economic implications
- B. Patients with severe sepsis (acute organ dysfunction) are at high risk for mortality
- C. Systemic inflammation is out of control, leading to coagulation and impaired fibrinolysis, which are key components of disordered homeostasis in patients with severe sepsis

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Swift Killer: Meningococcal Disease

Andrea M. Kline

Level: Advanced Practice

CONTENT DESCRIPTION

This session will review the latest in epidemiologic trends, evaluation and diagnosis of Meningococcal disease. Contemporary management strategies, applying evidence based medicine will be discussed. Affects of genomics and preventative strategies will be reviewed. Case study presentations will be used to illustrate the information discussed.

LEARNING OUTCOMES

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

1. Discuss latest epidemiologic trends in Meningococcal disease.
2. Identify risks for and appropriate prevention strategies for Meningococcal disease.
3. Discuss evidence based medicine in the management of Meningococcal disease.

SUMMARY OF KEY POINTS

- I. Review of Meningococcal Disease
 - A. Neisseria Meningitidis
 1. Bacteria
 - a. Gram negative dipococci
 - b. Frequent and asymptomatic colonizer of human respiratory tract
 - c. Ability to invade susceptible individuals
 - (1) Causing sepsis and meningitis
 - d. Glitza et al. 2007.
 - (1) German teenagers, 15-18 years of age
 - (2) Each swabbed 3 times with 2 month intervals
 - (3) Overall carriage rate of 18.8%
 - (4) Significant differences within given schools, towns, counties
 - (5) 60.6% not serogroupable, Group B dominated at 12.3%, Serogroup Y 9%, serogroup C 3.6%
 2. Transmission
 - a. Spreads across the epithelium
 - b. Requires expression of surface sialic acids
 - c. Both human and bacterial and host components have been implicated in susceptibility
 3. Incubation period
 4. Epidemiology
 - a. United States
 - b. World wide
 5. Pathophysiology
 - II. Genomics
 - A. Definition
 1. Role in Neisseria meningitidis

- III. Diagnosis
 - A. Gold standard
 - B. Rapid assays
- III. Management
 - A. Supportive care
 - B. Antimicrobial therapy
 - C. Potential adjunct therapies
- IV. Prevention
 - A. Vaccination
 1. Polysaccharide vaccine
 - a. Does not induce herd immunity
 - b. Limited effectiveness in children < 2 years of age
 2. Polysaccharide conjugate vaccine
 - a. Multivalent A, C, Y, W135
 - b. No vaccine for serogroup B available
 - c. Important effect on carriage
 - d. Effective in infants
 3. Outer membrane vesicle (OMV) vaccines
 - a. Disrupts serogroup B epidemics and outbreaks
 - 4 AICP and AAP recommendations
 5. Challenges with vaccination
 - B. Close contacts

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The Synergy Model: How to Make it Part of Your Daily Practice

John F. Dixon

Level: Beginner

CONTENT DESCRIPTION

AACN's Synergy Model for Patient Care has been in existence since the 1990's, but application of the model has yet to become widespread. The purpose of this session is to review the Synergy Model with a particular focus on strategies for integrating it into all aspects of daily practice and operations – professional practice models, management, education & orientation, clinical practice, research, and certification. Linkages between the Synergy Model and the Healthy Work Environment Standards and the 14 Forces of Magnetism will also be explored. Creation of a professional nursing practice model based on Synergy will be presented and how to use that model to set the vision and direction for practice and operations. Initiatives to be discussed include job descriptions and performance evaluations, behavioral interviewing, orientation and education design, precepting, professional nursing advancement programs, patient and population profiling, patient communication hand-offs, and certification exams and Synergy CERPs. This content is applicable to staff nurses, nurse managers, nurse educators, advanced practice nurses, and nursing faculty. No pre-requisite knowledge of Synergy is required to attend this session. Using Synergy as the driving force for practice and operations and implementing strategies discussed during this presentation will help to contribute to safe passage for patients, families, staff, and environments.

LEARNING OUTCOMES

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

1. Describe the application of the Synergy Model to patient care.
2. Identify two methods for incorporating Synergy into practice.
3. Begin typing here

SUMMARY OF KEY POINTS

- I. Describe the development of the Synergy Model
 - A. Work and research on which the Synergy Model is based
 - B. How Synergy reflects nursing practice
- II. The Synergy Model
 - A. Review the 8 characteristics with their definitions and levels for patients, units, and systems
 - B. Review the 8 competencies with their definitions and levels for nurses
 - C. Define what constitutes Synergy
 - D. Define Safe Passage

III. Strategies/Tools/Techniques for implementation in various spheres of practice and operations

- A. Professional practice models
- B. Management & operations
 1. Job descriptions
 2. Performance evaluation
 3. Behavioral interviewing
 4. Staffing
 5. Patient classification
- C. Education and orientation
 1. Integration into curricula
 2. Education activities and exercises
 3. Precepting
 4. Orientation exit goals
- D. Clinical practice
 1. Patient hand-offs - shift report, charge nurse report
 2. Professional nursing advancement programs
 3. Patient/population profiling
- E. Research
 1. Setting research agendas
 2. Linking to Safe Passage
- F. Certification
 1. Understanding the role of Synergy in certification exams
 2. Selecting educational offerings consistent with Synergy CERPs

IV. Synergy Linkages to Magnet and Healthy Work Environment

- V. Synergistic Nursing Practice
 - A. Making the shift to Synergy
 - B. Synergizing the 3 A's
 - C. Synergized Nursing Practice Outcomes

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Synergy on the Orient Express: Changing Priorities for Safer Outcomes

Sharon Gunn
Theresa Kaplan

Level: Beginner

CONTENT DESCRIPTION

Orientation of new graduates to critical care can be challenging for nurse educators, preceptors, managers and the new graduates themselves. Issues beyond just clinical aspects of orientation include the transition from student to practicing professional and organizational employee, socialization to the unit, and beginning the journey from novice to expert. After several months, the new graduate is expected to exit from orientation and be able to practice independently at baseline competency. We evaluated our orientation process looking for improvement opportunities to better meet this exit goal. Since our healthcare system's professional nursing practice model uses the Synergy Model as a foundational piece, we decided to "synergize" orientation by focusing on learnings and activities that were essential to and support Safe Passage. The purpose of this session is to share how we incorporated the Synergy Model into new graduate orientation in a 24-bed Medical ICU to include modifications made, new activities implemented, deleted activities, and associated documentation. Also presented will be lessons learned and how these changes have impacted the interactions between the nurse educator, preceptor, and the new graduate. Using Synergy as a framework for new graduate development helped to further contribute to safe competent nursing practice, collaborative care approaches, and a culture of inquiry where nurses continually question their practice. The content of this session is appropriate for all staff nurses, preceptors, nurse educators and managers. No prior knowledge of the Synergy Model or orientation processes is required.

LEARNING OUTCOMES

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

1. Describe the application of the synergy model to the orientation of new grads
2. Identify changes made to the orientation process that impacted safe competent nursing practice and collaborative care
3. Identify changes in the role of the preceptor, orientee and nurse educator that impacted orientation outcomes

SUMMARY OF KEY POINTS

- I. The Old Steam Engine - How orientation used to be:
 - A. Task oriented approach
 - B. Focus on time management
 - C. Focus on what you need to know
- II. Laying New Tracks - The Synergy Model and Orientation program
 - A. Baylor Healthcare System's Professional Nursing Practice Model

- B. The definition of Safe Passage
- C. Brief description of 8 core nurse competencies
- D. How development of these competencies were incorporated into an orientation tool
 1. Blended Synergy Model concepts with learning theories.
 2. Incorporated concepts inherent in AACN's Healthy Work Environment
- III. Creating New Modes of Travel - Strategies/Techniques for Implementation
 - A. Preceptor brainstorming/focus groups
 - B. Preceptor education
 - C. Weekly rounds with Preceptor/Orientee/Educator
 - D. Nursing Rounds focusing on Safe Passage practices
 1. Case Study
 2. Unsafe Room
 3. Communication Issues and patient handoffs
 4. Looking at the patient holistically
 - E. Constant contact with nursing education department
 - F. Creating methods to develop time management and increase exposure to procedures/interdisciplinary team
- IV. Railway Stops Along Our Journey - What the Outcomes were
 - A. Improved interactions with the interdisciplinary team
 - B. Improved self-confidence to ask questions about any aspect of nursing practice on our unit
 - C. Improved ability to independently access appropriate resources
 - D. Improved knowledge of bedside procedures, MD's to call
 - E. Improved communication between orientee, preceptor and educator
- V. New Railway Destinations – What's next?
 - A. Continued nursing rounds with focus on patient profiling
 - B. Day shift in the counts for 6 weeks following orientation
 - C. Welcoming committee/network night to facilitate socialization with members of the healthcare team
 - D. "Survivor Manual" with essential resource information necessary to succeed as an RN on the unit.

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The AACN Synergy Model. www.aacn.org

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Tackling Challenging Oncologic Complications in the ICU

Brenda Shelton

Level: Advanced Practice

CONTENT DESCRIPTION

The patient with cancer may be admitted to the ICU at the time of diagnosis, related to complications of therapy, or associated with progressive disease. With the availability of many successful anti-neoplastic therapies, many of these patients have the potential for prolonged disease-free survival with an excellent quality of life. This presentation will discuss outcome predictors for patients with cancer-related critical illness and provide a conceptual approach for management of the common symptoms of respiratory distress and abdominal pain. Critical care management of common oncology-related clinical complications include hemophagocytic syndrome, leukostasis, superior vena cava syndrome, tracheobronchial obstruction, and tumor lysis syndrome.

LEARNING OUTCOMES

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

1. Outline a method for assessing patients with cancer for prognostic variables that may predict outcomes of critical illness.
2. Describe the risk factors, clinical findings, and patient management for selected critical illnesses in patients with cancer.

SUMMARY OF KEY POINTS

- I. Overview of cancer patient outcomes with critical illness
 - A. Mechanical ventilation
 - B. Non-invasive ventilation
 - C. Dialysis
 - D. Cardiac arrest
- II. Treatment planning/ decision-making regarding critical care (Figure 1)
 - A. Type of cancer- hematologic malignancy versus solid tumor
 - B. Stage of cancer- newly diagnosed, during treatment, progressive disease, late effects
 - C. Cancer prognostic variables
 - D. Co-morbid health conditions
 - E. Reversibility of the crisis
 - F. Other pressing issues
- III. Common clinical problems
 - A. Respiratory distress
 1. Assess risk factors- cancer type, comorbid conditions
 2. Establish timing in relation to disease onset/ progression, treatment(s)
 3. Focus on infection risk
 4. Assess fluid balance

5. Rule out cardiac etiology
 6. Common diagnostic tests- Chest x-ray, Chest CT
 7. General interventions- bronchodilators, corticosteroids, antimicrobials, fluid restrictions, mechanical ventilation
- B. Abdominal pain
 1. Assess risk factors- cancer type, comorbid conditions
 2. Assess abdomen- location, pain characteristics, bowel sounds, fever, hypotension
 3. Common diagnostic tests- Abdominal x-ray, Abdominal CT, serum lactate, amylase, lipase, LDH
 4. General interventions- fluids, antimicrobials, surgical consult
- IV. Common complications requiring critical care support
 - A. Hemophagocytic syndrome
 - B. Leukostasis
 - C. Superior vena cava syndrome
 - D. Tracheobronchial obstruction
 - E. Tumor lysis syndrome

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The Tank is Half Full: Anemia's Impact on Chronic Diseases

Roxanne Garbez

Level: Intermediate

CONTENT DESCRIPTION

Anemia is observed in a myriad of chronic disease processes including congestive heart failure, renal failure, cancer and gastrointestinal conditions, and may contribute to increased morbidity and mortality. Knowledge of different types of anemias is invaluable to the understanding of these disease processes and medical management. Alterations in bone marrow and/or intra-cellular and extra-cellular processes related to RBC creation and maintenance within the vascular system is a common in chronic illness, or may be a consequence of medical management. Within the context of various case studies this session will focus on discussion of key concepts such as hematopoiesis, interpretation of a CBC result, diagnostic testing based on differential diagnosis, and management of selected chronic medical conditions associated with anemia. The purpose of this session is to describe the etiologies of select anemias and relate this to clinical understanding of differential diagnosis, diagnostic testing and medical management of specific chronic conditions. This session is targeted to experienced registered nurses.

LEARNING OUTCOMES

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

1. Examine hematopoiesis and its relationship to interpretation of a CBC result.
2. Analyze the relationship between anemia and selected chronic medical conditions.
3. Improve nursing practice outcomes through enhanced understanding of etiologies and management of anemia associated with chronic medical conditions.

SUMMARY OF KEY POINTS

- I. Hematopoiesis
 - A. Key concepts
 1. RBC components
 - a. Hemoglobin and Hematocrit
 - b. MCV
 - c. Cell Morphology
 2. Erythropoietin and renal function
 3. Intrinsic processes
 4. Extrinsic processes
 5. Mechanisms that alter RBC production
 - II. Types of anemia and related differential diagnoses
 - A. Microcytic
 1. Iron Deficiency
 2. Sideroblastic
 3. Thalassemia
 4. Lead poisoning

- B. Macrocytic
 1. Megaloblastic
 - a. Vitamin B12 deficiency
 - b. Folate deficiency
 - c. Drug induced
 2. Non-Megaloblastic
 - a. Advanced Liver Disease
 - b. Hypothyroidism
 - c. Splenectomy
- C. Normocytic
 1. Hemolytic
 - a. Intrinsic
 - b. Extrinsic
 2. Anemia of Chronic Disease
 3. Acute Blood Loss
 4. Mixed nutritional deficiencies
 5. Pregnancy
 6. Overhydration
 7. Endocrinopathies
 8. Liver Disease
 9. Aplastic Anemia

- III. Pathophysiology and management of anemia related to selected chronic conditions
 - A. Production of cytokines and leukotrienes
 - B. Alteration in renal function
 - C. Bone marrow suppression due to drug therapy
 - D. Transfusion
 - E. Congestive Heart Failure
 1. Erythropoietin and Iron
 - F. Renal Failure
 1. Pre-dialysis chronic disease
 - G. Cancer
 1. Erythropoiesis-stimulating proteins
 - H. Gastrointestinal conditions
 1. Identify underlying mechanism and customize treatment

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Targeting Sepsis: Strategies for Success

Ruth M. Kleinpell
Lynn Kelso

Level: Intermediate

CONTENT DESCRIPTION

Sepsis is a life-threatening condition that poses mortality risks for critically ill patients. The statistics regarding the incidence of sepsis are striking with reported rates of severe sepsis averaging up to 10 cases per 100 ICU admissions. Therefore, targeting sepsis is an important component in promoting best outcomes for critically ill patients. The purposes of this session are to present an overview of sepsis, highlighting nursing implications to promote early identification and treatment and to highlight successful nurse driven initiatives for targeting sepsis.

LEARNING OUTCOMES

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

1. Discuss important updates in sepsis care including evidence based treatment strategies
2. Discuss the use of nurse driven initiatives to target sepsis
3. Identify strategies for nurse driven initiatives aimed at early identification and treatment of sepsis

SUMMARY OF KEY POINTS

- I. Overview of Sepsis
 - A. Sepsis epidemiology: what's new?
 - B. Sepsis pathophysiology: the role of the immune system and genetics
 - C. Identification of sepsis in the clinical setting
- II. Focusing on Sepsis Identification
 - A. Screening for Sepsis
 - B. The SIRS criteria: how useful are they?
 - C. Implementing the Surviving Sepsis Campaign Guidelines: New Updates

- III. Strategies for Nurse Driven Initiatives Targeting Sepsis
 - A. Implementing sepsis protocols: factors to consider
 - B. Examples of strategies for nurse driven initiatives targeting sepsis
 - C. Showcasing improvements in sepsis care

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Therapeutic Humor Skills for a Healthy Work Environment

Scott C. Thigpen

Level: Intermediate

CONTENT DESCRIPTION

The goal of this session is to equip critical care nurses with therapeutic humor skills to strengthen healthy work environments.

Therapeutic humor has many roles in the unpredictable and uncontrollable health care environment. Therapeutic humor is an intervention that promotes health and wellness by stimulating a playful discovery, expression or appreciation of the absurdity or incongruity of life's situations. This intervention may enhance health or be used as a complementary treatment of illness to facilitate healing or coping, whether physical, emotional, cognitive, social or spiritual (Association for Applied and Therapeutic Humor). Humor can be used to get attention and gain control in situations filled with chaos. Therapeutic humor skills can serve as a tool for nurses who deal with professional problems, problem professionals, people problems and problem people. The physiologic benefits of humor include positive effects on the cardiovascular, pulmonary, immune, neurologic, endocrine, musculoskeletal, and gastrointestinal systems. A therapeutic laugh and left brain break can promote critical thinking, problem solving ability, and spark creativity. Humor can be used to add a dose of fun to the workplace. Patients, families and members of the health care team can benefit from a fun environment. Therapeutic humor skills can be used in groups to break the ice, promote healthy group dynamics, promote positive relationships, and shift the line in the sand. This presentation will focus on the five rights of humor administration. A review of literature will focus on research findings and implications for practice. A selection of props, play exercises, and games will be used to create a therapeutic humor environment. This session will provide an opportunity to have fun, identify resources and develop therapeutic humor skills through active participation. Laughter is a therapeutic communication skill that must be performed on a regular basis just like every other nursing skill to maintain competency. All participants will find this presentation to be fun as well as informative.

LEARNING OUTCOMES

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

1. Identify the roles of therapeutic humor in creating a healthy work environment.
2. Demonstrate therapeutic humor skills and identify resources available for creating a healthy work environment.
3. Evaluate the benefits of therapeutic humor skills in a healthy work environment.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Healthy Work Environments
- III. Therapeutic Humor – The Other Nursing Skill
- IV. The Mind-Body-Spirit Connection
- V. Therapeutic Humor Skills
- VI. Roles of Humor in Organization Culture
- VII. Humor, Creativity and Problem Solving
- VIII. Roles of Therapeutic Humor in Conflict Resolution
- IX. Therapeutic Humor – A Powerful Communication Tool
- X. Creating a Therapeutic Humor Environment
- XI. Discovering the “Fun” in “Dysfunction”
- XII. Advantages and Disadvantages of Laughter Programs
- XIII. Leading with Laughter
- XIV. Humor Resources
- XV. Prop Power

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- Jest for the Health of It, Patty Wooten, RN BSN. <http://www.jesthealth.com/>
- Karyn Buxman, RN, MSN, CSP, CPAE, Humorlab. <http://www.humorx.com/>
- Humor Therapy - Topic Overview. <http://www.webmd.com/balance/tc/humor-therapy-topic-overview>

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Therapeutic Hypothermia after Cardiac Arrest: Improving the Odds

Nicole Kupchik

Level: Beginner

CONTENT DESCRIPTION

Over 330,000 patients die each year from cardiac arrest. With increased availability of AEDs, early defibrillation and effective CPR, many patients are successfully resuscitated, but suffer permanent neurological damage. Without oxygen, brain death can occur in four to six minutes. Therapeutic hypothermia has been successfully instituted in many hospitals across the US, Europe and Australia. The purpose of this presentation is to review current research and discuss implications for critical care RNs caring for patients after cardiac arrest. A portion of the lecture will focus on the physiologic effects of anoxic brain injury. Benefits and adverse effects of hypothermia will be presented with key assessment points to observe ensuring safe patient care. Methods of cooling will be discussed as well as patient outcomes from our protocol. A case study will also be presented highlighting the use of therapeutic hypothermia from the time of arrest to induction of cooling to discontinuation and follow-up.

LEARNING OUTCOMES

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

1. Discuss the rationale & benefits for inducing hypothermia after cardiac arrest.
2. Identify potential risks and physiologic side effects associated with therapeutic hypothermia.
3. Describe steps for successful protocol development and implementation.

SUMMARY OF KEY POINTS

- I. History of cardiac arrest treatment and patient outcomes
 - A. Historical perspective of treatment strategies in cardiac arrest
 - B. Advanced in resuscitation
 - C. Outcome data
 1. American Heart Association
 2. Medic One Foundation
 - D. Early use of hypothermia
- II. Landmark Studies
 - A. Bernard trial-Australia
 - B. HACA trial- Europe
- III. Follow-up studies
 - A. Cooling in cardiogenic shock
 - B. Induction of cooling
 - C. Animal studies
 - D. Iced saline trials
- IV. Endorsement
 - A. ILCOR Advisory

- B. AHA ACLS Guidelines 2005
- V. Pathogenesis of anoxic brain injury
 - A. Phase 1: Complete ischemia
 - B. Phase 2: Neuroexcitotoxic cascade
 - C. Phase 3: Reperfusion injury
 - D. Benefits
 1. Decreased cerebral metabolic rate
 2. Decreased oxygen consumption
 3. Neuro-protection
 - E. Physiologic effects
 1. Electrolyte shifts
 2. Managing hyperglycemia
 3. Chronotropic
 4. Vasoconstriction
 5. Electrocardiographic changes
- VI. Methods of cooling
 - A. Early induction
 - B. Surface cooling
 - C. Catheters
 - D. Complications from overcooling
- VII. Nursing Considerations/Implications
 - A. Management of shivering
 - B. Monitoring complications of therapy
 - C. Temperature source
 - D. Digit oximetry
- VIII. Re-warming
 - A. Slow, controlled
 - B. New re-warming data
- IX. Protocol development and implementation
 - A. Education of nursing and physician staff
 - B. Common barriers
 - C. Evaluation of protocol therapy
 - D. Monitoring patient outcomes
 - E. Prognostication after arrest
- X. Case study
- XI. Web Resources:
 - A. www.therapeutichypothermia.com
 - B. www.med.upenn.edu/resuscitation/hypothermia
 - C. <http://hypothermia.uchicago.edu>

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The 3-Legged Stool: Research, EBP and Performance Improvement

Christine Hedges

Level: Beginner

CONTENT DESCRIPTION

Critical Care nurses are faced with a rapidly growing need to incorporate the best evidence to support clinical practice. With the explosion of information and emphasis on evidenced-based practice (EBP), nurses are challenged to practice according to best evidence in order to render excellent care. A critical skill for nurses today includes knowledge of the process of EBP, the distinction between EBP, research, and performance improvement (PI) including how they differ and interact; and knowledge of the skills needed to conduct an EBP investigation.

This presentation will discuss creative strategies used to implement EBP in an acute care hospital setting. Participants will learn to distinguish between PI, research, and EBP and discuss how they all support clinical practice. Participants will learn how to brainstorm clinical ideas, form clinical PICO questions and begin an EBP search, using examples from critical care and acute care NICHE settings. Basic concepts of data management will be discussed in terms of EBP. Links to Forces of Magnetism will be discussed.

LEARNING OUTCOMES

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

1. Describe a method for identifying clinical issues amenable to evidence-based practice investigation
2. Differentiate the processes of EBP, research and quality/performance improvement
3. Describe basic search strategies, data collection, evaluation and measurement techniques used in EBP and research

SUMMARY OF KEY POINTS

- I. Evidence Based Practice
 - A. Definition and purpose of EBP
 - B. Creating a culture of inquiry
 - C. Steps to get started with EBP in the hospital setting
 - D. Forming EBP Teams
 1. Role of EBP mentors and teams
 2. Examples from critical care and acute care NICHE units
 3. Formulating PICO questions
 - E. Formalizing the Process
 1. IRBs and research committee roles
 2. Systems support
 3. Role of hospital librarian

II. The foundations for practice and knowledge development: The 3 Legged Stool

- A. EBC model
- B. EBP
- C. Research
- D. PI/QI
- E. Clinical Examples
 1. Monitoring of Ventilator Associated Pneumonia (VAP)
 2. AACN Practice Alerts
 3. Use of Clinical Practice Guidelines

III. Resources for Evidence

- A. On-Line Resources and Scholarly databases
 1. Cochrane, DARE
 2. CINAHL, Medline
 3. EBP sites
 4. National Guideline Clearinghouse
- B. Baseline data collection and outcomes measures
- C. Data management basics
 1. Organizing Data
 2. Levels of Measurement
 3. Analyzing data
- D. Evaluating Data
 1. Evaluating results
 2. Examining strength and quality of evidence using hierarchy and scales
 3. Finding the right fit: "Will it work in my practice setting?"

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The Time Has Come: Ventricular Assist Devices for Heart Failure

Marcia Stahovich

Level: Beginner

CONTENT DESCRIPTION

The number of Ventricular Assist Devices (VADs) implanted is growing. The critical care nurse is in a unique position to educate chronic heart failure patients on options available to improve their quality of life, including VAD therapy, leading patients to seek healthcare centers that have VAD programs.

Over the past 20 years the VAD applications and technology have evolved dramatically. Originally pumps were used to salvage adult patients who failed to be weaned from cardiopulmonary bypass following routine cardiac surgical procedures. Pumps that were used in the early, failure-to-wean, experience were largely continuous-flow pumps (roller, centrifugal and axial flow). VADs can be broadly categorized as being either continuous flow (fluid dynamic) or pulsatile (volume displacement) and either can be used as short or long term support devices.

This course will begin with the history of devices, highlighting the different types of devices available including right ventricular assist devices (RVAD), left ventricular assist devices (LVAD), biventricular assist devices (BiVAD) which are FDA approved, in FDA clinical trials or future designs preparing to enter the market including their advantages and disadvantages. Indications for VAD use will be reviewed. Basic concepts of mechanical assist devices will be introduced including patient management strategies. Management of long term VAD patients involves community resources including home health nurses, cardiac rehabilitation centers and EMS who must be aware of the equipment and potential emergency procedures. Ethical issues of mechanical assist support including patient and societal issues, cost and quality of life will be presented. As centers gain more experience and referrals are made earlier in the disease process VAD patient care will be more streamlined and become the next treatment option for heart failure.

LEARNING OUTCOMES

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

1. Describe the evaluation process for a heart failure patient being evaluated for a VAD with the goal of Bridge to Transplant v. Destination Therapy.
2. Describe the difference between fluid dynamic (continuous flow) and pulsatile (volume displacement) pumps.
3. Discuss ethical issues the long term VAD patient and their caregivers will experience.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Heart Failure today

1. Patients being discharged from HF rose 175% from 1979-2004
2. Often sent home on inotropic therapy to hospice
- B. The future of Heart Failure
 1. Improved medications prolong life w/ HF
- C. The future of cardiac transplantation
 1. Patients may wait >1 year for donor heart
 2. >2800 patients waiting for a donor heart
- D. Stem cell therapy
 1. Improving in last 5 years as option
 2. Embryonic v. Adult stem cells
- E. VAD alternatives
 1. Failure to wean from CPB
 2. Implantable VADs become available
- II. Bridge to Transplant (BTT)
 - A. Prompted use of devices for DT
- III. Destination Therapy (DT)
 - A. Patients not meeting transplant criteria
 - B. REMATCH Study VAD v. OMM
- IV. Evaluation for VAD therapy
 - A. Inclusion/Exclusion Criteria BTT v. DT
 - B. Referrals from other centers to VAD center
- V. Short term v. Long term therapy
 - A. Short term (days to hours) < 2 weeks
 1. IABC
 2. Abiomed BVS 5000
 3. TandemHeart (percutaneous placement)
 4. Abiomed Impella
 5. CentriMag
 6. Biomedicus
 - B. Bridge to Recovery or Bridge to Bridge
- VI. Right, Left or Bi-Ventricular support
 - A. Heart is 2 pumps
 - B. Both sides fail Bi-VAD
- VII. Extra Corporeal Life Support (ECMO)
 - A. RN initiated team
- VIII. Bridge to Recovery
 - A. Weaning patients
 - B. Ventricular remodeling with rest
 - C. End organ recovery
- IX. Volume Displacement Pumps
 - A. Intermittant pulsatile flow
 - B. Valves and Bearings
 - C. Longevity
- X. Continuous Flow Pumps
 - A. Axial Flow Rotor in tube
 - B. Centrifugal flow Spinning cone (tornado)
 - C. Smaller patient size BSA <1.5

- D. Anticoagulation Coumadin, ASA
- XI. Nursing Care of the VAD Patient across the spectrum
 - A. Pre-op
 - 1. Nutritional support SI / SGA Infection – dental, podiatry
 - B. Postop Management
 - 1. Like CAB in OR, postop
 - 2. Right Heart Assist: Nitric Oxide, Isuprel,
 - 3. Antibiotics x 48 hours
 - 4. Leave OGT in until bowel sounds return, Early feeding
 - 5. Conditioning PT/ OT/ Speech
- XII. Patient self management
 - A. Teaching starts in ICU
 - B. Caregiver training
 - C. Living alone at home
- XIII. Community Expectations, Oh my!!
 - A. EMS basic education training by patient
 - B. Local Power company
 - C. Home Health Nurses
 - 1. Check and organize environment
 - 2. PT, OT until stable
 - D. Cardiac Rehab Programs
 - E. Hobbies, No Water Sports, Sexual Activities
 - F. Travel
 - 1. Letter to travel for TSA agents
 - 2. PBU in main cabin, extra batteries, Emergency batteries
 - 3. Contact nearest VAD center
- XIV. Ethical Issues of Long Term Support
 - A. Improve Quality of Life
 - B. Caregiver participation
 - C. When to stop
 - 1. Interdisciplinary Plan of Care
 - 2. DNR / AND Allow Natural Death
 - 3. Pain MD
 - 4. Hospice and Bereavement support
- XV. Our Experience
 - A. Young Mother “just wanted to see her 3 month old baby get older and my kids grow up.” She lived 1488 days.
 - B. Live as many as 500 miles away
 - C. They range in age from 16 to 84 years old
 - D. 16 year old patient to rate his quality of life on a scale of 1-10 = 8. His wish was that the batteries would last longer so he could go off road racing for a longer period of time.

- E. Sharp patient experience includes total days on pump of 23,714 days or 65 years, predominately spent at home actively participating in life.
- F. VADs are available for destination therapy for those not meeting transplant criteria. As centers gain more experience and referrals are made earlier in the disease process VAD patient care will be more streamlined decreasing length of stay and hospital care costs.
- G. Barney Clark 25 years ago, “ It’s been a pleasure to be able to help people and maybe you folks learned something” 1982

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Tissue Oxygenation, Perfusion and Gas Transport

Michelle Fournier

Level: Intermediate

CONTENT DESCRIPTION

Adequate oxygenation of a critically ill patient can be a challenge to the health care team. This presentation will link principles and interactions of gas transport (O_2 and CO_2), ventilation, and perfusion to ultimate delivery of oxygen to tissue cells. Common sources of hypoxemia will be discussed to include alveolar hypoventilation, ventilation-perfusion mismatching, shunting and diffusion abnormalities. Case presentations will be used to calculate Alveolar-arterial (A-a) gradients and discuss nursing and medical interventions to resolve varying sources of hypoxemia and inadequate perfusion.

LEARNING OUTCOMES

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

1. Calculate an A-a gradient or PaO_2/FiO_2 ratio and discuss their clinical significance.
2. Discuss the main sources of hypoxemia in common disease patterns to include: alveolar hypoventilation, ventilation-perfusion (V/Q) mismatching, pulmonary shunt and diffusion abnormalities.
3. Given a case presentation in which the patient has hypoxemia, formulate a proactive nursing management plan aimed at improving oxygen saturation and delivery.

SUMMARY OF KEY POINTS

- I. Comparison of pulmonary and systemic circulatory systems
 - A. Pulmonary Circulation
 1. Functions of pulmonary capillary bed
 2. Under most conditions, the pulmonary vessels act as passive, distensible tubes that enlarge and narrow with varying flow demand, keeping pressure relatively constant
 3. Most important cause of pulmonary artery vasoconstriction is low alveolar PO_2 .
 - B. System Circulation
- II. Ventilation
 - A. Mechanical movement of air in and out of lungs
 - B. Alveolar Ventilation – part of total ventilation taking part in gas exchange
 - C. Work of breathing: muscular effort required for ventilation
- III. Gas Behavior
 - A. Partial Pressure
 - B. Gas Composition

IV. Gas Transport.

- A. Delivery of O_2 to body cells and removal of CO_2 has 4 steps:
- B. Perfusion (Q)
 1. Flow = P/R (P = Pressure; R = Resistance)
Normal CO (Q) = 4 - 8L/min
 2. Actual perfusion occurs in capillaries
 3. Factors that can limit perfusion
 - a. Gravity
 - b. Lung Zones
 - c. Cardiac Output
 - d. Pulmonary vascular resistance (PVR) (influenced by PAP)
- C. Distribution of ventilation and perfusion
 1. Even distribution of ventilation (V) and perfusion (Q) = effective gas exchange
 2. Lung Zones: caused by the effects of hydrostatic pressure (gravity) in the pulmonary vessels
- D. Diffusion
 1. Passive process by which a gas moves from an area of higher partial pressure to an area of lower partial pressure
Diffusion = Surface Area x Driving Pressure x Solubility of the Gas
Tissue Thickness x Molecular Weight
 2. Diffusion of oxygen into the pulmonary blood
 3. Factors that affect rate of gas diffusion through the respiratory membrane
- E. Oxygen Transport
 1. 97% of O_2 is transported in a chemical bond with Hgb in the erythrocyte as oxyhemoglobin.
 2. 3% is carried as dissolved O_2 in the plasma and is measured as PaO_2 .
 3. The amount of dissolved O_2 is the primary factor that determines the amount of O_2 bonding with Hgb
- F. CO_2 transport in the blood: carried in three forms
 1. Dissolved (7 - 10 %) - measured by the $PaCO_2$
 2. Chemically combined with hemoglobin (30%)
 3. As bicarbonate (60 - 70% of the bicarbonate in the body)
 $CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$
- G. Oxyhemoglobin dissociation curve - relationship between oxygen saturation and PaO_2

V. Clinical Calculations

- A. A-a gradient: indicator of diffusion
 1. The A-a gradient ($PAO_2 - PaO_2$): the difference in

- partial pressure of oxygen in the alveolar gas and the pressure in the systemic arterial blood.
- The gradient is always a positive number. PAO_2 is always greater than PaO_2 .
 - Normal gradient is 3 – 16 mm, adults <30 yrs = < 10 mmHg, but with age $[2.5 + 0.25(\text{age})]$
 - Measures how efficient the lung is at equilibrating pulmonary capillary oxygen with alveolar oxygen. A large gradient generally indicates that the lung is the site of dysfunction.
 - PaO_2/FIO_2 ratio – normal is ~ 450
 - < 250 indicates acute lung injury
 - < 200 indicates ARDS

B. Alveolar Air

- As air passes is inhaled, it is warmed to 37°C and saturated to 100% humidification.
- $O_2 = 104$ mmHg $CO_2 = 40$ mmHg
- Formula; $PAO_2 = PIO_2 - (PaCO_2 \div 0.8)$
 $I =$ inspired
 $PO_2 = FIO_2(PB - 47)$
 47 mmHg = vapor pressure of water at 37°C
 $PIO_2 =$ pressure of inspired oxygen
 0.8 = assumed respiratory quotient (RQ)
 $FIO_2 =$ Fraction (%) of inspired oxygen
 A-a gradient = $PAO_2 - PaO_2 = [FIO_2(PB - 47) - (PaCO_2 \div 0.8)] - PaO_2$ OR $150 - PaCO_2 \cdot 1.8 - PaO_2$
 Example: ABG pH 7.4 PaO_2 90 $PaCO_2$ 40 Pt is on room air.

C. Pathologic conditions causing increased A-a gradients

VI. Delivery of oxygen to the body tissue cells

- Diffusion occurs along pressure gradients for O_2 , CO_2 , and water
- Hypoxemia
 - Alveolar hypoventilation (PAO_2 resulting in $PaCO_2$)
 - V/Q mismatch - the most common cause of hypoxemia (V/Q abnormality)
 - Low V/Q: inadequate ventilation to well perfused lung areas = shunt
 - Hypercapnia: generally not a problem unless severe shunting
 - Severe shunting (e.g., ARDS) is oxygen refractory

- High V/Q mismatch: poor perfusion of well ventilated lung areas = deadspace
 - Wasted ventilation
 - Most common cause is COPD, pulmonary embolus, also cardiogenic shock
- Diffusion defects: impaired gas diffusion across alveolar-capillary membranes
- Decreased Hgb

VII. Nursing Implications

- Clinical assessment of tissue oxygenation
 - Evidence of anaerobic metabolism
 - Perfusion status
 - End organ perfusion and function – neuro, myocardial, renal
 - Oxygen delivery
- Case Studies - Determine whether the following patients have a ventilation problem, an intrapulmonary oxygenation problem or both.

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Titration Vasoactive Drugs

Andrea Efre

Level: Beginner

CONTENT DESCRIPTION

The purpose of this presentation is to provide the learner with information about vasoactive medications used in emergency and critical care. When using vasoactive medication it is important to identify the goal of therapy and evaluate the response to treatment. The primary goal of most vasoactive medication is the manipulation of the preload, afterload or contractility. This presentation will review how this manipulation takes place and which medications are vasoconstrictive, vasodilatory or inotropic.

We will review the pathophysiology of the sympathetic and parasympathetic nervous system and discuss alpha and beta adrenergic receptors and their response to medications. We will consider the pharmacokinetics and pharmacodynamics of the most frequently used vasoactive medications. We will include calculation of dosages, and how to titrate a medication to achieve therapeutic goal. We will address the metabolism, elimination half lives and adverse reactions.

The medications to be discussed include: dopamine, dobutamine, epinephrine, norepinephrine, phenylephrine, phosphodiesterase (PDE) inhibitors, nitrates, and beta blockers. We will discuss the use of fluids, diuretics, calcium channel blockers, ACE inhibitors, and some antiarrhythmic medications. Combination therapy is often beneficial.

The presentation will identify the nursing implications in caring for a patient receiving vasoactive medications.

Additional monitoring techniques may be needed and possible adjustment of medications for special population groups such as the elderly or renally impaired.

The prerequisite for this class is a basic understanding of hemodynamics and the treatment of patients in an acute setting. This class would be beneficial to the new graduate, the seasoned ER or ICU nurse, the advanced practice nurse or pharmacist. Approximately 50% of the information presented is pharmacological, the other percentage includes assessment, nursing implications and patient outcomes

LEARNING OUTCOMES

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

1. Identify the goal of therapy and evaluate the response to treatment.
2. Understand the practice of titration of vasoactive drugs.
3. Categorize the most commonly used vasoactive medications and their expected therapeutic response.
4. Discuss nursing implications involved when caring for a patient receiving vasoactive medications.

SUMMARY OF KEY POINTS

- I. Goal of Therapy
 - A. To manipulate Preload, Afterload or Contractility
 - B. To treat an arrhythmia

- C. To support an organ
- D. Assess baseline
 1. Baseline Vital Signs
 2. Hemodynamic Monitoring (Invasive or Non-Invasive)
 3. Physical Examination
 4. Baseline Lab, radiology, EKG and diagnostic tests (know your renal and hepatic function)
 5. Know the History of Present Illness and Past Medical History
- E. Plan how evaluation of the success of therapy will be measured
 1. Stroke Volume, Cardiac Output, resolution of arrhythmia
- II. The goal of vasoactive medications is to manipulate the:
 - A. Preload
 - B. Afterload
 - C. Contractility
- III. Preload Manipulation
- IV. Afterload Manipulation
- V. Contractility Manipulation
- VI. Autonomic Nervous System
 - A. Alpha ()
 1. Peripheral Arterioles
 - B. Beta 1 (1)
 1. Myocardial muscle tissue and cardiac conduction system
 - C. Beta 2 (2)
 1. Vascular smooth muscle
 2. Bronchi and liver
 - D. Dopaminergic
 1. Renal and mesenteric vasculature
- VII. Pharmacological Manipulation of Receptors
- VIII. Combination Therapy
- IX. Dopamine
- X. Dobutamine
- XI. Epinephrine
- XII. Norepinephrine
- XIII. Phenylephrine
- XIV. Phosphodiesterase (PDE) Inhibitors
- XV. Nitrates
- XVI. Beta Blockers
- XVII. Calcium Channel Blockers
- XVIII. Diuretics
- XIX. ACE Inhibitors and ARB's
- XX. Antiarrhythmic
- XXI. Calculation of Medications
 - A. Basic Formulas
 - B. Drip Calculations

- C. Cal Factors
- XXII. Nursing Implications
 - A. Administration of Medications
 - 1. Safe Dosing
 - 2. Monitor carefully
 - 3. Follow your facility policy
 - 4. Hemodynamically stable
 - 5. Additional nursing roles
 - B. Evaluation of response
- XXIII. Patient and Family Teaching

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To Transfuse or Not to Transfuse? That is the Question!

Elaine C. Killough

Level: Intermediate

CONTENT DESCRIPTION

Anemia in critical illness is a common occurrence related not only to direct blood loss, but also to physiological mechanisms that inhibit the production of red blood cells, paralleling chronic disease. Anemia is typically treated with red blood cell transfusion, with a goal of optimizing tissue oxygen delivery. Despite multiple attempts to benchmark the optimal threshold at which to transfuse, the decision remains highly patient-specific. Over the past decade, many new clinical controversies have emerged which have caused us to question the assumed benefits of red blood cell transfusion. This session will describe the physiological pathways that lead to anemia in critical illness, and its outcomes. Current evidence to support decision-making about the appropriateness of transfusion, and the associated risks and benefits will be examined. Nursing interventions to maintain critical red blood cell mass, and innovative strategies to enhance oxygen delivery will be also be explored. Case studies will be included to support the evidence. A basic understanding of the concepts of oxygen delivery and the immune response is required for this session. The content is designed for critical care nurses who desire to broaden their understanding of anemia in critical illness and to utilize current evidence as a foundation for critical thinking and collaborative decision-making.

LEARNING OUTCOMES

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

1. Describe the multiple etiologies of anemia in critical illness and current practices to address it.
2. Compare and contrast the immune-mediated complications of red blood cell transfusion
3. Identify and apply three nursing strategies to support preservation of red blood cell mass.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Transfusion medicine: A brief overview
 - B. The current crisis
 - C. To transfuse or not to transfuse: Evolution of the controversy
- II. Transfusion: The Physiological Logic
 - A. Goal: Optimize tissue oxygenation
 1. Increase RBC mass
 2. Minimize morbidity and mortality
 3. Optimize functional outcomes
 - B. Role of hemoglobin: Oxygen transport
 - C. Impact of decreased hemoglobin
 1. Decreased O₂ carrying capacity
 2. Increased O₂ dissolved in plasma

- D. Compensatory mechanisms
 1. Cardiac output
 2. Vasodilation in microcirculation
 3. Increased O₂ extraction (shift to the right)
- III. Anemia in Critical Illness
 - A. Prevalence
 1. At admission
 2. Up to 95% by day 3
 3. Up to 80% at ICU discharge
 - B. Etiology: The obvious
 1. Blood loss
 2. Underlying co-morbidities
 - C. Etiology: The not so obvious: The anemia of critical illness
 1. Phlebotomy
 2. Stress-related GI bleeding
 3. Pharmacological interventions
 4. Inflammation: Cytokines inhibit the production of RBCs
 5. Imposed nutritional deficiencies (iron, folate, vitamin B)
 - D. Physiologic impact of anemia in critical illness: Impaired O₂ delivery
- IV. Re-examining the 10/30 Rule: The TRICC Study
 - A. The Question: Does a restrictive transfusion strategy produce significantly different outcomes when compared to a liberal strategy?
 1. Transfuse only if Hgb <7.0 g/dL and maintain at 7.0 – 9.0g/dL (restrictive)
 2. Transfuse if Hgb <10g/dL and maintain at 10 – 12g/dL (liberal)
 - B. Outcomes: Better for restrictive group except with significant cardiac disease
 - C. Avoiding the transfusion “trigger”
- V. Current Transfusion Practices
 - A. Multiple, large prospective studies
 - B. % transfused: 25.0 – 53.4
 - C. Mean pre-transfusion Hgb: 7.4 – 8.6g/dL
 - D. Benefits of leukoreduction
- VI. Downside of Transfusion
 - A. Where we’ve come from: Reduction of risk at source
 - B. Not there yet: Transfusion-transmitted infections
 - C. Immune-modulated complications: Immune activation I – The familiar
 1. Acute hemolytic reactions (AHTR)
 2. Delayed hemolytic transfusion reactions (DHTR)
 3. Febrile non-hemolytic transfusion reactions
 4. Anaphylactic reactions
 - D. Immune-modulated complications – Immune acti-

- vation II – The less familiar
 - 1. Transfusion-associated graft-versus-host disease (TAGVHD)
 - 2. Transfusion-related acute lung injury (TRALI)
 - 3. Second-hit hypothesis
- E. Transfusion-related immune modulation: Down-regulation (TRIM)
- F. Transfusion-related Acute Circulatory Overload (TACO)
- G. Differentiating TACO and TRALI
 - 1. Key assessments
 - a. PCWP
 - b. Pulmonary edema fluid protein concentration
 - c. BNP
 - d. Response to volume/preload reduction
 - e. Ejection fraction
 - f. Systolic B/P
 - 2. Absence/presence of ALI risk factors
- H. Addressing the immune-related downside: Future directions
- I. RBC storage issues
 - 1. Association between prolonged storage times and adverse outcomes
 - 2. Most frequently reported critical time: >14 days (standard is 21 days)
 - 3. Mechanism: The “storage lesion:” Impaired O₂ transport
- VI. State of the Science: Transfusion and Specific Patient Populations
 - A. Cardiovascular disease
 - B. Respiratory failure
 - C. Trauma
 - D. Surgical/perioperative
 - E. Sepsis
- VII. If Not Blood, What?
 - A. Bloodless approach to surgery
 - B. Erythropoietin
 - C. The future: Oxygen therapeutic agents
 - 1. O₂ delivery and volume replacement
 - 2. Two currently in advanced clinical trials

- VIII. Nursing Strategies to Address Anemia in the ICU
 - A. Proactive vs. reactive
 - B. Be aware: Current recommendations
 - C. Reduce blood loss
 - D. Optimize oxygenation
 - E. Advocate for adequate nutrition support
 - F. Support production of RBCs
 - G. Collaborative guidelines
- IX. Case Studies
- X. Conclusion: To Transfuse or Not to Transfuse?

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Top 5 Cardiac Studies

Nancy M. Albert

Level: Advanced Practice

COURSE DESCRIPTION

There are many new research studies published in many cardiac and general medicine journals throughout the year. Trying to determine which studies are the “Top 5” is difficult since various branches of cardiac medicine have individualized areas of focus (i.e., preventive cardiology, electrophysiology, interventional cardiology, cardiac surgery, heart failure and transplantation, and general (clinical) cardiology. So, the top 5 picks were based on the following principles: (a) different types of studies: drug, devices, etc.; (b) different patient populations: acute myocardial infarction, congestive heart failure, stable coronary disease, etc., (c) different clinical settings: ambulatory vs. emergency/critical/ or acute hospital care vs. chronic care and (d) large implications for cardiac nurses.

LEARNING OUTCOMES

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

1. Discuss risks of mortality, acute myocardial infarction, and stent thrombosis events in patients who have received drug-eluting or bare metal stents
2. State strategies to decrease delays in delivery of primary percutaneous coronary interventions
3. Describe which patient subgroups benefit most from intensive statin therapy and which require careful monitoring for adverse effects.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Elements of chosen studies that lend to generalizability
- III. Top 5 Cardiac Studies
 - A. Drug-eluting versus bare metal stents: effectiveness and safety
 - B. Effects of delays in coronary artery revascularization after ST elevation myocardial infarction
 - C. Intensive statin therapy to achieve very low low-density lipoprotein cholesterol levels in patients with coronary heart disease
 - D. Thiazolidinediones and heart failure in patients with cardiovascular disease and type 2 diabetes
 - E. Maintenance of sinus rhythm in atrial fibrillation or flutter with dronedarone

- IV. Themes of discussion for each topic above
 - A. Background of why the topic is important
 - B. Study methodology and results
 - C. Implications for evidence-based nursing care, nursing processes and/or nursing research
- V. Waiting for evidence to be presented via publication of cardiac research: Closing remarks

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Tu JV, Bowen J, Chiu M, et al. Effectiveness and safety of drug-eluting stents in Ontario. *New Engl J Med.* 2007;357:1393-1402.

Reducing Door to Balloon Times:

Szadkowska I, Goch JH, Polak L, Stepie H, Chizy ski K. The relationship between early recanalization and serum NT-proBNP levels in patients with a first ST-segment elevation myocardial infarction treated with primary coronary angioplasty. *Acta Cardiol* 2007;62:479-84.

Intensive Statin Therapy:

Afilalo J, Majdan AA, Eisenberg MJ. Intensive statin therapy in acute coronary syndromes and stable coronary heart disease: a comparative meta-analysis of randomised controlled trials. *Heart* 2007;93:914-921.

Thiazolidinediones and Heart Failure:

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Maintaining Sinus Rhythm:

Singh BN, Connolly SJ, Crijns HJ, et al. Dronedarone for maintenance of sinus rhythm in atrial fibrillation or flutter. *N Engl J Med* 2007;357:987-999.

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Top Ten New Drugs to Know for 2008

Melissa Pollard

Level: Advanced Practice

CONTENT DESCRIPTION

New drugs are frequently being released into the market. Many are similar to medications we already use and prescribe, but others are new in their actions, or indications, that require more study to become familiar with. This session will look at ten new drugs, or drugs with new indications, and discuss their benefits, side effects, and patient case studies.

LEARNING OUTCOMES

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

1. Participants will be able to identify indications for the drug
2. Participants will be able to discuss common side effects
3. Participants will be able compare this drug others available and discuss its benefits versus risks.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Medication
 - A. indications
 - B. prescribing details
 - C. side effects
 - D. benefits
- III. Case Studies

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Up to Date, Inc, founded by B. D. Rose & J.M. Rush. www.uptodate.com

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Top Ten Topics for Tracheostomy Management

Maureen A. Seckel

Level: Intermediate

CONTENT DESCRIPTION

Have you ever been perplexed as to whether you should inflate or deflate a tracheostomy cuff? Are you puzzled by what fenestrated tracheostomy tube is and how does it work? Why do some tracheostomy tubes have an inner cannula while others do not? This session is designed for all critical care nurses, from novice to expert, who desire practical and research based information about the care and management of patients with acute, short term, and long term tracheostomy tubes. You will also learn what steps in your protocols or procedures for airway management are based on science versus tradition. This session will also assist you in learning the “how to” on handling and preparing for common airway emergencies. Discussion will include a series of case presentations to promote assessment and decision making skills.

LEARNING OUTCOMES

By the end of this session the participant will be able to:

1. Understand anatomy and physiology pertinent to tracheostomy management.
2. Review cuff inflation/deflation strategies for speaking valve, oral intake, aspiration prevention and ventilator care.
3. Describe three emergency airway assessment and management strategies.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Important Moments in Tracheostomy History
- III. Review of Airway Anatomy and Physiology
- IV. Top Ten Topics
 - A. When and How to Trach
 - B. Types of Trach Tubes
 - C. Suctioning
 - D. Aspiration/VAP Prevention
 - E. Trach Care
 - F. Changing Trach Tubes
 - G. Fluids and Diet with a Trach
 - H. Speaking with a Trach
 - I. Capping, Corking, and Decannulation
 - J. Trach Teaching for Home Care
- V. Selected Case Studies on Common Airway Emergencies
- VI. Summary/Discussion/Questions Limit to a maximum of 2 pages!

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The Toxic 10: Tackling the Top 10 Meds & Mistakes Involved in Adverse Drug Events (ADE)

Deborah Tuggle

Level: Advanced Practice

CONTENT DESCRIPTION

More than 7000 Americans die from medication errors every year, yet nearly half of all adverse drug events (ADE) have some measure of “preventability.” Rather than errors of commission, many of these deaths are due to errors of omission. This session will address concepts related to medication mishaps and prepare the nurse for improving patient safety in their institution. A review of the top ten drugs involved in ADEs and the top ten errors committed by healthcare professionals will progress to a discussion of evidence-based solutions for improvement. New technology related to electronic prescriptions, computer-based drug calculations and other forms of pharmacovigilance will be included. Special tips for the bedside nurse will be highlighted along with quality improvement ideas for detoxifying broken drug systems.

LEARNING OUTCOMES

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

1. Review the top ten medications involved in adverse drug events.
2. Discuss the top ten mistakes made that lead to adverse drug events.
3. Describe current recommendations for the prevention of adverse drug events.

SUMMARY OF KEY POINTS

- I. Top 10 meds misused or mishandled by healthcare professionals and their involvement frequency
 - A. Insulin (4%)
 - B. Morphine (2.3%)
 - C. Potassium chloride (2.2%)
 - D. Albuterol (1.8%)
 - E. Heparin (1.7%)
 - F. Vancomycin (1.6%)
 - G. Cefazolin (1.6%)
 - H. Acetaminophen (1.6%);
 - I. Warfarin (1.4%)
 - F. Furosemide (1.4%)
- II. 5 Million Lives Campaign warnings related to high risk drugs including: narcotics, anticoagulants, sedatives, and insulin
- III. Top 10 mistakes
 - A. Dose error 31%
 - B. Frequency 17%

- C. Route error 10%
 - D. Missed dose 7%
 - E. Wrong technique 6%
 - F. Illegible order 6%
 - G. Duplicate therapy 5%
 - H. Known allergy 4%
 - I. Wrong drug/patient 4%
 - J. Drug-drug interaction 4%
- IV. Other mistakes (< 1% each) including: Equipment failure, inadequate monitoring, preparation error
 - V. Problems with look alike drugs, sound alike drugs, abbreviations, trailing zeros...
 - VI. The 5 rights for individual safety performance
 - VII. Fixing broken systems through: better communication, labeling, staffing, work conditions, staff and patient education, non-punitive reporting...
 - VIII. iHi 5 Million Lives Campaign Guidelines
 - IX. Computerized Order Entry
 - X. National ePrescribing Patient Safety Initiative
 - XI. Special tips for the bedside ICU nurse

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- National Coordinating Council for Medication Error Reporting and Prevention: www.nccmerp.org
- United States Pharmacopeia: www.usp.org

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Toxic Toll: Adverse Drug Reactions Requiring Critical Care

Angela Smith Collins

Level: Advanced Practice

CONTENT DESCRIPTION

Medications undergo rigorous scientific trials prior to market distribution. Nevertheless, no medication is risk-free. When a medication is released, all the identified side effects and adverse effects that occurred in the research population are listed. However, because of the unique genetic, cultural, gender, and environmental set of variables that comprise each person's physiology and pathophysiology, adverse effects may occur in association with any dose of a particular medication. Each patient expects to receive greater benefit from a medication than a risk. Estimates listed in the literature suggest that three to seven percent of all acute care admissions are due to serious adverse effects of medications. The intent of this interactive session is to delineate six different medications that may trigger an adverse drug reaction leading to life threatening consequences. The seven toxic drug reactions discussed in a case methodology are Steven-Johnson's syndrome, serotonin syndrome, photodynamic therapy, and phytonadione's black box, medication classes that promote suicide, prolonged Q-T medications, and propofol infusion syndrome. Data relevant to the prevention, intervention, and management will be presented. The relevant pharmacokinetics and pharmacodynamics to each agent and any identified reversal agents will be outlined.

LEARNING OUTCOMES

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

1. Discuss variables that influence or increase the likelihood of an Adverse Drug Reaction (ADR) occurrence.
2. Delineate case reports of seven drug classifications associated with adverse drug reactions.
3. Consider how skilled communication can improve data collection to build an evidence base about adverse drug reactions.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Definitions
 - B. Scope of Problem
- II. Adverse Drug Reactions
 - A. Who
 1. Vulnerable populations
 2. Unrepresented research subjects, gender, ethnicity
 3. Goldilocks
 - B. What
 1. Troublesome Medication Classes
 2. Polypharmacy
 - C. Where, How
 1. Critical Care Units

2. Emergency Department
- D. When
 1. Time course
 2. Dose
- III. Adverse Drug Reactions Requiring ICU Admission
 - A. Stevens-Johnson Syndrome/TENS
 1. Triggers
 2. Prognosis
 3. Prevention
 4. Intervention
 5. Management
 - B. Serotonin Syndrome
 1. Triggers
 2. Nursing Care
 3. Prevention
 4. Intervention
 5. Management
 - C. Photodynamic Therapy
 1. Populations
 2. Care modifications
 3. Prevention
 4. Intervention
 5. Management
 - D. The Lethal Black Boxes
 1. Vitamin K
 2. Dilantin
 3. Prevention
 4. Intervention
 5. Management
 - E. Medications and Suicide – More Black Boxes
 1. Interferon
 2. Accutane
 3. Prevention
 4. Intervention
 5. Management
 - F. Prolonging the QT – does every medication interfere?
 1. Prevention
 2. Intervention
 3. Management
 - G. Propofol Infusion Syndrome
 1. Prevention
 2. Intervention
 3. Management
- IV. Skilled Communication
 - A. Pharmacy input
 - B. Computer cross-checking
 - C. Real-time information
 - D. Rural challenges

- E. Post-marketing data collections
- F. Critical care units/high-risk

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Tracheostomies: Not Just Another Tube

Sarah Stranberg
Mary Fran Tracy

Level: Intermediate

CONTENT DESCRIPTION

Tracheostomies traditionally can be one of the most anxiety-producing pieces of equipment that nurses care for in a variety of settings. This evidence-based session will focus on increasing the acute and critical care nurse's understanding of the purpose, indications, and potential complications of tracheostomies, particularly in relation to endotracheal tubes. Tracheostomy placement timing and technique (percutaneous vs. open surgical) based on the latest evidence will be discussed and compared between different patient populations. A variety of tracheostomy tubes will be reviewed including the unique advantages of each. Procedures to diagnose airway complications, including aspiration, will be described and include video clips of bronchoscopy and swallow studies (videofluoroscopic and endoscopic). Issues related to both acute critical as well as long-term use of tracheostomies with or without mechanical ventilation will be detailed. The presenters will also utilize case studies to enhance comprehension and problem solving of care of tracheostomies. New trends in tracheostomy tubes and care will be presented. A multidisciplinary (respiratory therapy, speech pathology, nursing, and physicians) process improvement project that was focused on reducing tracheostomy complications will also be described including standard of care development and project outcomes. This session is designed for acute and critical care nurses from novice to expert to enhance clinical knowledge, skills, and comfort in caring for patients with tracheostomies based on the latest evidence.

LEARNING OUTCOMES

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

1. Describe basic tracheostomy tubes and rationale for placement in specific patient populations.
2. Identify potential complications associated with tracheostomy and describe practice strategies to prevent complications.
3. Describe key components of a multidisciplinary "trach team" and process improvement projects to improve the care of patients with tracheostomies.

SUMMARY OF KEY POINTS

- I. Airway: Back to the ABC's of critical care
- II. Tracheostomy
 - A. Indications for tracheostomy placement
 - B. Potential benefits of tracheostomy
 - C. Relevant anatomy and physiology
- III. Tracheostomy placement
 - A. Procedure
 1. Open
 2. Percutaneous

- B. Complications related to placement procedure
- C. Timing of placement
- IV. Potential complications
 - A. Complications of tracheostomies
 - B. Tracheoesophageal fistula
 - C. Tracheoinnominate fistula
 - D. Preventing complications
- V. Tracheostomy issues in specific critical care populations
 - A. Traumatic brain injury
 - B. Spinal cord injury
 - C. Trauma
 - D. Cervical spine surgery
 - E. Other neurocritical care
 - F. Cardiothoracic surgery
- VI. Tracheostomy tube
 - A. Components
 - B. Difference in tubes
 - C. New trends in tracheostomy tubes
 - D. Mini-trach II
- VII. Tracheostomy and swallowing function
 - A. Incidence of dysphagia/aspiration
 - B. Mechanism of dysphagia associated with artificial airways
 - C. Evaluation of dysphagia
 1. Speech-Language Pathology
 2. Clinical assessment
 3. Modified (videofluoroscopic) barium swallow studies
 4. Fiberoptic endoscopic evaluation of swallowing (FEES)
- VIII. Tracheostomy and communication/speech
 - A. Speech options
 - B. Passy-Muir Speaking Valves
 1. Indications
 2. Safety concerns
- IX. Successful management of patients with tracheostomy
 - A. Interdisciplinary "trach team"
 - B. Standards of care
 - C. Resources for staff
 1. Manual
 2. Safety/ care cards
 - D. Rehabilitation
 - E. Patient/family/staff education and collaboration

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Tracheostomy and Weaning from Mechanical Ventilation

Richard H. Kallet
Sponsored by Dale Medical Products

Level: Intermediate

CONTENT DESCRIPTION

Tracheostomy, a surgical opening in the anterior neck to allow ventilation, is used to relieve upper airway obstruction, to facilitate long-term mechanical ventilation, airway protection and secretion removal. The purported advantages of tracheostomy include improved patient comfort and pulmonary hygiene; both decreased anatomic dead-space and work of breathing that facilitates weaning from mechanical ventilation.

This presentation begins with a brief review of the pertinent anatomy and physiology along with a succinct description of traditional surgical and the newer percutaneous dilatory procedures. The primary focus of the presentation will be on the purported role of tracheostomy in facilitating weaning from mechanical ventilation. This will involve a detailed review of the theoretical advantages of tracheostomy: namely, a reduction in both dead-space ventilation and sedation, improvements in patient comfort, pulmonary mechanics, work of breathing, and pulmonary hygiene.

LEARNING OUTCOMES

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

1. Identify the anatomic structures pertinent to performing a tracheostomy.
2. Differentiate between the surgical and percutaneous dilational techniques for performing a tracheostomy.
3. List both the short-term and long-term risks associated with a tracheostomy.
4. Describe the effects of tracheostomy tubes on dead-space ventilation and work of breathing relative to translaryngeal endotracheal tubes.

SUMMARY OF KEY POINTS

- I. Brief Review:
 - A. Anatomical landmarks,
 - B. Indications
 - C. Risks (short and long term).
- II. Theoretical Advantages
 - A. Reduced Dead-Space
 - B. Improved Pulmonary Mechanics
 - C. Decreased Work of Breathing
 - D. Reduced Risk of Aspiration
 - E. Improved Pulmonary Hygiene
 - F. Decreased Duration of Mechanical Ventilation
- III. Clinical Evidence, A critical review of studies on:
 - A. Reduced Dead-Space
 - B. -Improved Pulmonary Mechanics
 - C. Decreased Work of Breathing
 - D. Reduced Risk of Aspiration
 - E. Improved Pulmonary Hygiene
 - F. Decreased Duration of Mechanical Ventilation
- IV. Summary Remarks
 - A. Do tracheostomies reduce weaning time or do clinicians approach patients with tracheostomies differently from those with endotracheal tubes?
 - B. Who benefits from tracheostomies? - timing and target population.
 - C. The direction of future clinical trials

Transformers: The Making of a Critical Care Nurse

Kelly Murphy
Kassie Basnight

Level: Beginner

CONTENT DESCRIPTION

This session will discuss the development of a program for new RN's/GN's to rotate through 10 different acute care areas. It is called the "Rotational Program". New nurses begin a critical care transition program by attending 40 hours of PRE-ECCO class. PRE-ECCO is a class where the new critical care nurses learn and do extensive hands on skills like managing basic equipment, giving medications, learning communication skills, assessment, documentation and safety skills. Then they start a portion of the program where they are paid to complete 8 hours of the ECCO module at home per week and do an 8-hour workshop building on the skills in the ECCO module. The workshop includes preceptors-instructors from the different ICU units that are experts in the chosen module. They set up equipment and do mock situations to prepare the students for real events. The RN's work 2 twelve hour shifts per week with a preceptor during this time also. At the end of the ECCO module portion of the class they complete a week of advanced skills, which includes items, like advanced hemodynamics and advanced modes of ventilation. They are building on the basic skills they have previously acquired. The first rotation is a 24-week rotation and the rest of the rotations are 10 weeks only. When they rotate to a new unit they go back on Orientation for the first 2 weeks and then work independently for the second half. The first rotation is longer because they are acquiring the basic ICU skills to take care of any ICU patients. This program has been very successful and we have trained about 134 nurses so far. One additional benefit that we received from the program is that we have developed nurses that can take care of any patient in any unit and are not distracted by off-service patients arriving due to high census. The specific details and success of this program in orientating new nurses to critical care will be discussed.

LEARNING OUTCOMES

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

1. Describe the needs assessment and development of this program.
2. Explain the importance of the PRE-ECCO Phase to assist the preceptors by building a framework from which to build upon.
3. Describe the rotational portion of the program.

SUMMARY OF KEY POINTS

- I. Theory behind development of program
 - A. Needs Assessment for Recruiting & Developing Critical Care Nurses
 1. GN's in Critical Care

2. Expansion
3. Attrition
- B. Interviewing & Hiring Process
- II. Initial Introduction/Orientation
 - A. Video Clip- Orientees
 - B. Schedules
 1. PRE-ECCO Week
 2. EKG Week
 3. Home Day
 4. Workshops
 5. Two (12 Hours shifts) with Preceptor
 - C. Ranking of Units –Match Day
 - D. Payroll
- III. Pre-ECCO – Day 1
 - A. Advice from previous group
 - B. Tour the Units
 - C. Advice from Preceptors
 - D. Pictures/ Mottos
 - E. Advice for Success
 - F. The Right Fit
- IV. Clinical Skills Week
 - A. Basic ICU Equipment
 - B. Monitors
 - C. Assessment/ Medications
 - D. Documentation/Paperwork
 - E. Communication
 1. Bedside Battles/M.D.'s
 2. Co-workers/Ancillary Staff
 3. Supervisors
 - F. Safety
 - G. Infection Control
 - H. Families
 - I. Prioritizing
 - J. Getting "Help"
 - K. Report
 - L. Orientation Progress Reports
 - M. Preceptor Feedback
 - N. Critical Thinking
 - O. Scenarios
- V. EKG Week
 - A. Afternoons with:
 1. Monitor Technician
 2. Unit Clerk
 3. Respiratory Therapist
- VI. First Assignment
 - A. ECCO Orientation
 - B. Two (12 hour shifts with Preceptor)

- C. Pyramid of Clinical Competence
- VII. Preceptor Preparation
 - A. Explaining ECCO
 - B. Correlating Weekly assignments
 - C. Preceptors in Paradise- Incentive Package
 - D. Even Preceptors Need Breaks
- VIII. Adjuncts to ECCO Program
 - A. Advanced Skills Week
 - 1. Advanced Hemodynamics
 - 2. Advanced Modes of Ventilation
 - 3. Code Blue Scenarios
 - 4. Brain Death & Donor Care
 - 5. Trauma Update Day
- IX. Final Exam
 - A. 12 lead ECG Interpretation
 - B. STAT Nurse Day
- X. Second Rotation
 - A. Competency Tools take a walk
 - B. Clinician Handoff
 - C. Specialty Areas- ED, PACU, Pediatrics

- XI. Development of Orientation Paperwork
 - A. Rotational Book
 - B. Competency Tools
- XII. Evaluation of Program
 - A. More Hands on for Pre-ECCO
 - B. More Preceptor Communication
 - C. Staff Success and Personal Tug of War
 - D. Module Stories
- XII. Future of Program
 - A. Growth of Management Team

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Transfusion Alternatives

Jacqueline Morgan

Level: Intermediate

CONTENT DESCRIPTION

With the increased demand for blood and blood component therapy in light or stagnated supply, it is beginning more important to plan appropriately for surgical procedures; use fluid resuscitation and auto transfusion appropriately; and also to develop alternatives to hemoglobin as an oxygen carrier. This presentation will describe appropriate fluid choices for resuscitation based on evidence for specific diagnosis. It will review the role of exogenous erythropoietin and the nursing implications of pharmacologic alternatives for hemoglobin.

LEARNING OUTCOMES

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

1. Describe the mechanism of action and nursing implications of hematopoietic growth factors
2. Describe the mechanism of action and nursing implications of volume expansion and autologous donation
3. Describe the mechanism of action and nursing implications of oxygen carriers

SUMMARY OF KEY POINTS

- I. Synthetic growth factors
 - A. Candidates: chronic renal failure, patients receiving chemotherapy, HIV patients receiving AZT, Hepatitis C patients on Rebetol, surgical patients
 - B. Colony Stimulating Factors: Epoetin alpha, Epoetin Beta, Darpoetin alpha
 1. Actions: increases the number of progenitor cells, augments hemoglobin synthesis, enhances the release of reticulocytes from bone marrow, improves red blood cell viability, and influences tumor regression
 - C. Issues: Iron must be optimized to be effective, the surgical patient needs to begin therapy up to three weeks prior to surgery, Hematocrit must be followed and dosing based upon level and the rate at which the level has risen
 - D. Contraindications: uncontrolled hypertension, hypersensitivity to mammalian cells, allergy to albumin

- E. Cardiovascular effects: free hemoglobin causes an elevation of oncotic pressure resulting in renal dysfunction, coagulopathy, decreased GFR, liver failure, and hypertensive episodes
- II. Volume Expansion
 - A. Fluid Therapy; Colloids versus Crystalloids
 1. Colloids: prompt but expensive, are of varying cell size thus can cause damage to capillary membranes
 2. Crystalloids: Isotonic (NS, LR) remains in the vascular space until overloaded, hypotonic (1/2 NS, 1/4 NS) hydrates intracellular space, Hypertonic (D10) pulls fluid from the intracellular and interstitial space
 3. The dextrose controversy: D5W is isotonic in solution, hypotonic in the body.
 4. LR changes into lactate in the liver, NaCL can lead to chloride acidosis
 - B. Blood Salvage option
 1. Cell saver: compatible, easy to collect, disadvantage is that platelets and clotting factors can be removed
 2. Autotransfusion: blood is compatible, easy to collect, risk of infection, risk of DIC
 - III. Oxygen Carriers
 - A. Pleurofluorocarbon emulsion: synthetic fluorinated hydrocarbons capable of dissolving oxygen and delivering it to tissues
 - B. Hemoglobin solutions: human or bovine hemoglobin that is cross linked or polymerized and added to electrolyte solutions.

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Transfusion Turmoil: Benefits versus Risk of TRALI and TACO

Pamela Bolton
Jane Whalen

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this session is to discuss the benefits and risks of transfusing blood and blood components. Transfusion practices, pathophysiological derangements of transfusion, complications, and treatment of these resulting conditions in the critical care population will be explored. Evidenced-based strategies will be discussed to prevent transfusion-related complications and transmission of infections. Finally, the information will be synthesized for the participant through the use of patient case reports to exemplify their role in the identification and treatment of these potentially lethal complications.

LEARNING OUTCOMES

1. Identify the risks and benefits of blood and blood product transfusion.
2. Discuss pathophysiological derangements of transfusion of blood and blood products.
3. Differentiate the clinical presentation and treatment of TRALI, Transfusion Associated Circulatory Overload (TACO), transfusion reactions, and product contamination.

SUMMARY OF KEY POINTS

- I. History of Transfusion practices
 - A. Benefits / Risks
 - B. Transfusion = Transplant
 - C. Statistics
 1. ICU Transfusions
 2. Transfusion related mortality
 3. Underdiagnosing / underreporting complications
- II. Transfusion Practices
 - A. Anemia in critical care
 1. Phebotomy associated anemia
 2. Chronic / acute anemia
 3. Treatment
 - B. Transfusion Guidelines
 1. General ICU patient population
 - a. Packed red blood cells
 - b. Fresh frozen plasma
 - c. Platelets
 - d. Cryoprecipitate
 2. Special patient populations
 - a. Cardiovascular
 - b. Oncology
 - c. Nephrology

III. Complications of Blood & Blood Product Transfusion

- A. Infections
 1. Ventilator Associated Pneumonia
 2. Bacteremia
 3. West Nile Virus
 4. Hepatitis C
- B. TRALI / TACO
 1. Diagnosis of TRALI / TACO
 - a. Respiratory insufficiency
 - b. Degree of respiratory compromise requires medical intervention
 - c. Onset of symptoms is temporally related to transfusion
 - d. No other clinical cause (ABO incompatibility, volume overload, allergic reaction, sepsis)
 2. Pathophysiology: Transfusion-Related Immunomodulation
 - a. Donor human leukocyte antigen (HLA) or granulocyte-specific antibodies
 - (1) Neutrophil sequestration in the lung
 - (2) Complement activation
 - (3) Endothelial cell damage
 - (4) Alveolar capillary leak
 - b. Reaction stimulated from incompatible donor white blood cells (WBCs)
 - c. Donor & recipients acquire antibodies from exposure to foreign WBCs through pregnancy or from transfusions
 3. Clinical Presentation
 - a. TRALI
 - (1) Recent transfusion of packed red blood cells, fresh frozen plasma, platelet, granulocytes; less likely with intravenous immunoglobulin and cryoprecipitate
 - (2) Respiratory distress, acute hypoxemia, non-cardiogenic pulmonary edema, fever, and hypotension
 - b. TACO
 - (1) Recent transfusion
 - (2) Respiratory distress, acute hypoxemia, cardiogenic pulmonary edema
 4. Treatment
 - a. Evidenced based transfusion practices
 - b. Blood Bank Notification
 - (1) Unit quarantine of units from same donor
 - (2) To defer an implicated donor
 - (3) FDA Notification
 - c. Supportive therapy

- C. Transfusion reaction
- D. Product Contamination
- IV. Alternatives to Blood Product Transfusion
- V. Case Studies
 - A. Cardiovascular surgery patient
 - 1. FFP transfusion
 - 2. TRALI related mortality
 - B. Cardiovascular surgery patient
 - 1. PRBC transfusion
 - 2. TRALI / TACO complication
 - C. Neurosurgical patient
 - 1. FFP transfusion
 - 2. TRALI related mortality
- VI. Future Considerations
- VII. Summary / Questions & Answers

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Traumatic Brain Injury: Caring for our Wounded Warriors

Charles Kuhens

Level: Intermediate

CONTENT DESCRIPTION

Traumatic Brain Injury (TBI) has become one leading source of morbidity in the current war on terrorism in Iraq and Afghanistan. Historically military medical personnel have been trained to care for penetrating brain injuries; however, with the advancement of body armor, there are more extremity and closed head injuries. The overwhelming majority of these TBI patients have mild/moderate concussion syndromes and sometimes not from a single source or blast, rather over an accumulation of a number of smaller blast over months. Many of the symptoms are those similar to Post Traumatic Stress Disorder (PTSD) such as irritability, mood swings and difficulty concentrating. Critical care nurses need to be able to recognize these similarities and incorporate the suspect of blast exposure to their care. The military is currently conducting research on the primary blast wave in brain injury. The military medical establishment has been keen to look into lessons learned from sports concussion and civilian mild TBI literature; however there are still gaps as related to explosion blast waves and combat. The returning war veterans are going to have a new set of needs and military as well as civilian health care providers, families and society as a whole need to increase their TBI knowledge.

LEARNING OUTCOMES

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

1. Identify the increased role TBI has taken directly attributed to the war on terrorism in Iraq and Afghanistan.
2. Understand the relationship between TBI patients and mild/moderate concussion syndromes and it's relationship to single vs. accumulated blast injuries
3. Identify the signs and symptoms of mild TBI and compare them with those of Post Traumatic Stress Disorder

SUMMARY OF KEY POINTS

- I. Epidemiology/Neuropathology Overview
 - A. Mechanisms of Injury
 - B. Hospitalization Trends
 - C. Post-concussive Symptoms
 1. Recovery
 2. Persistent Symptoms
 3. Repeat Concussions
- II. Relevance of TBI to military
 - A. Body Armor Advancement
 - B. Improvised Explosive Devices

- III. Blast Injury
 - A. Primary
 - B. Secondary
 - C. Tertiary
 - D. Quaternary
- IV. Shock Wave and Brain Injury
- V. TBI and PTSD incidence
 - A. Similar presentations
 - B. Co-morbidities
- VI. Post-deployment TBI Questionnaire
- VII. Current and Future Initiatives
 - A. Theater treatment archive
 - B. Joint VA/DoD enhanced case management
 - C. Telemedicine/telehealth initiatives
 - D. DARPA-BAA: PREVENT Preventing Violent Explosive Neurologic Trauma

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Triple Threat of Liver Failure: Renal, Pulmonary and Adrenal

Diane Byrum

Level: Advanced Practice

CONTENT DESCRIPTION

This purpose of this session is to discuss etiologies, sequelae, signs/ symptoms and management of the patient with liver disease who develops a concomitant failure of the renal system, pulmonary system and adrenal glands. Hepato syndromes are related to the systemic vasodilatation of liver failure. Altered blood flow to the kidney as a result of renal vasoconstrictions leads to prerenal failure in which the structures of the nephron are intact. Concomitant pulmonary failure results from vasodilatation of the pulmonary capillaries leading to AV shunting and V/Q mismatch. Finally, adrenal failure may result from the general adaptation to the stress of chronic liver failure. With renal, pulmonary or adrenal failure superimposed on liver failure, the prognosis is poor and management is difficult.

LEARNING OUTCOMES

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

1. Discuss the normal functions of the hepatic system.
2. Identify pathophysiological changes that occur with liver failure which lead to subsequent hepatorenal failure, hepatopulmonary failure and hepatoadrenal failure.
3. Discuss multidisciplinary management strategies for the prevention and treatment of hepato syndromes.

SUMMARY OF KEY POINTS

- I. Liver Functions
 - A. Carbohydrate
 1. glycogen storage
 2. gluconeogenesis
 3. blood glucose homeostasis
 - B. Protein
 1. breakdown AA for energy
 2. ureagenesis (removal NH₃)
 3. plasma protein synthesis
 - C. Lipid
 1. b-oxidation of fatty acids (ATP)
 2. formation ketones
 3. formation lipoproteins
 4. cholesterol synthesis
 5. phospholipid synthesis
 6. conversion of CHO/protein to lipids
 - D. Miscellaneous
 1. vitamin storage
 2. synthesis of coagulation factors
 3. iron storage
 4. detoxification/modification/excretion of drugs, hormones, toxins

- E. Circulatory/Immune
 1. Storage/filtration of blood
 2. ½ total body lymph formation
 3. Kupffer cell – phagocytizes 99% of intestinal bacteria translocation into portal circulation
 - F. Secretory/Excretory
 1. Bile production
 - a. Billirubin metabolism (by product of hemolysis)
- II. Hepatorenal Syndrome (HRS) - Acute renal failure which occurs without another cause in a person with advanced, chronic liver disease, liver failure and portal hypertension. It is characterized by abnormal arterial circulation including renal vasoconstriction with extra-renal circulation with arterial vasodilatation MAP 60-65 mmHg
 - A. Frequency – 10% in patients with cirrhosis and ascites
8-40% in patients with decompensated liver disease
 - B. Risk factors
 1. serum Na⁺ excretion
 2. dilutional hyponatremia
 - a. free water excretion
 3. Low MAP
 - a. plasma renin
 - b. plasma norepinephrine
 4. low plasma osmolality
 5. high urine osmolality
 6. Ascites
 7. Esophageal varices
 8. Poor nutritional status
 - a. serum urea
 - b. serum creatinine
 - C. Pathophysiology
 1. Hypoperfusion of kidneys (functional renal failure)
 - a. Underfill theory – traditional theory
 - (1) contraction of EABV (major event) systemic circulation – MAP
 - (2) portal pressure leads to excessive lymph
 - (3) lymph exceeds thoracic duct capacity (ascites)
 - b. Underfill- Revised Theory
 - (1) peripheral vasodilatation (major event) + renal vasoconstrictors
 - (2) Sodium retention (H₂O)
 - (3) impaired pressor response
 - c. Overflow theory –
 - (1) inappropriate retention Na⁺ (major event)
 - (2) portal pressor/excessive lymph

2. Type 1
 - a. 25% patients spontaneous bacterial peritonitis (SBP)
 - b. Acute liver failure, alcoholic hepatitis, decompensated cirrhosis
 - c. Rapid deterioration of renal function
 - (1) doubling of SCr > 2.5 mg/dl
 - (2) 50% reduction creatinine clearance < 20 ml/min
 - d. Survival is counted in weeks – usually 2 (80% mortality)
 - e. Jaundiced and significant coagulopathy
 - f. Death is a combination of hepatic/renal failure/varices
 3. Type 2 – Slow form
 - a. Renal impairment less rapidly progressive
 - b. Prognosis remains poor
 - c. Survival time measured in months
 - D. Diagnosis of HPS – no definitive test- ***presence decreased GFR in absence of other causes for ARF
 1. Major Criteria (all are required to diagnosis HPS)
 - a. decreased GFR (serum creatinine > 1.5 mg/dl or 24 creatinine clearance lower than 40 ml/min.
 - b. absence of shock, bacterial infection, fluid losses, nephrotoxic agents
 - c. no improvement after diuretic withdrawal and volume expansion
 - d. proteinuria less than 500 mg/d
 2. Minor Criteria (not required supportive only)
 - a. urine volume less than 500 ml/d
 - b. urine sodium less than 10 mEq/L
 - c. urine osmolality greater than plasma osmolality
 - d. serum sodium less than 130 mEq/L
 3. Other Criteria
 - a. CBC
 - b. Electrolytes
 - c. BUN/creatinine
 - d. Blood cultures
 - e. Cryoglobulins
 - f. Urinalysis
 - g. Urine electrolytes
 - E. Management of HRS
 1. optimal fluid management – fluid challenge look for renal response
 2. decrease precipitating factors (STOP – NSAID, neomycin, aminoglycosides, declomycin, lactulose,
 3. optimisation of renal hemodynamics
 - a. increase MAP to 85-90 mmHg
 - b. Dopamine – renal vasodilator
 - c. Ornipressin – vasopressin analogue with preferential splanchnic vasoconstriction leads to decreased renin/angiotensin, improved renal clearance and sodium excretion (2 hr infusion 6 IU/h)
 - d. Terlipressin – synthetic analogue of vasopressin, 4 hr half-life
 - e. Midodrine (a-adrenergic agonist)/octreotide (inhibit endogenous vasodilators)
 - f. Endothelin antagonists (BQ123 –trial GFR)
 - g. N-Acetylcysteine – powerful antioxidant acts on free radicals
4. Paracentesis – decreases renal venous pressure
 5. Renal support – CRRT, MARS (albumin dialysate)
 6. Surgical – TIPS
 7. Hemodialysis
 8. Liver transplant
- III. Hepatopulmonary Syndrome – Triad (liver disease, increased A-a gradient, intrapulmonary vascular disease)
 - A. Clinical presentation
 1. PaO₂ < 60 mmHG
 2. May be asymptomatic
 3. Platypnea
 4. Orthodeoxia
 - B. Pathophysiology
 1. Intrapulmonary vascular abnormalities
 - a. AV shunting
 - b. V/Q mismatch
 - c. Pulmonary capillary dilatation 500mm (vasodilators; vasoconstrictor inhibitors) Normal pulmonary vasoconstriction in response to hypoxemia is blunted
 - d. Diffusion perfusion deficit – DLCO
 - C. Diagnostic testing
 1. ABG's
 2. Response to 100% O₂
 3. Chest x-ray
 4. Pulmonary function test
 5. 2D ECHO
 - D. Management
 1. Almitrin bimesylate
 2. Methylene blue
 3. Garlic extract
 4. Embolotherapy
 5. Shunt
 6. Transplant
- IV. Heptoadrenal Syndrome – Adrenal failure as a result of liver failure
 - A. Decreased amounts corticosteroid-binding globulin and decreased binding affinity
 - B. Glucocorticoid receptor down-regulation – random
 - C. Criteria for heptoadrenal failure
 1. Random cortisol < 20 g/dl with hypoxemic failure, hypotension, requiring pressors
 2. Random cortisol level < 15/dl in non-highly stressed patients
 3. Four groups
 - a. chronic liver failure
 - b. fulminant liver failure
 - c. Post-op transplant steroid free immunosuppression
 - d. Remote liver transplant > 6 months

D. Findings:

1. 72% had adrenal insufficiency - impaired cortisol synthesis
2. Cholesterol precursor for cortisol (HDL preferred)
 - a. apolipoprotein (apo) A-1 major protein component synthesized by liver
 - b. Low HDL lessens response of cosyntropin
 - c. Half life 5.8 days – more affect in chronic liver failure
 - d. Transplant decreases levels (apo)A-1
3. Increased endotoxin levels and proinflammatory mediators (TNF)
 - a. bacterial translocation decreased kuppfer cell activity
 - b. lipopolysaccharide and TNF may inhibit cortisol synthesis
 - c. TNF, IL 1, IL 6 decrease synthesis of (apo)A-1
 - d. TNF decreases number cortisol receptors

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TUBERCULOSIS: Back From the Past With a Vengeance

Scott C. Thigpen

Level: Intermediate

CONTENT DESCRIPTION

Tuberculosis (*Mycobacterium tuberculosis*) is as old as mankind and the most common cause of death due to a single infectious agent worldwide. It is estimated that between 2002 and 2020, approximately 1000 million people will be newly infected, over 150 million people will get sick, and 36 million will die of Tuberculosis (TB) if proper control measures are not instituted. TB was thought to be a disease of the past but a resurgence has been seen in elderly and nursing home patients, immigrants from high-prevalence countries, patients with HIV, homeless persons, alcoholics and illicit drug users. The slow growing tubercle bacillus can live for long periods of time and even healed lesions in the body can come alive later to cause reactivation of the disease. Participants will explore the pathophysiology of pulmonary and extrapulmonary TB and correlate physical assessment findings, chest X-ray findings, and the microscopic examination of acid-fast bacilli (AFB), and sputum cultures with the diagnosis of TB. Extrapulmonary TB can be cultured from infected body tissue or fluid in adrenal glands, bones and joints, gastrointestinal tract, genitourinary tract, lymph nodes, meninges, pericardium, peritoneum and pleura. TB must be viewed based on a multisystem approach. Participants will learn the correct method for administering and reading the Mantoux skin test in healthy individuals and immunocompromised individuals. The pharmacologic management of patients will be reviewed and special consideration will be placed upon the treatment of multidrug-resistant tuberculosis (MDR-TB) infections. Adverse reactions, toxicity, side effects and serial laboratory test monitoring will be outlined. Evidenced based practice strategies will be presented through the use of a case study. Participants will learn how to create healthy work environments through the use of HEPA filters, ultraviolet germicidal irradiation, and negative-pressure isolation rooms to prevent new exposures.

The goal of this session is to equip nurses with the skills to care for patients with TB

LEARNING OUTCOMES

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

1. Explore the epidemiology and pathophysiology of *Mycobacterium Tuberculosis* (TB)
2. Outline the modes of transmission for pulmonary and extrapulmonary TB, diagnostic testing methods, and pharmacologic therapies for the treatment of TB and multiple drug resistance TB.
3. Identify methods for the prevention of TB transmission and strategies for the creation of healthy work environments through infection control measures.

SUMMARY OF KEY POINTS

- I. Tuberculosis: Back From the Past with a Vengeance
- II. Epidemiology
- III. Identification of at risk populations: elderly and nursing home patients, immigrants from high-prevalence countries, patients with HIV, homeless persons, alcoholics and illicit drug users
- IV. Pathophysiology: *Mycobacterium Tuberculosis*
- V. Transmission of *Mycobacterium Tuberculosis*
- VI. Primary Tuberculosis
- VII. Extrapulmonary Tuberculosis: adrenal glands, bones and joints, gastrointestinal tract, genitourinary tract, lymph nodes, meninges, pericardium, peritoneum and pleura.
- VIII. Tuberculosis and HIV Infection
- IX. Diagnostic Testing: Chest Radiograph, Mantoux Testing, Sputum Testing
- X. Pharmacologic Management:
 - A. Isoniazid (INH), Rifampin (RIF), Pyrazinamid (PZA), and Ethambutol (EMB), Streptomycin sulfate, Capreomycin (Capastat) Clofazimine (Lamprene), Cycloserin (Seromycin), Ethionamide (Trecator), Dapsone (Avlosulfon), Ciprofloxacin (Cipro), Levofloxacin (Levaquin)
- XI. Multiple Drug Resistance TB
- XII. Hepatotoxicity: Increased Risk with Ethanol Use, HIV, and Viral Hepatitis
- XIII. Prevention of Disease Transmission
- XIV. Court Mandated DOT (Directly Observed Treatment)
- XV. Healthy Work Environments: HEPA Filters, Ultraviolet Germicidal Irradiation, Negative-pressure Isolations and Personal Respiratory Protection
- XVI. Patient and Community Education

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Tuberculosis Elimination, Education and Training Materials

<http://www.cdc.gov/nchstp/tb/xdrtbupdate.htm#xdrtb> CDC
Division of Tuberculosis Elimination, Extensively Drug-
Resistant Tuberculosis (XDR TB) October 2006

<http://www.cdc.gov/nchstp/tb/pubs/tbfactsheets/ichcs.htm> CDC
Division of Tuberculosis Elimination, Infection Control in
Health-Care Settings April 2006

<http://www.cdc.gov/nchstp/tb/faqs/qa.htm> CDC Division of
Tuberculosis Elimination, Questions and Answers About TB
2005

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Type Necrotizing Fasciitis: Flesh-eating Disease

Sandra O'Sullivan

Level: Beginner

CONTENT DESCRIPTION

Critical care nurses are responsible for the nursing care of many patients with sepsis, but none are more critically ill than those with necrotizing fasciitis. Knowledge of the disease, signs and symptoms, treatment options, multiple complications, and expected clinical outcomes are essential knowledge for the critical care nurse providing quality care to the patient.

Necrotizing fasciitis is a bacterial infection that is characterized by the destruction of skin and the soft tissues beneath it, including fat and the fascia covering the muscles. The tissues often die rapidly and the patient is sometimes said to be infected with 'flesh-eating' bacteria with the most common culprit being *Streptococcus pyogenes*. When this rare, but often fatal (mortality rate about 30%), infection is encountered in the critical care unit, the nurse must be cognizant of the risk factors, pathogenesis, symptoms, diagnosis and treatments for this potentially lethal infection.

This session discusses necrotizing fasciitis using an evidence-based approach from the current research literature and a patient case study. By incorporating the latest research into the care of the patient with necrotizing fasciitis, the critical care nurse can gain an expanded understanding of the condition and its potential effects on the critically ill patient and others.

LEARNING OUTCOMES

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

1. Discuss the pathophysiology and risk factors for the development of necrotizing fasciitis.
2. Identify clinical treatments, expected outcomes, and complications associated with the pathogen causing necrotizing fasciitis
3. Analyze the evidence-based research literature on necrotizing fasciitis and coordinate the team interventions needed to optimize the outcome of a critically ill patient with the infection.

SUMMARY OF KEY POINTS

- I. Epidemiology
 - A. Definition
 1. Insidiously advancing soft tissue infection
 2. Characterized by widespread fascial necrosis
 3. History
- B. Pathogenesis/etiology
 1. Organism spread and effect
 2. Important bacterial factors exist
- C. Risk factors for development: Diabetes mellitus, Surgery, Trauma, Infectious processes, other
- D. Types
 1. Type I – polymicrobial

2. Type II – group A streptococcal or staphylococcal (flesh-eating) and others
 3. Type III – gas gangrene or clostridial myonecrosis – recent trauma or surgery
 4. Variant of Type I is saltwater NF – infection with salt water containing a *Vibrio* species
- II. Pathophysiologic changes d/t infection
 - A. Cellular
 - B. Tissue
 1. After 2-3 days
 2. Multiply rapidly in SQ tissue
 3. Invade the lymphatic system and blood vessels: organism blocks lymph and blood vessels in the area of the wound impeding the immune system's ability to fight the infection
 - C. Organ
 1. Pain
 2. Crepitation occasionally – indicates bacterial gas
 3. Painless ulcers with black necrotic eschar
 - D. System: multi-system organs involved and septicemia leads to severe systemic toxicity and rapid death
 - III. Clinical manifestations and complications
 - A. Integumentary
 - B. Neurologic
 - C. Cardiovascular
 - D. Pulmonary – may be intubated : ventilator management and prevention of secondary infection
 - E. Renal – can be impaired depending on CV response.
 - F. Gastrointestinal – ileus is common
 - G. Hematologic – DIC may occur late or early depending on patient
 - H. Metabolic – depends on nutrition, health status before and stress response
 1. Psychological – support of patient and family
 - IV. Diagnostic studies
 - A. Microbiologic - Determine causative organism
 - B. Radiology –
 - C. CT or MRI scan – more sensitive to demonstrate free air and determines the extent of NF
 - D. Blood profile
 - E. Others
 - V. Diagnostic Criteria
 - A. Mental status changes
 - B. Generalized malaise, tachycardia
 - C. Systemic toxic symptoms
 - D. Focal necrosis with microvascular thrombosis and leukocytes in tissue samples from the involved tissue
 - E. Extensive necrosis of the superficial fascia
 - F. Pain – more severe than expected for appearance of the site
 - G. Little effect of antibiotic treatment

VI. Medical treatment interventions

A. System support

1. Pulmonary- supportive of normal function
2. Cardiovascular – maximum support for function
3. Renal – maintain and monitor for adequate perfusion and removal of wastes. Risk for renal failure is high
4. Gastrointestinal –essential to maintain adequate nutritional status
5. Hematologic
6. Neurologic
7. Metabolic
 - a. Blood sugar monitoring especially with hyperalimentation and tube feedings
 - b. Metabolic profile monitoring and support

B. Treatment

1. Medications to treat the infection
 - a. Antibacterials – antibiotics
 - b. anti-virals - interferons
 - c. Anti-fungals - prototypes
2. Surgical debridement
3. Hyperbaric oxygen therapy – after first two treatments
4. Intravenous immunoglobulin (IVIG)

VII. Wound care

A. Dressings

1. Antimicrobial –
2. Advanced dressings-vacuum- assisted devices
3. Bioengineered tissues

B. Hyperbaric oxygen

C. Wet-to-dry or moist - STRICT STERILE TECHNIQUE

D. Other considerations in wound management

VIII. Expected clinical outcomes

- A. Eradicate the infection
- B. Prevent complications
- C. Reduce morbidity

IX. Nursing diagnoses – functional health patterns

- A. Tissue integrity, altered
- B. Tissue perfusion, altered
- C. Risk for altered fluid volume, deficit
- D. Altered cardiac output
- E. Ineffective breathing pattern
- F. Altered nutrition, decreased
- G. Psychosocial support

X. Current research and application to functional health patterns – articles related to each nursing diagnosis or functional health pattern– see reference list. Most references talk of cases

A. Pictures and cases on the internet

B. Internet sites

1. eMedicine Specialties Dermatology .> BACTERIAL
2. <http://www.lib.uiowa.edu/hardin/md/necrotizing-fasciitis.html>
3. MEDLINEplus Health Encyclopedia: National Library of Medicine
4. National necrotizing fasciitis foundation website:
5. 37,800 sites on Google!

XI. Case study – summary

XII. Questions and answers

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Understanding the Shock Syndrome

Christie Artuso

Level: Intermediate

CONTENT DESCRIPTION

Shock is simply a process that eventually results in the systematic failure of all body systems from a gradual decrease in tissue perfusion. Early detection and recognition of the progressive clinical sequelae that accompany the shock syndrome is critical to patient outcomes and decreasing morbidity and mortality. The acute care clinician must have a thorough understanding of the three major categories of shock, the precipitating conditions that can lead to the development of this often fatal condition, and the interventions that will halt its progression. Early detection, early intervention, and close observation are key components that lead to optimal patient outcomes. Participants will develop with an understanding of the pathophysiology of the shock syndrome, the three major categories of shock (cardiogenic, hypovolemic, and distributive), and the sub-categories associated with distributive shock (septic, neurogenic, anaphylactic). Patient case studies will be used to illustrate the clinical presentation of each category of shock. The phases of the shock syndrome will be discussed as well as critical nursing interventions associated with each phase. A final case study will discuss the presentation of a post-partum patient who developed septic shock from an unknown and undetected infection. The lessons learned through understanding the pathophysiology of shock and its clinical sequelae will optimize patient outcomes in any acute care setting and decrease the need for critical transfers to an intensive care unit during the latter phases of this often fatal syndrome.

LEARNING OUTCOMES

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

1. Identify the three major categories of shock
2. Identify the three sub-categories of distributive shock and appropriate interventions associated with each
3. Identify compensatory mechanisms of shock and the clinical sequelae associated with early compensated shock and uncompensated [progressive] shock.

SUMMARY OF KEY POINTS

- I. Defining shock
 - A. Definitions and description
 - B. Classifications
 1. Hypovolemic
 2. Cardiogenic
 3. Distributive
 - a. Anaphylactic
 - b. Neurogenic
 - c. Septic

- II. Hypovolemic
 - A. Definition
 - B. Common Causes
 - C. Populations affected
 - D. Case study
- III. Cardiogenic
 - A. Definition
 - B. Common Causes
 - C. Case Study
- IV. Distributive
 - A. Definition and Types
 1. Anaphylactic
 - a. Common Causes
 - b. Presentation
 - c. Management
 2. Neurogenic
 - a. Common Causes
 - b. Presentation
 - c. Management
 3. Septic
 - a. Common causes
 - b. Presentation
 - c. Management
 - V. Phases of Shock
 - A. Compensatory
 1. Compensatory mechanisms
 2. Clinical pearls!
 - B. Progressive
 1. Clinical presentation
 2. Treatment modalities
 - VI. Case Study Illustration

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Undifferentiated Shock: A Systematic Diagnostic Approach

Timothy R. Wolfe

Level: Intermediate

CONTENT DESCRIPTION

Evaluation and treating acutely unstable patients requires a clear mind and a broad differential diagnostic list to ensure all possible causes of instability are considered. Because this situation is invariably stressful and time dependent, memory tools such as pneumonics are extremely useful. An easily remembered mnemonic for the evaluation of undifferentiated hemodynamic instability is END-SHOCK Kills. This stands for Endocrine and Environmental shock (E), Neurogenic shock (N), Drug induced shock (D), Septic shock (S), Hypovolemic shock (H), Obstructive shock (O), and Cardiogenic shock (C). The K stands for Kills. This session will use real life, seemingly confusing and complex case studies to demonstrate how this mnemonic can be easily applied to systematically evaluate patients with undifferentiated shock. Additional information of special focus will be a discussion on the subtle clinical findings suggesting a patient has tissue hypoperfusion well before they develop hypotension. Several cases will also assist in bring out the concepts of toxidromes, relative bradycardia, disease specific arrhythmias that should not be treated with standard ACLS methods and ventilator induced instability from high intrathoracic pressures. The lecture is appropriate for all levels of ICU and ER nurses, but may be especially appreciated by nurses with a few years of clinical experience who wish to enhance their rapid diagnostic thinking skills.

LEARNING OUTCOMES

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

1. Develop an easily remembered differential diagnosis for shock – allowing you to rapidly assess most unstable emergency department (and ICU) patients.
2. Recognize subtle clinical hints suggesting a patient is in shock.
3. Discuss unique or special situations where shock presentations or interventions may not seem intuitive.

SUMMARY OF KEY POINTS

- I. Develop an easily remembered differential diagnosis for shock – allowing you to rapidly assess most unstable emergency department (and ICU) patients.
 - A. Mnemonic for hemodynamic instability: END-SHOCK KILLS
 - E - Endocrine (Adrenal insufficiency, hypothyroid, DKA),
Environment (hypo-hyperthermia, anaphylaxis)
 - N - Neurogenic
 - D - Drug induced (is there a toxidrome?)
 - S - Sepsis
 - H - Hypovolemic (bleeding, dehydration)

O - Obstructive (tension pneumo/ITP, tamponade, IAH/ACS, PE)

C - Cardiogenic

K – Kills

II. Multiple case studies will be presented with clinical scenarios, lab tests, ECG, X-ray and CT findings. Each of these cases will demonstrate a different cause of hemodynamic instability that is often obscured unless careful attention is paid. By managing these complex cases, the attendee will see how the use of a standardized approach can assist them coming to the correct diagnosis in a timely fashion even with a difficult presentation.

A. Recognize subtle clinical hints suggesting a patient is in shock.

Restless, Anxious, Agitated, Confused!!

Tachypnea

Thirsty

Cool, moist skin, mottled extremities

Oliguria

Tachycardia, narrow pulse pressure

Hypotension – late finding!!

III. Discuss unique or special situations where shock presentations or interventions may not seem intuitive.

A. Some patients with drug induced toxidromes, electrolyte imbalances, arrhythmias, hormonal deficiencies and electrolyte disturbances become unstable and can be very tricky to sort out. Several cases will include examples of these unique scenarios to emphasize these findings in a clinical scenario.

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Update on the Pharmacotherapy of Acute Hypertension

Joseph F. Dasta

Level: Advanced Practice

CONTENT DESCRIPTION

The purpose of this session is to review the pharmacology, pharmacokinetics, dosing considerations, adverse drug reactions, and monitoring parameters of parenteral drugs used to acutely lower blood pressure. I will also present data on a recent survey of drug usage for this condition.

LEARNING OUTCOMES

By the end of this session the participant will be able to:

1. List the drugs and drug classes used to treat acute hypertension.
2. Describe the benefits and hazards of each drug
3. Appreciate the need to develop guidelines for acute hypertension

SUMMARY OF KEY POINTS

- I. Drugs currently used for acute hypertension
 - A. Labetolol
 - B. Sodium nitroprusside
 - C. Nicardipine
 - D. Hydralazine
 - E. Enalaprilat
- II. Drugs in development
 - A. Clevidipine
- III. Discussion points for each drug
 - A. Pharmacology

- B. Pharmacokinetics
 - C. Adverse drug reactions
 - D. Dosing
 - E. Monitoring
 - F. Role in therapy
- IV. Survey of acute hypertension
 - A. Target blood pressure in stroke and non-stroke patient
 - B. Drugs of first choice
 - C. Drugs of second choice
 - D. Which monitoring parameters are used
 - E. Are guidelines used?

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Using AACN Practice Alerts to Improve Patient Outcomes

Nancy Richards
Maureen Seckel
Debra Kramlich
Mark Weber
Kate Moore
Anna Bourgault

Level: Beginner

CONTENT DESCRIPTION

Would you like to provide excellent care that improves patient outcomes? Are you interested in knowing how your unit or your own practice compares to national standards in critical care? Join us for an informative session to explore the history and implementation of AACN Practice Alerts. Practice Alerts contain evidence-based practice expectations and the rationale for cutting edge and problematic issues in critical care practice. We will also provide easy to use tips for implementing Practice Alerts on your unit. Whether you are a staff nurse or a nurse leader, novice or expert, you will leave this session feeling empowered to be a change champion for evidence-based practice.

LEARNING OUTCOMES

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

1. Validate and expand knowledge on current AACN Practice Alerts.
2. Identify practice gaps in your own critical care unit.
3. Increase awareness of the Practice Alert toolkits to facilitate implementation of changes in practice.

SUMMARY OF KEY POINTS

- I. Introduction
- II. History of AACN Practice Alerts
 - A. Intent of Practice Alerts
 - B. Process for development
 - C. Role of the Evidence Based Practice Resource Work Group
 - D. Keeping up with new evidence
- III. Availability of AACN Practice Alerts
 - A. Website @www.aacn.org
 - B. AACN News: new releases and revisions
 - C. AJCC: new releases and revisions
- IV. Future Directions for Practice Alerts
 - V. Detailed Overview: 2008 Revisions to Selected Practice Alerts
 - A. Ventilator Associated Pneumonia
 1. Expected practice
 2. Scope and impact of the problem
 3. Supporting evidence
 4. Actions for nursing practice
 5. Education program
 6. Data collection instructions and audit tool

- B. Dysrhythmia Monitoring
 1. Expected practice
 2. Scope and impact of the problem
 3. Supporting evidence
 4. Population specific information
 5. Actions for nursing practice
 6. Education program
 7. Data collection instructions and audit tool
- C. ST Segment Monitoring
 1. Expected practice
 2. Scope and impact of the problem
 3. Supporting evidence
 4. Population specific information
 5. Actions for nursing practice
 6. Education program
 7. Data collection instructions and audit tool
- VI. Implementation of AACN Practice Alerts
 - A. Tips to get started
 - B. Toolkits
 - C. Audit tools
 - D. Role of staff nurse to nurse leader
- VII. Summation/Discussion/Questions

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