

Geriatric Syndromes

M. Lynn Rodgers

Level: Beginner

CONTENT DESCRIPTION

Geriatric Syndromes are groups of specific signs and symptoms that occur more often in the elderly and can impact patient morbidity and mortality. Normal aging changes, multiple co-morbidities, and adverse effects of therapeutic interventions contribute to the development of Geriatric Syndromes. These syndromes are becoming increasingly important for the nurse to consider as our patient population ages and have been included in AACN's 2006 edition of its Core Curriculum for Critical Care Nursing. This session will discuss these Geriatric Syndromes, including sleep disorders, problems with eating or feeding, incontinence, confusion, falls, and skin breakdown. Delirium, depression, dementia, and pain will also be addressed. Prevalence, etiology, prevention, and nursing interventions for each of these syndromes will be outlined. Several age-appropriate assessment tools and scales will be presented to assist the participants in identifying those elderly patients at risk for these Geriatric Syndromes. This session is appropriate for any novice or expert nurse that cares for the geriatric patient in any inpatient or outpatient practice setting. Basic knowledge of geriatric growth and development is a prerequisite for this session. By the end of this presentation, participants will have a better understanding of commonly encountered signs and symptoms as applied to the geriatric patient. With an enhanced knowledge of Geriatric Syndromes, assessment, care, and management of elderly patients can be improved and age-specific complications may be decreased.

LEARNING OUTCOMES

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

1. Define Geriatric Syndromes and their contributing factors.
2. Identify assessment tools to evaluate Geriatric Syndromes.
3. Discuss nursing interventions that can improve management of Geriatric Syndromes.

SUMMARY OF KEY POINTS

I. Introduction

- A. Americans are living longer
 1. The number of older Americans is growing
 2. The proportions of older Americans is growing
- B. Atypical Presentations in Older Patients
 1. Functional decline
 2. Misleading symptoms
 3. Signs of one disease obscured by another
 4. No presentation at all
- C. Elderly Are Different
 1. 1/3 related to disease processes
 2. 1/3 related to disuse/activity
 3. 1/3 related to aging process

II. What are Geriatric Syndromes?

- A. Definition
 1. Broad categories of signs and symptoms common in the geriatric population
 2. Major focus for nursing research and best practice guidelines
 3. Great impact on geriatric morbidity and mortality
- B. Contributing Factors
 1. Normal changes of aging
 2. Multiple diagnoses
 3. Adverse effects of therapeutic interventions
 4. Polypharmacy
 5. Polyprovider

III. SPPICES Pneumonic for Geriatric Syndromes

- A. Sleep Disorders
- B. Problems with Eating or Feeding
- C. Pain
- D. Incontinence
- E. Confusion
- F. Evidence of Falls
- G. Skin Breakdown

IV. Sleep Disorders

- A. Etiology
 1. Age Specific
 - a. Quantity of sleep in 24 hours is unchanged
 - b. More daytime naps
 - c. Quality of night time sleep decreases
 - d. Increase in Sleep Stages 1 and 2
 - e. Decrease or Absence in Sleep Stages 3 and 4
 - f. Less tolerance to changes in sleep-wake cycle
 2. Environmental Factors
 - a. Noise
 - b. Room Temperature
 - c. Uncomfortable Mattress
 - d. Bedfellow Habits
 - e. Strange Environment
 3. Medication Adverse/Side Effects
 - a. Antidepressants
 - b. Caffeine
 - c. Analgesics
 - d. Diuretics
 4. Disease Related
 - a. BPH
 - b. Heart Failure/Angina
 - c. Over Active Bladder
 - d. GERD
 - e. COPD
 - f. Restless Leg Syndrome
 - g. Sleep Apnea
 - h. Musculoskeletal/Arthritis Pain

- i. Anxiety/Depression
 - j. Dementia
- B. Results of Sleep Deprivation in the Elderly
 - 1. Forgetfulness
 - 2. Disorientation/Confusion
 - 3. Cognitive Impairment
 - 4. Restlessness/Wandering/Falls
 - 5. Depression
 - 6. “Sundowner Syndrome”
 - 7. Use of OTC Sleep Aids
- C. Sleep Assessment
 - 1. Determine bedtime patterns and rituals
 - 2. Determine amount of sleep and nap time
 - 3. Determine prescription/OTC drugs and herbs taken
 - 4. Determine sleep environment
 - 5. Determine bathroom needs during sleep
- D. Interventions to Promote Sleep
 - 1. Normalize sleep patterns/rituals
 - 2. Time medications appropriately
 - 3. Avoid stimulants before sleep
 - 4. Natural light in afternoon
 - 5. Routine exercise
 - 6. IF needed use medications cautiously
 - 7. Consider side effects of medications prescribed
 - 8. Evaluate/treat medical problems first
- V. Problems with Eating or Feeding/Nutritional Issues
 - A. Etiology - DETERMINES
 - 1. Disease
 - a. 80% have diagnoses that cause nutritional problems
 - b. Dietary limitations due to diagnoses
 - c. Dyspnea/Heart Failure/COPD
 - d. Pressure Ulcers
 - e. Cancer
 - 2. Eating Poorly
 - a. Poor food choices
 - b. Few fresh fruits/vegetables
 - c. Use of prepackaged/preprepared foods
 - 3. Tooth Decay/Oral Pain
 - a. Poorly fitting dentures
 - b. Poor dental health
 - c. Less dentist care
 - d. More oral disease
 - 4. Economic Hardship
 - a. 40% have income less than \$6000 a year
 - b. Less money for food and transportation
 - c. Healthier food is more expensive
 - 5. Reduced Social Contact
 - a. One third live alone
 - b. Mealtime is lonely
 - c. Food is uninteresting
 - d. Many don’t cook for just themselves
 - e. Depression results in decreased interest in eating
 - B. Multiple Medications
 - a. Decreased appetite is a common side effect
 - b. Dehydration, constipation, nausea, and vomiting are common
 - c. Some medications can cause decrease in nutrient absorption
 - d. Many medications require dietary alterations
 - e. Some patients get “full” of pills and can’t eat
 - 7. Involuntary Weight Loss/Gain
 - a. Basic metabolic rate decreases
 - b. Need for Vitamin D, B6, calcium, and fiber increases
 - 8. Need Assistance with Self Care
 - a. Trouble with walking and shopping
 - b. Unable to cook
 - c. Can’t feed self well
 - 9. Elderly More than 80 Years
 - 10. Sensory Impairment
 - a. “We eat with our senses”
 - b. Decrease in ability to smell and taste food
 - c. Vision and hearing problems can impair transportation and cooking
 - B. Signs of Malnutrition
 - 1. Pale skin with poor turgor
 - 2. Dull eyes
 - 3. Swollen lips/dry swollen red tongue
 - 4. Extreme thinness/muscle wasting/edema
 - C. Signs of Dehydration
 - 1. Orthostatic dehydration/decreased urine/dizziness
 - 2. Confusion/Irritability
 - 3. Poor skin turgor/dry mucous membranes
 - 4. If severe, shock-like symptoms
 - D. Signs of Vitamin and Mineral Deficiency
 - 1. Oral cavity - cheilosis
 - 2. Eyes - scleral changes, Bitot’s Spots
 - 3. Face - seborrhea-like dryness/redness of nasolabial folds and eyebrows
 - 4. Upper extremities- purple blotches, “cellophane skin”
 - 5. Lower extremities - epidermis/scalp flaking, cracking between islands of hyperkeratosis
 - 6. Abdomen/buttocks - waxy, perifollicular hyperkeratosis
 - E. Nutritional Assessment
 - 1. Examine mouth/oral cavity/skin
 - 2. Evaluate serum albumin, hgb, hct, B12, iron, and transferrin levels
 - 3. Assess medication regime
 - 4. Review comorbidities
 - 5. Consider usual weight
 - 6. Obtain diet history - intake for 5-7 days
 - 7. Look for patterns, preferences, percentages
 - 8. Determine socioeconomic and cultural factors
 - 9. Use Mini Nutritional Assessment or Nutritional Self Assessment Checklist

- F. Intervention
 1. Consult dietitian
 2. Involve family and patient
 3. Use community resources
 4. Prevent dehydration
 5. Prevent food/drug interactions
 6. Resolve feeding and environmental problems
 7. Consider appetite stimulants
- VI. Pain
 - A. Myths of Pain in the Elderly
 1. Normal part of aging
 2. Have higher pain tolerance
 3. They complain a lot about pain
 - B. Truths about Pain in the Elderly
 1. Contributes to depression, anxiety, decreased socialization, and decreased functional ability
 2. Those with dementia/cognitive impairment may not be able to express pain
 3. Smaller doses of opiates may be needed due to decreased drug clearance/metabolism
 4. Elderly are at risk of undertreatment of pain.
 5. Pain treatment is a major element in improving QOL
 6. Consider existing chronic pain and its treatment when treating acute pain
 - C. Recognizing Pain in the Elderly
 1. Sources of pain
 2. Behavioral responses
 3. Sympathetic responses
 4. Parasympathetic responses
 - D. Treatment of Pain in the Elderly
 1. Medications to Avoid/Use Cause Cautiously
 - a. 48 drugs listed in Beer's Criteria
 - b. Propoxyphen
 - c. Meperidine
 - d. Mixing agonist/antagonist analgesics with agonist analgesics
 - e. NSAIDS
 2. Analgesics for the Elderly
 - a. Acetaminophen
 - b. Methadone
 - d. Fentanyl patches
 - e. Lidocaine patches/gel
 3. Adjunctive Drug Therapies
 - a. Antiemetics
 - b. Antihistamines
 - c. Phenothiazine
 - d. Steroids
 - e. Capsaicin
 - f. Neurontin, Lyrica for neuropathic pain
 - g. Antidepressants - SSRIs
 4. Pain Relief in Acute Care
 5. Non-Pharmacological Pain Relief
 - E. Principles of Pain Management in the Elderly
 1. ALWAYS ask about pain
 2. Pain is what the patient reports
- 3. Pain is a tremendous impact on QOL
- 4. Determine cause of pain but treat even without determining cause
- 5. Use drug and nondrug therapies
- 6. Consider drug costs, insurance coverage, and pharmaceutical company patient assistance programs
- 7. Consider drug side and adverse effects, drug - drug and drug - food interactions
- 8. Treat depression and anxiety
- 9. Address drug addiction, dependence, and tolerance
- 10. Evaluate treatment plan regularly
- 11. Keep patient and family involved and assure they understand the plan
- VII. Incontinence
 - A. Impact of Incontinence on QOL
 1. Physical
 2. Psychological
 3. Social
 4. Domestic
 5. Occupational
 6. Sexual
 - B. \$16.3 Billion Annually – \$12.4 Billion for Women/\$3.8 Billion for Men
 1. Routine Care – 70%
 2. Nursing Home Admissions – 14%
 3. Treatment – 9%
 4. Complications – 6%
 5. Diagnostic Workup – 1%
 - C. Types of Urinary Incontinence
 1. Urge - due to detrusor irritability and abnormal bladder contraction
 2. Stress - due to reduced sphincter resistance and sudden increased abdominal pressure
 3. Overflow - due to over distention of bladder that exceeds outlet resistance
 4. Functional - due to physical or psychological inability to reach toilet facilities
 - D. Causes of Urinary Incontinence
 1. Pelvic floor laxity
 2. Stool impaction or constipation
 3. Diabetes mellitus or insipidus
 4. Spinal cord injury
 5. Neurological disease
 6. Delirium or depression
 7. Restricted mobility
 8. Infection
 9. Atrophic vaginitis/urethritis
 10. Prostate problems
 11. Fluid, alcohol, caffeine intake
 12. Urological or gastrointestinal surgery
 13. Medications - anticholinergic, diuretics, narcotics
 - E. Questions to Ask
 1. Do you have any frequency, urgency, or burning?
 2. Do you ever sneeze/cough and wet your pants?
How often do you go at night?

3. Do you ever limit fluids or hold diuretics?
 4. Do you dribble getting to the bathroom ?
 5. Do you plan your life around bathroom availability?
 6. Do you wear a pad?
- F. Behavior Modification
1. Stop and stay still
 2. Squeeze pelvic floor muscles
 3. Relax rest of body
 4. Concentrate on suppressing urge
 5. Wait until the urge subsides
 6. Walk to bathroom at normal pace
- G. Other Modifications
1. Adjust daily fluid intake and reduce nighttime fluids
 2. Eliminate bladder irritants such as caffeine, alcohol, nicotine
 3. Prevent constipation
 4. Adjust timing of medications
 5. Treat infections
 6. Improve and arrange environment conditions
 7. Bladder and habit retaining
 8. Intermittent catheterization
 9. Absorbent pads/adult diapers
- H. Other Medical Procedures or Devices
1. Urodynamic evaluation
 2. Surgery
 3. Collagen injections
 4. Clamping devices
 5. Pessary placement
- I. Medication Therapy
1. Anticholinergic/Antimuscarinic Agents
 2. Antidepressants - Sinequan, Amyltriptyline
 3. Alpha Agonist agents for stress incontinence
 4. Vaginal estrogen cream
 5. Sleeping medication
- J. Fecal Incontinence
1. Affects 2-7% of adults
 2. Affects 45% of nursing home residents
 3. Associated with increased mortality
 4. 2/3 will not discuss with healthcare provider
- K. Causes of Fecal Incontinence
1. Decreased mental function
 2. Physical disease
 3. Medication effect - quinidine, antibiotics, magnesium containing antacids, levadopa, colchicine, theophylline, NSAIDS, lactulose, sorbitol in sugar free foods
- L. Questions to Ask
1. How many BMs do you have daily?
 2. Do you have trouble with your bowels?
 3. What do you use to help your bowels?
 4. Do you ever not make it to the bathroom or wear protective undergarments?
 5. What type of toilet facilities do you have?
- M. Treatment of Fecal Incontinence.
1. Identify cause
2. Complete history and physical
 3. Modify patient environment
 4. Modify diet
 5. Evaluate and change medications
 6. Decrease stool frequency
 7. Improve stool consistency
 8. Bowel retraining
 9. Surgery
- VIII. Confusion: Delirium, Depression, Dementia
- A. Dementia
1. Definition - organic mental disorder with slow loss of intellectual abilities that interfere with functioning
 2. Characteristics
 - a. Aphasia
 - b. Apraxia
 - c. Agnosia
 - d. Decreased intellectual function
 - e. Personality changes
 - f. Loss of short term memory early; long term memory later
 - g. Inability to recognize people
 - h. Uncovered or worsened by stress of hospitalization
 - i. Increased risk of delirium
 3. Behaviors
 - a. Paranoia, delusions, illusions, hallucinations
 - b. Wandering, insomnia, undressing
 - c. Incontinence
 - d. Increased risk of delirium
 - e. Harm self or others, yelling or screaming
 - f. Repeating questions
 - g. Inappropriate sexual behaviors
 4. Causes
 - a. Alzheimers
 - b. Vascular - multi-infarct
 - c. Brain tumor
 - d. AIDS
 - e. Huntington's Disease
 - f. Parkinson's Disease
 - g. B12 or folate deficiency
 - h. Long term alcohol abuse
 - i. Hypothyroidism
 - j. Untreated syphilis
 - k. Chronic liver or kidney disease
 5. Assessment
 - a. History and physical
 - b. Appropriate diagnostic exams
 - c. Mini Mental Status Exam
 6. Treatment for Dementia
 - a. Identify and treat cause
 - b. Avoid anticholinergic agents
 - c. Antipsychotic agents
 - d. Hormone therapy
 - e. Medications - Aricept, Namenda, Exelon, Reminyl

- B. Delirium
 - 1. Definition - reversible abrupt impairment of global cognitive impairment
 - 2. Characteristics
 - a. Acute confusional state and encephalopathy
 - b. Altered mental state
 - c. 25% of hospitalized elderly
 - d. 65% of postoperative elderly greater than 70 years old
 - e. Increased risk of death or poor outcome
 - 3. Behaviors
 - 4. Causes
 - a. Drug use
 - b. Electrolyte imbalance
 - c. Lack of drugs (withdrawal or pain)
 - d. Infection
 - e. Reduced sensory input
 - f. Intracranial events
 - g. Urinary incontinence/fecal impaction
 - h. Myocardial infarction
 - 5. Confusion Assessment Method (CAM) Diagnostic Algorithm
 - 6. Treatment for Delirium
 - a. Determine cause and treat
 - b. Medication for behavior

- C. Depression
 - 1. Statistics
 - a. 10-15% of all elderly
 - b. 43% of those in acute care
 - c. 75% of LTC residents
 - 2. Signs and Symptoms
 - a. Vegetative
 - b. Psychological
 - 3. Behaviors
 - a. Sleep changes
 - b. Interest in activities decreases
 - c. Guilt or remorse
 - d. Energy decreased
 - e. Concentration problems
 - f. Appetite changes
 - g. Psychomotor agitation or retardation
 - h. Suicidal thoughts
 - 4. Assessment/Treatment
 - a. Geriatric Depression Scale
 - b. Evaluate for suicide risk
 - c. Evaluate for physical causes
 - d. Medication/treatment

IX. Evidence of Falls

- A. Statistics
 - 1. 25% occur in 70 year old
 - 2. 35% occur in those over 75 years
 - 3. 85% occur in the home
 - 4. Injury rate in hospitals from falls is 10-25%
 - 5. 10% fatal falls involve stairs
 - 6. 15-33% of falls in the elderly result in injury
 - 7. 6% of elderly medical expenses are from falls

- 8. 5% of falls result in fractures
- 9. 60% of all nursing home resident fall yearly

B. Who Falls?

- 1. Active elderly fall more
- 2. Frail elderly have more repeated falls

C. Contributory Factors of Falls in the Elderly

- 1. Medication Effects
- 2. Sensory Deficits
- 3. Physical Limitations
- 4. Cognitive Problems
- 5. Environmental Traps

D. Assessment for Fall Risk

- 1. Hendrich II Fall Risk Model
- 2. Get Up and Go Test

E. Fall Prevention

- 1. Lifestyle changes
- 2. Physical and Occupational Therapy
- 3. "Fall proof" the environment
- 4. Evaluate medications

X. Skin Breakdown

A. Statistics

- 1. Affects 1 million adults
- 2. Present in 5-10% of all those in healthcare settings
- 3. One year mortality is 40%
- 4. Cost is \$15,000 per ulcer
- 5. Annual cost in US is \$1.5 billion a year

B. Age Related Skin Changes

C. Pressure Ulcers

- 1. Can add 16 days to a hospital stay
- 2. Pain in 2/3 of those with Stage II or greater
- 3. Associated with cellulitis, osteomyelitis, abscesses, and sepsis
- 4. Increases morbidity and mortality

D. Staging of Pressure Ulcers

- 1. Stage I
- 2. Stage II
- 3. Stage III
- 4. Stage IV

E. Braden Risk Assessment

F. Prevention of Skin Breakdown

G. Treatment of Skin Breakdown

XI. Summary

BIBLIOGRAPHY/WEBLIOGRAPHY

- International Continence Society: www.icsoffice.org
- Inouye, SK, et al. Clarifying confusion: the confusion assessment method. A new method for detection of delirium. *Ann Intern Med.* 1990; 113(12):941-948.
- Mathias S, Nayak USL, Isaacs B. Balance in elderly patients: the "get-up and go" test. *Arch Phys Med Rehabil.* 1986;387-389. Add More
- The Hartford Institute for Geriatric Nursing: www.hartfordign.org

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Getting it Write: Reviewing and Revising for Publishing Success

Sarah A. Martin

Level: Beginner

CONTENT DESCRIPTION

The purpose of this session is to present an overview of the manuscript review process to facilitate successful publishing activities by interested participants. Prospective authors will be apprised of by whom and what reviewers are looking for. The components of a review, how feedback is given and tips for revision and resubmission will be described. Examples of reviews will be presented and the use of an electronic editorial system will be described. The production process following manuscript acceptance will be summarized. The goal of this session is to encourage reluctant clinicians fearful of the review process to publish their practice.

LEARNING OUTCOMES

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

1. Discuss the composition of an editorial board and review panel.
2. List ten essential facets of a manuscript that a reviewer will critique.
3. Describe the process of submitting a manuscript, format that feedback will be tendered and appropriate author response.

SUMMARY OF KEY POINTS

- I. Who reviews manuscripts?
 - A. You and a trusted colleague
 - B. Seek doctorally prepared colleague for research
 - C. Editor(s)
 - D. Member of an editorial board
 - E. Reviewer(s)
 - F. Journal Specific-double blind, open, self-selected
- II. Associate Editor Pearls
 - A. Negotiate authorship as step #1
 - B. Computer access
 - C. Accordion file
 - D. Index cards
 - E. Archiving electronic files
 - F. "Writing" books
- III. The Submission Process
 - A. Only submit to one journal
 - B. Acknowledge ethics statement
 - C. Sign copyright release
 - D. Follow author guidelines
- IV. Manuscript Submission
 - A. Electronic Editorial Systems
 - B. Some journals you submit electronic files
 - C. Some journals you submit electronic files/hard copies

- V. Ethical Editorial Consideration
 - A. Plagiarism
 - B. Self plagiarism
 - C. Research data
 - D. Council of Science Editors White Paper
- VI. Review Components
 - A. Confidential process
 - B. The big picture versus the details
 - C. Probable timeline
- VII. Essential Facets of the Review Procedure
 - A. The title
 - B. Originality
 - C. Appropriate topic/style for journal
 - D. Organization of content
 - E. Accuracy of content
 - F. Completeness of content
 - G. Grammar
 - H. References (content and format)
 - I. Tables and figures
 - J. Research-Use the research appraisal checklist
- VIII. Feedback from Reviewers/Examples of Reviews
 - A. Generally on journal review template form
 - B. May use WORD function "Track Changes"
 - C. General categories: accept, accept with revisions, revise and resubmit, reject
 - D. Editor(s) will make final determination
 - E. Open review sites (www.nursing-research-editor.com/authors/open-enrs.php)
- IX. Revision and Resubmission
 - A. Goal is to make your manuscript the best that it can be
 - B. Expect to revise
 - C. Respond to feedback in writing if not in agreement with review
 - D. Timely resubmission
 - E. Resubmit with different reviewers possible
- X. Success
 - A. Acceptance
 - B. Production process
 - C. Review of galleys and queries
 - D. Celebrate

BIBLIOGRAPHY/WEBLIOGRAPHY

- Alspach JG. What journal editors would like from reviewers. *Crit Care Nurse*. 1994;14(6):13-16.
- Barnum BS. *Writing and Getting Published: A Primer for Nurses*. New York, NY:Springer Publishing Company, Inc.; 1995.
- Dougherty MC. The value of peer review. *Nurs Res*. 2006;55:73-74.

Nelms BC. The importance of the review process-and reviewers.
J Pediatr Health Care. 2006;20:1-2.

Oermann MH, Wilmes, NA. How accurate are references in
nursing journals? Nurse Author Ed. 2005;15(4):1-4.

Scott-Lichter D, and the Editorial Policy Committee, Council of
Science Editors. CSE's White Paper on Promoting Integrity
in Scientific Journal Publications, Reston, Va: CSE; 2006

Truss L. Eats, Shoots & Leaves. New York, NY: Gotham
Books.; 2004.

Nurse Author & Editor. Available at [http://www.
NurseAuthorEditor.com](http://www.NurseAuthorEditor.com). Accessed on December 7, 2007.

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Giving and Receiving Bad News: Lessons I Learn

Rhonda S. Fishel

Level: Intermediate

CONTENT DESCRIPTION

Conveying bad news is one of the most difficult yet important responsibilities in healthcare. It is even more challenging when the family is not expecting to receive bad news. Receipt of bad news results in varying degrees of anxiety, uncertainty, confusion, helplessness, and loss of control. In our role as patient advocate, nurses must have the knowledge and tools to provide support to the patient and family. This presentation focuses on 3 scenarios in which bad news is given: sudden death, end of life situations, and receipt of a bad diagnosis—such as cancer. Strategies based on experience and evidence based medicine are offered to aid nurses with this task. The session explores the Provider/Patient relationship and offers insight as to how it can be mutually beneficial and inspirational. Participants will also learn what it means to receive bad news and how this can affect health-care workers.

LEARNING OUTCOMES

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

1. Discuss the barriers to delivering bad news to patients and families.
2. Discuss at least one technique for delivering bad news: bad diagnosis/death.
3. Describe how receiving bad news affects us as health care providers and how we can use what we learn to help our patients.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Delivering bad news – a multidisciplinary responsibility
 - A. What is bad news?

- B. What can get in the way?
 - C. What does the patient/family hear?
 - D. Sudden death
 - E. Bad diagnosis
 - F. End of life issues
- III. Evidence-based approaches
 - A. Patient-Provider Communication
 1. SPIKES Protocol
 2. Communication with compassion
 3. How the patient family respond
 4. The concept of service
 - B. Patient-centered approaches
- IV. A Personal Perspective
 - A. Hearing bad news
 - B. What helps
 - C. Looking to the future
- V. Summary

BIBLIOGRAPHY/WEBLIOGRAPHY

- Baile WF, Buckman R, Lenzi R, Glober G, Beale EA, Kudelka AP. SPIKES—A six-step protocol for delivering bad news: Application to the patient with cancer. *The Oncologist*, 2000; 5 (4): 302-311.
- Miller J. & Cutsall S. *The Art of Being a Healing Presence*. Willow Green Publishing: Ft Wayne, IN, 2001.
- Remen R. *My Grandfather's Blessings: Stories of Strength, Refuge, and Belonging*. Riverhead Books: New York, New York, 2001.
- Vandekeift G. Breaking bad news. *American Family Physician* 1999; 64(12): 1975-1978.

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Giving Bad News: A Patient and Family Centered Approach

Becky Pierce

Level: Beginner

CONTENT DESCRIPTION

This presentation will explore the challenges and successes of truly partnering with patient families in the ICU setting. Included are findings from a quality improvement project where 50 families of patients who died in the ICU were interviewed. The results provided powerful motivation to increase family presence and provide more meaningful opportunities for families to share their perspective. A review of the concepts of patient and family centered care will be discussed. The Institute for Healthcare Improvement's Collaborative Model for Change was used on an institutional level with great success. This process as well as the challenges of culture change in the ICU and throughout the hospital will be shared. Harborview Medical Center received the Society of Critical Care Medicine's award for Patient and Family Centered Care in 2008 because of this work. The speaker was one of the co-chairs of the learning collaborative work at the medical center and will share the concepts, successes and pitfalls to avoid as we all work toward transforming care in the ICU by implementing Patient and Family Centered Care in a genuine way.

LEARNING OUTCOMES

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

1. Discuss concepts of Patient and Family Centered Care.
2. Recognize the power of direct feedback from and involvement of patient and families in design of best practices - especially regarding communication in stressful situations.
3. Discuss the utilization of the IHI Collaborative Model for Improvement for a hospital wide initiative.

Going for the Gold: Reclaiming our Priorities with Evidence-Based Practice

Margo A. Halm

Level: Intermediate

CONTENT DESCRIPTION

In *Crossing the Quality Chasm*, the Institute of Medicine (IOM) called for radical redesign of the healthcare system to improve the safety and quality of care. In an effort to reduce the research-practice gap, one of the report's main recommendations was to advance knowledge and skill development of clinicians from all disciplines in evidence-based practice (EBP). In order to ensure critical care nursing practice is based on the most current evidence and thus, has the highest clinical effectiveness known to science, our professional practice environments must be retooled. The purpose of this mastery session is to teach critical care nurses how to traverse the landscape of various levels and strength of evidence when participating in teams to develop or adopt EBP clinical guidelines that seek to improve nursing effectiveness outcomes. Using contemporary clinical questions, a basic research primer will be infused, focusing on advantages and limitations of various types of designs and samples as well as statistical significance and consistency of findings, to jumpstart nurses knowledge and comfort in reading various forms of evidential summaries related to their practice. Concentrating on the first three points of the ACESTAR model and using the Essential Competencies for Evidence-Based Practice in Nursing (Stevens, 2005) as a backdrop, participants will learn the vital skills and steps involved in appraising or developing clinical EBP guidelines: 1) identifying researchable questions (DISCOVERY); 2) searching databases and critically appraising available evidence (SUMMARY), a component that will include an online demonstration of a basic literature search on a contemporary practice question; and 3) summarizing evidence into clinically usable forms and integrating guidelines into practice (TRANSLATION). Examples of how essential components of guideline development can be matched with the skill levels of interdisciplinary team members will be discussed so that effective partnerships and mentoring experiences for clinicians are co-created.

LEARNING OUTCOMES

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

1. Outline the components of a focused and researchable 'PICO' or 'PS' question.
2. Discuss steps in conducting a search to locate various forms of evidence on a chosen topic.
3. Identify key criteria of appraisal checklists to critique the strength of evidence and decide whether it should be incorporated into clinical practice guidelines.

SUMMARY OF KEY POINTS

- I. International Call for Evidence-Based Practice (EBP): The New Paradigm in Health Care

- A. Crossing the Quality Chasm
 1. Bridging 'what we know to be best nursing care' & 'what is actually practiced'
 2. IOM report recommended healthcare redesign
 - a. Safe, timely, efficient
 - b. Effective, patient-centered, equitable
 3. EBP key solution for redesign
 - a. EBP – Conscientious use of best research evidence, in combination with a clinician's expertise & patient's values/preferences, in determining type of care to be delivered
 - b. Essential Competencies for EBP in Nursing (Stevens, 2005)
 - B. Magnet Standards for Nursing Excellence
 1. Importance of research to advance practice & improve patient outcomes
 2. Demonstration that nursing care is based on evidence
- II. ACESTAR Discovery Phase - Searching for Generated Knowledge
- A. Identify researchable clinical problem to promote best practice
 1. Clinical problems that regularly arise in practice
 2. Questions that ask "what is the best way to ___" (or is one therapy more effective than another in producing a desired health outcome)
 - B. Formulating searchable clinical question
 1. PICO format (Quantitative)
 - a. P - Patient population, or problem
 - b. I - Independent variables, or intervention of interest (e.g. preventive, etiologic, diagnostic, prognostic, therapeutic, meaning, managerial, economic)
 - c. C - Comparison (groups of patients or strategies being compared, if applicable)
 - d. O - Outcomes of interest (i.e., patient/family, provider or organizational)
 2. PS format (Qualitative)
 - a. P - Population
 - b. S - Situation
 - C. Establish interdisciplinary team
 1. Key stakeholders to reduce bias & enhance quality
 2. Involvement instills ownership to enhance future use
- III. ACESTAR Summary Phase - Gathering the Evidence
- A. Understanding the Hierarchy of Pre-Processed Evidence
 1. Systems – Clinical practice guidelines (AACN Practice Alerts, Joanna Briggs Institute, National

- Guidelines Clearinghouse), evidence-based textbooks
- 2. Synopses of syntheses – Database of Abstracts of Reviews of Effects (DARE), Evidence-based abstract journals (Evidence-based Nursing)
- 3. Syntheses – Systematic reviews (Cochrane library, Worldviews on Evidence-based Nursing, Agency for Healthcare Research & Quality, health care journals)
- 4. Synopses of single studies – Evidence-based abstract journals (Evidence-based Nursing)
- 5. Single studies – PubMed clinical queries (filtered searches); CINAHL, Medline, PsychInfo; handsearching (only if no systematic review or if outdated)
- B. Critiquing available evidence
 - 1. Critical appraisal of validity & generalizability of relevant retrieved evidence
 - a. Critical appraisal checklists: www.gla.ac.uk/departments/generalpractice/ca_check.htm
 - b. Appraisal of Guidelines for Research & Evaluation: www.agreecollaboration.org
 - 2. Primer on how to critically appraise research or guidelines
 - a. Purpose - Research questions
 - b. Literature review
 - c. Methods – Design, sample, measures, data collection procedures
 - d. Results & Limitations – Qualitative vs. quantitative
 - e. Implications for practice
- C. Assigning levels and strength of evidence (AACN, 1997)
 - 1. Level I - Manufacturer's recommendations only
 - 2. Level II - Theory based (no data); expert consensus recommendations may exist
 - 3. Level III - Laboratory data, no clinical data to support recommendations
 - 4. Level IV - Limited clinical studies to support recommendations
 - 5. Level V - Clinical studies in 1 or 2 populations/situations to support recommendations
 - 6. Level VI - Clinical studies in variety of populations/situations to support recommendations
- IV. ACESTAR Translation Phase - Readying Evidence for Practice
 - A. Creating synthesized summary of evidence
 - 1. Clinical guideline – A systematically developed statement to assist decision-making about appropriate health care for specific clinical circumstance
 - 2. Benefits of clinical guidelines
 - a. Patients/families
 - b. Clinicians
 - c. Organizations
 - d. Health care policy
 - B. Attributes of Quality EBP Guidelines
 - 1. 'Best & latest' research evidence for each health

- outcome of interest ... aka valid, reliable & current
- 2. Clinically applicable ... aka target patient population (with exceptions) explicitly defined
- 3. Clear ... aka logical & unambiguous
- 4. Balanced ... aka potential advantages & disadvantages of interventions specified (extent of confidence that adherence will 'do more good than harm')
- 5. Adaptable ... aka able to change as knowledge advances
- 6. Feasible for setting, including cost-benefit analysis
- 7. Implementable & measurable
- 8. Peer-reviewed - External review for clinician & administrative feedback
- 9. Organizationally endorsed, with plan for scheduled review & revision
- C. Presentation
 - 1. Recommendations explicitly linked to evidence (with accompanying rationale)
 - 2. Format
 - a. Full guideline
 - b. 2 page user-friendly summary sheets (protocols, algorithms)
 - c. Reminder prompts in electronic clinical decision support
- D. Examples
 - 1. AACN Practice Alerts: Family Presence during Resuscitation (recommended)
 - 2. EBP Guidelines: Normal saline instillation with suctioning (not recommended); Trendelenburg position for hypotension (not recommended); music therapy to reduce pain/anxiety after cardiac surgery (recommended)
- V. Getting the Gold Together through Collaboration
 - A. Working in teams
 - 1. Utilizing skills & competencies of team members
 - 2. Collaborating with interdisciplinary colleagues
 - B. Fostering mentoring experiences
 - 1. Staff nurse-Clinical nurse specialist partnerships
 - 2. Professional development – EBP skills to advance nursing care

BIBLIOGRAPHY/WEBLIOGRAPHY

- Duffy M. Resources for critically appraising quantitative research evidence for nursing practice. *Clinic Nurs Specialist* 2005; 19 (5), 233-235.
- Duffy M. Resources for critically appraising qualitative research evidence for nursing practice clinical question, *Clinic Nurs Specialist* 2005; 19 (6), 288-290.
- Fineout-Overholt E, Hofstetter S, Shell L, Johnston L. Teaching EBP: Getting to the gold: How to search for the best evidence. *Worldviews Evidence-based Nursing* 2005; (4thQ): 207-211.
- Fineout-Overholt E, Melnyk B, Schultz A. Transforming health care from the inside out: Advancing evidence-based practice in the 21st century. *J Professional Nursing* 2005; 21 (6): 335-344.

- Flemming K. Asking answerable questions. *Evidence-Based Nursing* 1998; 1 (2): 36-37.
- Graham I, Harrison M, Brouwers M, Davies B, Dunn S. Facilitating the use of evidence in practice: Evaluating and adapting clinical practice guidelines for local use by health care organizations. *JOGNN*, 2002; 31: 599-611.
- Spasser M. Evidence-based nursing resources. *Mel Refer Services Quart* 2005; 24 (2): 71-85.
- Stevens K. *Essential Competencies for Evidence-based Practice in Nursing*. San Antonio TX: The University of Texas Health Science Center, 2005.

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Grants and Grub: Chapters and Vendors Working Together

Tonya Skeen
Jan Teal

Level: Beginner

CONTENT DESCRIPTION

Example: One purpose of this session is to increase the knowledge and understanding of regulations and guidelines that govern the sponsorships of medical device and pharmaceutical companies. Meanwhile creative thinking and discussion will take place in order to optimize ways for local chapters to partner and build relationships with these companies.

LEARNING OUTCOMES

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

1. Identify at least three vendor activities that are regulated by legislation.
2. Name at least two guidelines to be followed when providing continuing education credit to program participants.?
3. Name at least three points to consider when soliciting vendor sponsorship.

SUMMARY OF KEY POINTS

- I. Regulation overview
 - A. Food and Drug Administration Guidance for Industry
 - B. 2007 United States Senate report
 - C. Educational activities
 - D. Research grants
 - E. Medical device versus pharmaceutical industries
- II. Guidelines for Chapters
 - A. Rules and regulations
 - B. Program approval
 1. American Association of Critical-Care Nurses
 2. Other associations
 - C. Educational content and materials

III. Soliciting sponsorship

- A. Documentation of request
- B. Event budget
- C. Knowledge of marketplace, competitors
- D. Skilled communication

IV. Discussion

V. Conclusion]

BIBLIOGRAPHY/WEBLIOGRAPHY

- Chapter Module: Rules and Regulations, American Association of Critical-Care Nurses. Available at: [http://www.aacn.org/aacn/chapters.nsf/Files/Rule&Reg/\\$file/Rule&Reg.pdf](http://www.aacn.org/aacn/chapters.nsf/Files/Rule&Reg/$file/Rule&Reg.pdf). Accessed December 6, 2007.
- Committee Staff Report to the Chairman and Ranking Member, Use of Educational Grants by Pharmaceutical Manufacturers. Available at: <http://www.senate.gov/~finance/press/Bpress/2007press/prb042507a.pdf>. Accessed December 6, 2007.
- Continuing Education Program Approval: Handbook & Application, American Association of Critical-Care Nurses. Available at: [http://www.aacn.org/pdfLibra.NSF/Files/progappr/\\$file/ProgAppr.pdf](http://www.aacn.org/pdfLibra.NSF/Files/progappr/$file/ProgAppr.pdf). Accessed December 6, 2007.
- Decision Making Guide, American Nurses Credentialing Center. Available at: <http://www.nursecredentialing.org/accred/dguide.html>. Accessed December 6, 2007.
- Oncology Nursing Society, ONS Foundation, Oncology Nursing Certification Corporation, and the ONSEdge Commercial Support Position. Available at: <http://www.ons.org/publications/positions/corporateSupport.shtml> Accessed November 11, 2007.
- Guidance for Industry: Industry-Supported Scientific and Educational Activities, U.S. Department of Health and Human Services, Food and Drug Administration, Office of Policy, November, 1997. Available at: <http://www.fda.gov/cvm/Guidance/guide65.pdf>. Accessed December 6, 2007.

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The “HA HA HA’S” of the Sepsis Bundles

Nicholas F. Montanaro

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this session is to examine sepsis, the sepsis bundles, and their role in the management of the septic patient. Complex cellular descriptions characteristic of sepsis will be discussed, along with the evidence based treatments employed in the care of this patient population. The two main bundles will be described including the resuscitation and management bundles.

LEARNING OUTCOMES

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

1. Understand the statistics for sepsis, who is at risk, and the path of sepsis.
2. Understand the pathophysiology behind sepsis and the development of altered organ function as a result of the sepsis cascade.
3. Understand the sepsis bundles responsible for the resuscitation and management of the patient with sepsis.
4. Understand other considerations for patients with sepsis and pediatric considerations.

SUMMARY OF KEY POINTS

- I. Sepsis statistics
 - A. Number of cases each year
 - B. Rate of growth of cases
 - C. Who is at risk?
 - D. Mortality
- II. The path of sepsis
 - A. Uncomplicated sepsis
 - B. Severe sepsis
 - C. Septic shock
- III. The Surviving Sepsis Campaign
 - A. Why it was formed
 - B. Campaign goals
 - C. Formation of the bundles of treatment
- IV. Sepsis pathophysiology
 - A. SIRS

- B. Complications from sepsis
- C. Arachidonic acid cascade
- D. Stages of shock
 1. Initial
 2. Compensatory
 3. Progressive
 4. Refractory
- V. Resuscitation bundles
 - A. Lactate levels
 - B. Hydration
 - C. Antibiotics
 - D. Goals to achieve with resuscitation
- VI. Management bundles
 - A. Hormone replacement
 - B. Airway considerations
 - C. Hyperglycemia management
 - D. Drotrecogin alfa
 - E. Source control
 - F. Other considerations
 - G. Pediatric considerations

BIBLIOGRAPHY/WEBLIOGRAPHY

- Ahrens, T: Sepsis: Stopping an insidious killer. What you must know to understand, identify, and treat this lethal condition. *American Nurse Today* 2007; Volume 2: 36-40.
- Dellinger RP, Carlet JM, Masur H, et al. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med*. 2004; 32 (3): 858-873.
- Hotchkiss RS, Karl IE. The pathophysiology and treatment of sepsis. *N Engl J Med*. 2003; 348: 138-150.
- IHI.org
- Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001; 345 (19): 1368-1377.
- survivingsepsis.org

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Healthy, Safe Work Environments: Creating Effective Teams

Beth LaVelle

Level: Intermediate

CONTENT DESCRIPTION

Excellent teamwork is critical to patient safety and healthy work environments. Critical Care staff are on numerous teams. Some teams are unit-based while others - such as Rapid Response Teams and Code Teams - extend past the familiar walls of our Critical Care Units. Intelligence, dedication, and clinical expertise do not necessarily translate into high-performance team behavior, particularly when the teams are so variable and the work is under stressful conditions. Fortunately, crisis/crew resource management (CRM) and teamwork are skills that can be taught, practiced, and evaluated. This workshop is designed to help clinicians evaluate the current performance of their clinical teams and identify strategies to improve them.

LEARNING OUTCOMES

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

1. Analyze obstacles that get in the way of one of your teams being able to perform effectively.
2. Select concrete strategies to address one of the teamwork obstacles you identified.
3. Identify ways that CRM principles and strategies can be applied to ineffective Critical Care or facility-based committees.

SUMMARY OF KEY POINTS

- I. Critical Care Teams
 - A. Clinical Action/Direct Patient Care Teams
 1. Unit-based
 2. Off-the-unit: Rapid Response Teams, Medical Emergency Teams, Code Teams
 - B. Committees
- II. Performance obstacles/ teamwork challenges
 - A. High stakes: patient safety
 - B. High variability: patients & their locations, team members
 - C. High pressure: sense of urgency, incomplete information, competing tasks & responsibilities, interruptions, fatigue
 - D. The Five Dysfunctions of a Team (Lencioni, 2002)
- III. Principles of teamwork Crisis/crew resource management & related strategies
 - A. Role clarity
 1. Who will do what (& how do you know?)
 - B. Effective communication
 1. Quickly size up the situation
 2. Skillful, tactful questions & clear requests
 3. Good listeners who verify information
 4. Clear, concise, accurate, directed, closed loop
 5. SBAR (situation, background, assessment, recommendation)

6. Psychological safety
 7. Assertion /critical language
- C. Utilization of resources
 1. Support
 2. Patient information
 3. Supplies, equipment
 4. Too much help?
 - D. Situational/global awareness
 1. Perspective
 2. Exercises: visual cognition/change blindness
- IV. Limitations
 - A. Human factors
 - B. "Silence Kills" (VitalSmarts, AACN, Crucial Conversations, 2005)
 - V. Promote high-performing teams
 - A. Culture of collaboration with respect and a sense of purpose
 - B. Attitude adjustments
 - C. Communication skills
 - D. Educational strategies
 1. Reflection: Debriefing, M & M
 2. Simulation (CA Rauen: "You can't just throw a book at them.")
 - E. Supporting behaviors
 - VI. Reflect
 - A. Are you satisfied with the performances of your clinical teams?
 - B. Are your committees effective?
 - C. Personal responsibility + commitment: What can YOU do to help?

BIBLIOGRAPHY/WEBLIOGRAPHY

- Dunn WF, Ed. Simulators in Critical Care and Beyond. SCCM; 2004. See Fiedor M L & DeVita, M Human simulations & Crisis-Team Training and Raemers DR. Team-oriented medical simulation.
- Gurses SP, Carayon P. Performance obstacles of intensive care nurses. *Nursing Research*. 2007; 56(3), 185-194.
- Kurtz S, Silverman J, Draper J. *Teaching and Learning Communication Skills in Medicine* (2nd Ed). Oxford: Radcliffe Publishing; 2005.
- Lencioni, P. *The Five Dysfunctions of a Team: A Leadership Fable*. San Francisco: Jossey-Bass; 2002. Leonard M, Graham S, Bonacum D. The human factor: the critical importance of effective teamwork and communication in providing safe care. *Qual Saf Health Care* 2004; 13 (suppl 1), i85-90. Maxfield D, Grenny J, McMillan R, Patterson K, Switzler A. *Silence Kills. The Seven Critical Conversations for Healthcare*. VitalSmarts, LC; 2005. www.viscog.beckman.uiuc.edu or www.viscog.com

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Healthy Work Environment Return on Investment

Nancy Blake
Mary Bylone

Level: Beginner

CONTENT DESCRIPTION

This session will give an overview of the Healthy Work Environments and discuss return on investment as a means to justify the actual implementation of the standards. The HWE Toolkit will be described, as well as an assessment tool to use in your unit. There will also be a detailed discussion of implementation in an intensive care unit and the actual return on investment. This discussion will include examples of areas to address in each of the standards.

LEARNING OUTCOMES

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

1. List the 6 HWE Standards
2. Describe the HWE Toolkit
3. Describe the steps to go about addressing the return on investment in their work area

SUMMARY OF KEY POINTS

Given the healthcare environments that we work in it is important to do whatever can possibly be done to make the work environment a place in which retention is high and outcomes are the best they can be. Anyone who has read the HWE standards understands that they are important in all of the environments that we work. Poor communication has been linked to major errors and poor patient outcomes. Lack of meaningful recognition and collaboration have been linked to high turnover. These, in turn, link to high costs. If nurses are to make their optimal contributions in caring for patients and families, establishing and sustaining healthy work environments must be a priority in all hospitals and healthcare systems.

It is important that everything be done to make our work environments healing and humane as well. It is also important that the best care be given at the lowest cost given the rising costs of healthcare, with low reimbursement. In the year 2005, the U.S. spent 16% of the Gross National Product on Healthcare. Healthcare

spending is 4.3 times what is spent on national defense. Insurance costs are rising at an alarming rate. In 2006, insurance premiums rose 7.7%, which was two times the rate of inflation.

This session will begin with a brief overview of each of the HWE standards: skilled communication, true collaboration, meaningful recognition, effective decision making, appropriate staffing and authentic leadership. The critical elements for the standards will also be discussed.

The HWE Toolkit will be described and successful implementation will be discussed. A brief overview of building the case for the return on investment will be discussed. This will be followed by a detailed discussion of one unit's successful implementation of the standards which resulted in an annual \$2M savings on the budget. The impact on patient outcomes will also be discussed.

BIBLIOGRAPHY/WEBLIOGRAPHY

- American Association of Critical Care Nurses. (2005) AACN Standards for Establishing and Sustaining Healthy Work Environments: A Journey to Excellence. *AJCC* 14 (3): 187 - 197.
- Heath, J., Johanson, W., Blake, N. (2004) Healthy Work Environments: A validation of the literature. *JONA* 34: 524 - 530.
- Schmalenberg, C and Kramer, M. (2007) Types of Intensive Care Units with Productive Work Environments. *AJCC* 16 (5): 458 - 468.
- Shirey, Maria R. Authentic Leaders Creating Healthy Work Environments for Nursing Practice. *AJCC* 15 (3) 256 - 267.
- Thornby, Denise. (2006) Beginning the Journey to Skilled Communication. *AJCC* 17 (3): 266 - 271.
- www.aacn.org/AACN/hwe.nsf/vwdoc/HWEhomepage
www.nchc.org/facts/cost.shtml
www.vitalsmarts.com/healthcare.aspx

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Healthy Work Environments: No Place for Bullies

Karen Stutzer-Treimel

Level: Intermediate

CONTENT DESCRIPTION

Two characteristics of a healthy work environment are skilled communication and meaningful recognition. Unfortunately workplace incivility and communication that is rude and demeaning persists in our environments, negatively impacting on the quality of working relationships. Retention of qualified staff and patient safety are jeopardized when communication is disrespectful and unprofessional. A variety of explanations including oppression theory, disenfranchising work practices, aggression breeding aggression and poor self esteem have been explored as reasons for the persistence of this unhealthy behavior. Solutions are complex and require organizational commitment to supporting a healthy work environment. Unit cultures supportive of staff and managerial attention to supporting staff are key in the prevention of horizontal violence. Finally, individual courage to support colleagues and confront negative behavior is vital to breaking the cycle of horizontal violence.

LEARNING OUTCOMES

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

1. Identify sources of horizontal violence in the workplace.
2. Identify strategies to prevent the negative impact of horizontal violence on the individual nurse and in the workplace.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Nurse to nurse hostility: The Scope of the Problem
 - B. Terms used in the literature
 1. Bullying
 2. Horizontal violence
 3. Verbal abuse
- II. Historical Overview of Horizontal Violence
 - A. Organizational hierarchy
 - B. Subordinate role of nurse
- III. Theoretical framework
 - A. Oppression theory
 - B. Time task work imperatives
 - C. Intergenerational workforce
 - D. Clique formation
 - E. Low self esteem
- IV. Impact of nurse to nurse hostility
 - A. Personal
 1. Stress related illness

2. Errors
 3. Leaving positions
 4. Withdrawal
 5. Burnout
- B. Patients
 1. Errors
 - C. Organizational
 1. Quality concerns
 2. High turnover
 3. Difficulty recruiting
- V. Solutions
 - A. Organizational
 1. Senior management commitment
 2. Disruptive behaviors polices
 3. Leadership development
 - B. Managerial
 1. Foster postive unit culture
 2. Address disruptive behavior
 3. Ongoing self development
 - C. Personal
 1. Self reflection
 2. Break the silence: Call it what it is
 3. Role model positive behavior
 - VI. Resources
 - A. AACN Healthy Work Environment Standards
 - B. Website: <http://www.aacn.org/AACN/hwe.nsf/vwdoc/HWEHomePage>
 - C. A Promise to Each Other

BIBLIOGRAPHY/WEBLIOGRAPHY

- Baltimore, J.J. (2006). Nurse collegiality: Fact or fiction? *Nursing Management*, May 2006, pp. 26-36.
- Bartholomew, K. (2006). Ending Nurse-to-Nurse Hostility: Why Nurses Eat Their Young and Each Other. HCPro: Marblehead, MA
- Dunn, H. (2003). Horizontal violence among nurses in the operating room. *AORN Journal*, 78(6), 977-988.
- Farrell, G. A. (2001). From tall poppies to squashed weeds: Why don't nurses pull together more? *Journal of Advanced Nursing*, 35(1), 26-33.
- Hutton, S. A. (2006). Workplace incivility: State of the science. *Journal of Nursing Administration (JONA)* 36(1), 22-28.

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Heart Sounds: Adult Cardiac Abnormalities

Barbara A. Erickson
Sponsored by 3M Health Care

Level: Expert

CONTENT DESCRIPTION

The purpose of this interactive session is to provide an opportunity for discussion and interactive learning to the critical care practitioner in differentiating clinical findings and heart sounds for common adult cardiac abnormalities. A basic auscultation course is a prerequisite. Participants are expected to be knowledgeable in the recognition of normal heart sounds and in differentiating S1, S2, S3, S4 and splits. A heart sound simulator and individual stethophones will be used with slides of physical presentation. “Pearls” for arriving at nursing diagnosis and interventions are shared with participants.

LEARNING OUTCOMES

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

1. interventions: normal heart, acute myocardial infarction, and mitral valve prolapse.
2. Differentiate the following cardiac abnormalities in an adult and the clinical implications for nursing diagnosis/ interventions: mitral regurgitation, aortic regurgitation, and aortic stenosis.
3. Differentiate the following cardiac abnormalities in an adult and the clinical implications for diagnosis/ interventions: pulmonic stenosis, subvalvular aortic stenosis (IHSS), and mitral stenosis.

SUMMARY OF KEY POINTS

Case Study #1

James is a 13 year old white male who presents for a cardiac evaluation prior to joining his school’s football team. He has had the usual childhood illnesses. There is no personal or family history of diabetes mellitus, hypertension, rheumatic fever, or heart disease. He has no complaints.

Physical Findings: Normal

Symptoms: None

Auscultatory Findings:

LLSB: Split S1; S1 louder S2

Apex: Single S1; S1 louder S2; S3 & S4

Base R: Single S1 & S2; S2 louder S1

Base L: Single S1; S2 physiological split; S2 louder S1

Nursing Diagnosis: Normal 13-year old

Nursing Interventions: No cardiac interventions.

Psycho-social for teenager.

Case Study #2

M.I. is a 60 year old school teacher who was admitted to the CCU after experiencing severe substernal crushing pain (8/10) which radiated to his left shoulder and elbow while lecturing to his afternoon

class. The pain was associated with weakness, nausea, and copious diaphoresis.

Physical Findings:

Blood pressure normal

Pulse pulsus alternans

JVP elevated

Lungs basilar moist inspiratory rales.

Symptoms (Common): Pain, nausea, diaphoresis, and palpitations.

Auscultatory Findings:

First Sound soft

Paradoxically split second

Systolic murmur (apex)

S4

S3

Pericardial Friction Rub

Nursing Diagnosis—Alterations in:

1. Cardiac output and tissue perfusion due to ischemia
2. Comfort due to pain
3. Fluid and electrolyte imbalance due to anorexia, nausea, decreased fluid intake, or increased retention.
4. Knowledge deficit related to pathology, tests, drugs, and treatments

Nursing Interventions:

1. Pain relief (nitro, narcotics)
2. Provide rest
3. Monitor rhythm, labs, pressures
4. Balance fluid and electrolytes
5. Allay anxiety
6. Administer Medications
7. Educated relating to deficits

Case Study #3

M.P. is a 23 year old college student. She has been experiencing chest pain, not related to exertion, and palpitations. She states that her heart “beats fast”; “starts and stops suddenly”; and that she feels “tired”.

Physical Findings: Normal or Thin, (pectus excavatum, pectus carinatum, scoliosis and/or kyphosis may be present).

Symptoms (Common): Most asymptomatic

Atypical or nonanginal chest pain

Fatigue and dyspnea

Palpitations

TIAs (1/3 of those with TIAs under 45 due to MVP)

Anxiety and lassitude

Auscultatory Findings:

Mid or late systolic clicks

Late Systolic Murmur

Click Murmur moves closer to S1 with maneuvers that reduce ventricular volume. Moves farther from S1 with maneuvers that increase ventricular volume.

Nursing Diagnosis—Alterations In:

1. Cardiac Output
2. Comfort due to pain
3. Coping due to anxiety
4. Knowledge deficit relating to pathology, tests, drugs, and treatments.

Nursing Interventions:

1. Monitor physical response
2. Educate relating to MVP, infection (invasive procedures or dental), medication (Beta Blockers)
3. Provide psychological support.

Case Study #4

M.R. is a 37 year old accountant, who had acute rheumatic fever at 11 years of age. After recovery he had no further difficulty and was active in sports. He was rejected from military service at age 18 because of a murmur. He admits to rare palpitations “heart skips a beat”. He is being referred by his company’s insurance program for evaluation of a cardiac murmur.

Physical Findings:

Carotid pulse small and sharp in upstroke
VP dominant increased “A” wave
Apical Impulse vigorous

Symptoms (common):

Asymptomatic (with slight abnormality)
Fatigue, cough, palpitations (with Mild)
Dyspnea, orthopnea or PND from CHF (more severe)

Auscultatory Findings:

Pansystolic Murmur
[With Moderate Severity]
Wide Split Second
S3

Increased Intensity P2

[Slight Severity]

Late Systolic Murmur (radiates LLSB and Base R)

Nursing Diagnosis—Alterations in:

1. Cardiac output due to Mitral Regurgitation
2. Comfort due to fatigue, cough, dyspnea, especially at night (Paroxysmal Nocturnal Dyspnea [PND])
3. Tissue perfusion; cerebral (syncope)
4. Coping ineffective (anxiety)
5. Knowledge deficit relating to pathology, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Administer medications
3. Provide psychological support
4. Educate relating to pathology of Mitral Regurgitation, tests, drugs, and treatments

6. Preop and postop nursing care as indicated:

Case Study #5

A.S., a 57 year old mechanic, has noted increasing shortness of breath with mild exertion over the past three months. Echocardiographic findings show a bicuspid aortic valve. He denies chest pain or syncope. He is referred for evaluation of a murmur.

Physical Findings:

Narrow Pulse pressure
Arterial Pulses small and slow rising
Pulsus alternans (often)
Forceful & sustained apical impulse
Systolic Thrill (Base R, SCV notch, carotids)

Symptoms (Common):

Dyspnea, orthopnea, PND
Nonproductive cough with CHF
Pressing substernal pain or syncope on exertion

Auscultatory Findings:

Aortic Ejection Sound
[Moderate Severity]
Systolic Ejection Murmur
Normal Split S1
Normal Split S2
[Severe]
M1 closure muffled
Ejection sound absent
A2 (absent, delayed, or paradoxically split)
Systolic Ejection Murmur
S4

Nursing Diagnosis—Alteration In:

1. Cardiac output due to Aortic Stenosis
2. Comfort due to fatigue, cough, dyspnea, especially at night (Paroxysmal Nocturnal Dyspnea [PND])
3. Tissue perfusion; cerebral (syncope)
4. Coping ineffective (anxiety)
5. Knowledge deficit relating to pathology, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Administer medications
3. Provide psychological support
4. Educate relating to pathology of Aortic Stenosis, tests, drugs, and treatments
5. Preop and postop nursing care as indicated:

Case Study #6

P.S., a 22 year old male clerk has had a cardiac murmur that was detected at 7 years of age. He was asymptomatic until one year ago when, after a severe upper respiratory infection, he noted mild dyspnea on exertion. At present, he becomes dyspneic after relatively mild exertion.

Physical Findings:

Arterial pulse normal
BP normal or narrow pulse pressure
Moon facies (severe dysfunction)
Cyanosis (severe dysfunction)

Sustained impulse LLSB
JVP increased "A" wave (severe dysfunction)

Symptoms (common):

Exertional dyspnea

Fatigue

No orthopnea or PND

Auscultatory Findings:

Pulmonic Ejection Sound

Systolic Ejection Murmur

Nursing Diagnosis—Alterations in

1. Cardiac output due to Pulmonic Stenosis
2. Comfort due to fatigue, cough, dyspnea
3. Coping ineffective (anxiety)
4. Knowledge deficit relating to pathology, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Administer medications
3. Provide psychological support
4. Educate relating to pathology of Pulmonic Stenosis, tests, drugs, and treatments
5. Preop and postop nursing care as indicated:

Case Study #7

I.H.S.S., a 64 year old retired bookkeeper, was admitted to CCU because of severe, sharp chest pain of several hours duration. He has been aware that he had a cardiac murmur for at least 20 years, but was in good health until 2 years ago when he began experiencing dull pain in the right side of his chest that was precipitated by exertion and only partially relieved with nitroglycerine. He has become progressively more dyspneic and has had several episodes of dizziness but never fainted.

Physical Findings:

Carotid Pulse, sharp upstroke

Apical impulse forceful and sustained

JVP dominant "A" wave

Symptoms (common):

Asymptomatic or

[Without Obstruction]

Exertional Dyspnea, chest pain, presyncope or syncope and CHF

Frequent Dysrhythmias

[With Obstruction] Dynamic and increase with anything which:

increases myocardial contractility

decreases left ventricular volume

decreases left ventricular afterload

Auscultatory Findings:

Late Systolic Ejection Murmur

S4

Paradoxical Split Second

S3

Nursing Diagnosis—Alterations in:

1. Cardiac output due to IHSS
2. Comfort due to fatigue, dyspnea,
3. Coping ineffective (anxiety)

4. Impaired mobility related to activity intolerance

5. Knowledge deficit relating to pathology, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Educate relating to pathology of IHSS, tests, drugs, and treatments
3. Administer medications (Avoid Digitalis)
4. Provide psychological support
5. With syncope elevate legs
6. Preop and postop nursing care as indicated

Case Study #8

M.S., a 61 year old housewife, was referred because of a CVA that cleared without residual. She had an episode of rheumatic fever at age 8; on recovery she had no limitations. She had three children without difficulty. At age 59, she was told that she had a cardiac murmur. Three months ago, she began to experience episodes of palpitations and a decrease in exercise tolerance. Two weeks ago she experienced right-sided weakness that cleared within 2 days. Her ECG shows atrial fibrillation.

Physical Findings:

Mitral Facies

JVP abnormal "A"

Right ventricular hypertrophy

Bilateral inspiratory moist rales

Wheezes (from assoc. bronchitis)

Symptoms (common):

Dyspnea on exertion

Atrial Fibrillation

Orthopnea and PND (severe abnormality)

Hemoptysis (15%)

Embolization

Fatigue (d/t decreased CO)

Auscultatory Findings:

Pre systolic Murmur

Loud S1

Opening Snap

Mid Diastolic Rumble

Loud P2 (with pulmonary hypertension)

Nursing Diagnosis—Alteration In:

1. Cardiac output due to Mitral Stenosis
2. Tissue Perfusion (symptoms due to emboli from Atrial Fibrillation)
3. Comfort due to fatigue, cough, dyspnea, especially at night (Paroxysmal Nocturnal Dyspnea [PND])
4. Coping ineffective (anxiety)
5. Impaired mobility related to activity intolerance
6. Knowledge deficit relating to pathology of Mitral Stenosis, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Administer medications
3. Provide psychological support
4. Educate relating to pathology of Mitral Stenosis, tests, drugs, and treatments

5. Preop and postop nursing care as indicated:

Case Study #9

A.R., a 49 year old laborer, had an attack of rheumatic fever at 9 years of age and again at 13 years of age. A cardiac murmur was detected during the second attack. He was not permitted to participate in physical education in high school and was rejected from military service because of the murmur.

He was asymptomatic until 36 years of age when he noticed mild exertional dyspnea. During the past three months, he has noticed increasingly more severe dyspnea, fatigue and weakness

Physical Findings:

Wide Pulse Pressure

Water Hammer Pulse (Rapid rising & Collapsing)

Corrigan's Sign (Carotid pulse visible)

Quincke's Sign (Pulsating Nail Bed)

DeMusset's Sign (Systolic Head Nodding)

Symptoms (common):

Rare, unless lesion severe

Exertional Dyspnea

Dyspnea at Rest

Angina

CHF

Auscultatory Findings:

Aortic Ejection Sound (may not be present)

Systolic Ejection Sound (may not be present)

Early Diastolic Murmur

Nursing Diagnosis—Alteration In:

1. Cardiac output due to Aortic Regurgitation
2. Comfort due to dyspnea, angina, CHF
3. Impaired mobility related to activity intolerance
4. Coping ineffective (anxiety)
5. Knowledge deficit relating to pathology of Aortic Regurgitation, tests, drugs, and treatments.

Nursing Interventions:

1. Provide balance of rest and activity
2. Administer medications
3. Provide psychological support
4. Educate relating to pathology of Aortic Regurgitation, tests, drugs, and treatments
5. Preop and postop nursing care as indicated

BIBLIOGRAPHY/WEBLIOGRAPHY

Erickson BA. Heart Sounds and Murmurs Across the Lifespan. 4th ed. St. Louis, MO: CV Mosby Company, 2003

Erickson, B.A. Identifying complete heart block in elderly patients. *American Nurse Today*. (Nov. 2006), 1(2), 16-17.

Erickson BA. Heart Sounds and Murmurs: A Practical Guide. 3rd ed. St. Louis, MO: CV Mosby Company, 1997.

Erickson BA. Detecting Abnormal Heart Sounds. *Nurs* 86. January 1986:58-63.

Harvey WP, Canfield DC: Clinical auscultation of the cardiovascular system: tapes with companion tests, Newton, NJ: Laënnec Publishing, 1989.

The Auscultation Assistant. <http://www.wilkes.med.ucla.edu/inex.htm>

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Heart Sounds: Geriatric Auscultation

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Sponsored by 3M Health Care

Level: Expert

CONTENT DESCRIPTION

The purpose of this interactive session is to provide an opportunity for discussion and interactive learning to the critical care practitioner in differentiating clinical findings and heart sounds for geriatric patients. A basic auscultation course is a prerequisite. Participants are expected to be knowledgeable in the recognition of normal heart sounds and in differentiating S1, S2, S3, S4 and splits. A heart sound simulator and individual stethophones will be used with slides of physical presentation. “Pearls” for arriving at nursing diagnosis and interventions are shared with participants.

LEARNING OUTCOMES

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

1. Differentiate by their heart sounds the following cardiac variations of heart sounds in the elderly and their clinical implications: Heart rate, S1 and S2 splits, S3 and S4.
2. Differentiate by their heart sounds the following cardiac variations of heart sounds in the elderly and their clinical implications: mitral regurgitation, tricuspid regurgitation, aortic stenosis and pulmonic stenosis.
3. Differentiate by their heart sounds the following cardiac variations of heart sounds in the elderly and their clinical implications: mitral stenosis, tricuspid stenosis, aortic regurgitation and pulmonic regurgitation

SUMMARY OF KEY POINTS

- I. Review
 - A. When to use bell and diaphragm of stethoscope
 1. Bell chest piece
 - a. Apply lightly so there is not after imprint
 - b. Low frequency sounds are best heard
 2. Diaphragm chest piece
 - a. Apply firmly leaving after imprint
 - b. High frequency sounds are best heard
 - B. Requirements of Auscultation
 1. Quiet, well lit, warm room
 2. Disrobed patient (scope placed directly on skin)
 3. Examine in 3 positions (sitting, lying on back, turned to left lateral recumbent)
 4. Examine from right (stretches tubing of scope and decreases extraneous sounds)
 5. Use “Bell” and “Diaphragm” (differentiate low from high frequencies)
 6. Selective Listening
 - C. Sequence of auscultation: Listen to all valve areas using first the “diaphragm” and then the “bell”
 1. Left Lateral Sternal Border (LLSB):

- a. Fourth intercostal space to left of sternum
- b. Tricuspid and right heart sounds
2. Apex:
 - a. Fifth intercostal space in mid-clavicular line
 - b. Mitral and left ventricular sounds
3. Base Left:
 - a. Second intercostal space to left of sternum
 - b. Pulmonic sounds
4. Base Right:
 - a. Second intercostal space to right of sternum
 - b. Aortic sounds
5. Erb’s Point: (Listen here if a murmur is heard.)
 - a. Third intercostal space to left of sternum
 - b. Murmurs of aortic and pulmonic origin
- D. Normal S1, S2, and splits
 1. First Sound Productions: Due to closure of mitral (M1) and tricuspid (T1) valves.
 - a. M1:
 - (1) First audible component of first sound
 - (2) Occurs just after mitral valve closes
 - (3) Occurs 0.02 to 0.03 seconds after left ventricular pressure equals left atrial pressure.
 - (4) Higher intensity and frequency than T1
 - (5) Best heard with diaphragm
 - (6) Discernible over much of precordium but especially at Apex
 - b. T1:
 - (1) Second audible component of first sound
 - (2) Occurs just after tricuspid valve closes
 - (3) Best heard at LLSB
 - (4) High frequency sound
 - (5) Best heard with diaphragm
 - c. Split of first Sound:
 - (1) Both components of sound are audible
 - (2) Heard best at LLSB
 - (3) Sounds are 0.02 seconds apart (just within the ear’s ability to hear)
 - (4) Split is normal (common in children; only heard in about 50 percent adults)
 2. Second Sound Production: Due to closure of Aortic (A2) and Pulmonic (P2) valves; left-sided mechanical events precede right.
 - a. A2 :
 - (1) First audible component of a normal second sound
 - (2) Usually more energy behind its production
 - (3) Audible at all auscultatory sites
 - (4) High frequency sound which is best heard with a diaphragm

- (5) Best heard at Base Right where aortic components radiate
- b. P2:
 - (1) Second audible component of a normal second sound
 - (2) Softer of the two components
 - (3) Audible at Base Left where pulmonic components radiate
 - (4) High frequency sound which is best heard with diaphragm
- 3. Splits: Both components making sound are audible
 - a. Split First: mitral and tricuspid sounds audible and best heard LLSB
 - b. Split Second: aortic and pulmonic sounds audible and best heard Base Right with split occurring on Inspiration and single sound on Expiration
- E. Normal sounds at various auscultatory sites
- II. Variations of heart sounds in the elderly
 - A. Heart rate
 - 1. Rates of 40 beats/minute may be normal
 - 2. Result of decreased beta-adrenergic responsiveness
 - 3. Differentiate from low rates from abnormal conduction
 - B. S1 split
 - 1. More marked in elderly
 - 2. May confuse with ejection sound or S4
 - C. S2 split
 - 1. May not be audible
 - 2. Due to prolonged left ventricular ejection and isovolumic contraction times.
 - D. S4
 - 1. May be heard in healthy elderly (not heard in younger except athletes)
 - 2. May also be sign of heart disease
 - 3. After age 75, many have S4 at apex and usually have
 - a. Left ventricular hypertrophy (LVH)
 - b. Impaired diastolic relaxation (on echo)
 - 4. The auscultatory findings of a S4 are:
 - a. Low frequency sound in front of the first sound
 - b. Best heard at apex
 - c. Best heard with bell lightly held.
 - E. S3
 - 1. Over age 40 suggests ventricular failure or volume overloading
 - 2. Suggests valvular heart disease
 - 3. In elderly should not be considered physiological but abnormal
 - 4. The auscultatory findings of pathological S3 are:
 - a. Low frequency sound after the second sound
 - b. Best heard at apex
 - c. Best heard with bell lightly held
- III. Systolic murmurs in the elderly
 - A. Aortic Sclerosis
 - 1. Most common condition of elderly with systolic murmur
 - 2. 50% of 85 year olds have
 - 3. Result of degenerative fibrous thickening and stiffening of base aortic cusps
 - a. No commissural fusion
 - b. No impairment of cusp mobility
 - c. No obstruction
 - 4. Not hemodynamically significant
 - 5. The common auscultatory findings of aortic valve sclerosis are:
 - a. Systolic ejection murmur
 - (1) Murmur peaks early
 - (2) Harsh is quality
 - (3) Heard equally well with either a bell or diaphragm chestpiece
 - (4) Heard best at base right
 - (5) Rarely radiates to the carotid arteries.
 - (6) May be considered an “innocent” murmur
 - B. Aortic stenosis
 - 1. Increases with age
 - 2. Manifestation of calcific degeneration
 - a. Congenitally abnormal aortic valve--six decade
 - b. Previously normal aortic valve--seventh decade.
 - c. Narrow pulse pressure may be absent in the elderly
 - d. Carotid upstroke may not be slow in the elderly because vascular compliance is diminished.
 - 3. The auscultatory findings of aortic valve stenosis with moderate stenosis are:
 - a. Systolic ejection murmur
 - (1) Murmur less intense in elderly
 - (2) Harsh quality murmur heard
 - (3) Heard equally well with either bell or diaphragm
 - (4) Best heard at base right
 - (5) Radiates down to apex and bilaterally into neck
 - b. Aortic ejection sound
 - (1) Usually not audible in elderly
 - (2) If present, high frequency
 - (3) Heard best with diaphragm
 - (4) Heard best at base right and on straight line down to apex
 - c. Normal split S1
 - (1) Heard at 4th ICS left sternal border
 - (2) Heard with diaphragm
 - (3) May be increased and confused with ejection sound or S4
 - d. Physiologically Split S2 (splits on inspiration; single on expiration)
 - (1) May not be audible in elderly
 - (2) Heard at base right
 - (3) Heard best with diaphragm
 - (4) The auscultatory findings of aortic valve stenosis with severe stenosis are:
 - a. Systolic ejection murmur
 - (1) May be less than grade II/VI in intensity

- (2) Harsh quality murmur heard equally well with either bell or diaphragm
 - (3) Heard best base right
 - (4) Radiates down to apex and bilaterally into neck
 - b. M1 closure muffled (Split S1 not heard at LLSB)
 - c. Aortic ejection sound absent
 - d. S4
 - (1) Low frequency sound in front of S1
 - (2) Heard best apex
 - (3) Heard with bell lightly held
- C. Pulmonic stenosis
1. Uncommon in elderly. Etiology most commonly congenital lesion (VSD or Tetralogy of Fallot) or rarely carcinoid heart disease.
 2. A narrow pulse pressure is found in this pathology.
 3. The auscultatory findings of pulmonic stenosis are:
 - a. Pulmonic ejection sound (usually absent in elderly)
 - (1) High frequency sound after S1
 - (2) Best heard at base left and on a straight line from base left to the epigastric area.
 - b. Systolic ejection murmur
 - (1) Harsh quality
 - (2) Medium frequency
 - (3) Heard equally well with either bell or diaphragm chestpiece
 - (4) Best heard at base left
 - (5) Radiates along upper left sternal border
- D. Mitral regurgitation
1. Congenitally abnormal aortic valve--seventh decade
 2. Previously normal aortic valve--eighth decade
 3. Degenerative changes with calcification of mitral annulus or valve ring
 - a. Hinder normal contraction of ring during systole
 - b. Produces mitral regurgitation
 4. The auscultatory findings of mitral regurgitation are:
 - a. Systolic murmur
 - (1) Late systolic (slight regurgitation)
 - (2) Holosystolic (severe regurgitation)
 - (3) Blowing quality
 - (4) Best heard with diaphragm
 - (5) Best heard apex
 - (6) Radiates to left axilla and left sternal border; may radiate to base left.
 - b. Wide split second
 - (1) May not be audible in elderly
 - (2) If audible, heard at base right
 - (3) Heard with diaphragm
 - c. S3
 - (1) Low frequency sound after S2
- (2) Heard best apex
- (3) Heard with bell lightly held
- E. Tricuspid regurgitation
1. Pure tricuspid regurgitation uncommon in elderly
 2. May be component of pulmonary hypertension
 - a. Right ventricle dilates which stretches tricuspid annulus
 - b. Valve becomes incompetent
 3. The auscultatory findings of tricuspid regurgitation are:
 - a. Systolic murmur
 - (1) Late systolic (slight regurgitation)
 - (2) Holosystolic (severe regurgitation)
 - (3) Blowing quality
 - (4) Best heard with diaphragm
 - (5) Best heard apex
 - (6) Radiates to left axilla and left sternal border
 - b. Wide split second
 - (1) May not be audible in elderly
 - (2) If audible, heard at base right
 - (3) Heard with diaphragm
 - c. S3
 - (1) Low frequency sound after S2
 - (2) Heard best apex
 - (3) Heard with bell lightly held
- IV. Diastolic murmurs in the elderly
- A. Mitral stenosis
1. Usually of rheumatic origin
 2. May not become acutely symptomatic except if atrial fibrillation acutely develops
 3. The auscultatory findings of mitral stenosis are:
 - a. Louder than normal first sound
 - (1) Best heard LLSB
 - (2) Best heard with diaphragm
 - b. Opening snap after second sound
 - (1) Heard at apex
 - (2) Best heard with diaphragm
 - c. Mid Diastolic murmur
 - (1) Rumble in quality
 - (2) Best heard with bell lightly held
 - (3) Best heard at apex
 - (4) Radiates to left axilla
- B. Tricuspid stenosis
1. Rare in elderly
 2. Often seen in multivalvular rheumatic disease
 3. Isolated tricuspid stenosis will manifest signs of right heart failure
 4. The auscultatory findings of pure tricuspid stenosis are:
 - a. Louder than normal first sound
 - (1) Best heard LLSB
 - (2) Best heard with diaphragm
 - b. Opening snap after second sound
 - (1) Heard at LLSB
 - (2) Best heard with diaphragm

- c. Mid Diastolic murmur
 - (1) Rumble in quality
 - (2) Best heard with bell lightly held
 - (3) Best heard at LLSB
 - (4) Radiates along left sternal border
 - (5) Inspiration increases intensity of murmur
- C. Aortic regurgitation
 - 1. Less common in the elderly than is aortic stenosis.
 - 2. It may be due to valvular degeneration, congenital bicuspid aortic valve, rheumatic heart disease, infective endocarditis or systemic arterial hypertension
 - 3. The auscultatory findings of aortic regurgitation are:
 - a. Early diastolic murmur
 - (1) Blow in quality
 - (2) Best heard at Erb's point
 - (3) Best heard with diaphragm
 - (4) Radiates down left sternal border
 - (5) Having patient sit up and lean forward in deep exhalation or lean back on elbows brings out this soft murmur
 - b. Aortic ejection sound after S1
 - (1) Usually not audible in elderly
 - (2) If present, high frequency
 - (3) Heard best with diaphragm
 - (4) Heard best at base right and on straight line down to apex
 - D. Pulmonic regurgitation
 - 1. Usually result of pulmonary hypertension
 - a. Due to left-sided-heart failure or
 - b. primary pulmonary disease
 - 2. The auscultatory findings of pulmonic regurgitation are:
 - a. Early diastolic murmur
 - (1) Rough in quality
 - (2) Best heard at base left
 - (3) Best heard with bell

- 4. Radiates upper left sternal border
 - b. Pulmonic ejection sound (may not be present)
 - (1) High frequency sound after S1
 - (2) Heard best base left and on straight line down to epigastrium

- V. Things to remember about Heart Sounds in the Elderly
 - A. Heart Sounds vary from those of younger adults
 - 1. Sounds may be similar
 - 2. Interpretation may be different
 - B. What is abnormal in younger adult may be normal in elderly
 - C. Before reaching a decision, consider:
 - 1. Age
 - 2. If sound is a normal variant for the age
 - 3. Presence of Signs and/or Symptoms
 - 4. Other Findings

BIBLIOGRAPHY/WEBLIOGRAPHY

- Beers MH, Berkow R Editors. The Merck manual of geriatrics. 3rd ed. New Jersey: Merck Research Laboratories; 2000, 863-879.
- Erickson BA. Heart Sounds and Murmurs across the Lifespan. 4th ed. St. Louis, MO: CV Mosby Company; 2003, 110-118.
- Erickson, B.A. Identifying complete heart block in elderly patients. *American Nurse Today*. (Nov. 2006), 1(2), 16-17.
- Harvey WP, Canfield DC: Clinical auscultation of the cardiovascular system: tapes with companion tests, Newton, NJ: Laënnec Publishing, 1989.
- Tallis R, Fillit H, Brocklehurst JC. Brocklehurst's textbook of geriatric medicine and gerontology. 5th Ed. New York: Churchill Livingstone; 1998, 337-343.
- The Auscultation Assistant. <http://www.wilkes.med.ucla.edu/inex.htm>

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Heart Sounds: Murmurs, Clicks, and Rubs

Barbara A. Erickson

Sponsored by 3M Health Care

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this interactive session is to provide the critical-care practitioner an opportunity for interactive learning and discussion on differentiating and explaining abnormal heart sounds and murmurs and their implications for patient care through the use of a heart sound simulator and individual stethophones. A basic recognition of the normal heart sounds--S1, S2, S3, and S4 is essential.

LEARNING OUTCOMES

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

1. Differentiate the physiologic basis for the ejection sound, opening snap, mid systolic click, S4 and S3 and explain their clinical implications for nursing diagnosis/interventions.
2. Differentiate the physiologic basis for systolic murmurs and explain their clinical implication for nursing diagnosis/interventions.
3. Differentiate the physiologic basis for diastolic murmurs and rubs and explain their clinical implications for nursing diagnosis/interventions

SUMMARY OF KEY POINTS

I. Review Normal Heart Sounds

Case Presentation:

Alice is a 14 year old white female who presents for a cardiac evaluation prior to joining her school's soccer team. She has had the usual childhood illnesses. There is no personal or family history of diabetes mellitus, hypertension, rheumatic fever, or heart disease. She has no complaints.

Physical Findings: Normal

Symptoms: None

Auscultatory Findings:

LLSB: Split S1; S1 louder S2

Apex: Single S1; S1 louder S2

Base R: Single S1 & S2; S2 louder S1

Base L: Single S1; S2 physiological split;

S2 louder S1

Nursing Diagnosis: Normal 14-year old

Nursing Interventions: No cardiac interventions.

Psycho-social for teenager.

II. Sounds Around the First Sound

A. Ejection Sounds

1. High pitched "clicking" sounds occurring very shortly after the first sound
2. Usually heard best at base right or base left with diaphragm
3. Of aortic or pulmonic origin and produced when blood is ejected from right or left ventricle

B. Aortic ejection sounds:

1. Heard anywhere on straight line from base right to apex
2. Sound not affected by respiration
3. Heard in:
 - a. Valvular aortic stenosis
 - b. Aortic insufficiency
 - c. Coarctation of the aorta
 - d. Aneurysm of the ascending aorta

C. Pulmonic ejection sounds:

1. Heard anywhere on straight line from base left to epigastrium
2. May increase with expiration and decrease with inspiration in patient with pulmonary valve stenosis
3. Heard in:
 - a. Pulmonary valve stenosis
 - b. Pulmonary embolism
 - c. Pulmonary hypertension
 - d. Hyperthyroidism.
 - e. Conditions causing enlargement of pulmonary artery

III. Midsystolic Clicks

A. High pitched sound(s) that may be isolated or multiple

B. Frequently heard in mitral valve prolapse:

1. Ballooning of one of the mitral valve leaflets into left atrium
2. Usually posterior leaflet
3. At the point of maximal ventricular ejection

C. Click heard when the chordae tendinae, which may be longer than normal, suddenly stop the ballooning leaflet.

D. Occur as least 0.14 seconds after the first sound

E. Usually heard best at apex or LLSB with diaphragm

F. Click moves closer to S1 with maneuvers which REDUCE ventricular volume (standing, valsalva, tachycardia, amyl nitrate)

G. Click moves farther from S1 with maneuvers which INCREASE ventricular volume (squatting, bradycardia, propranolol, pressers)

IV. S4

A. Low frequency sound heard just before the first heart sound

B. Heard best with the bell

C. Result of decreased ventricular compliance or increased volume of filling

D. May be sign of ventricular stress

E. Heard in:

1. Primary myocardial disease

2. Coronary artery disease
 3. Hypertension
 4. Aortic and pulmonic stenosis (severe)
 5. Delayed A V conduction
 6. Heart failure: (more likely to be heard in Diastolic dysfunction when ventricles cannot fill properly; less likely to be heard in Systolic dysfunction when heart unable to contract effectively)
 7. Some normal hearts
- V. Sounds Around the Second Sound
- A. Opening Snap
 1. Short, high frequency snap that occurs after S2
 2. Result of audible opening of the mitral valve due to stiffening (i.e., MS) or increased flow (i.e., VSD or PDA)
 3. Best heard with diaphragm
 4. Loudest between the apex and LLSB
- VI. S3
- A. Low frequency sound heard just after S2
 - B. Heard best with the bell
 - C. Result of decreased ventricular compliance
 - D. May be sign of ventricular distress
 - E. Heard in :
 1. Youth
 2. Coronary artery disease
 3. Heart failure: (first clinical sign: more likely to be heard in Systolic Dysfunction when heart unable to contract effectively; less likely to be heard in Diastolic Dysfunction when ventricles cannot fill properly)
 4. Cardiomyopathies
 5. Incompetent valves (mitral tricuspid, aortic)
 6. Left to right shunts (VSD or PDA)
- VII. Differentiating S3 from Opening Snap
- A. Opening snap
 1. occurs earlier after S2
 2. high frequency sound
 3. best heard with diaphragm
 - B. S3
 1. occurs later than the opening snap
 2. low frequency sound
 3. heard best with bell lightly held
- VIII. Murmurs
- A. Definition sustained noises that are audible during the time periods of systole, diastole, or both
 - B. Production Factors
 1. Backward regurgitation through leaking valve, septal defect, or arterio venous connection (i.e., patent ductus)
 2. Forward flow through narrowed/deformed valve
 3. High rate of blood flow through normal or abnormal valves
 4. Vibration of loose structures within the heart (i.e., chordae tendinae)
 5. Continuous flow through A V shunts
 - C. Characteristics to be noted
 1. Location of the valve area where heard best
 2. Loudness (intensity). Use 1-6-grading system
 3. Frequency (pitch) i.e., low, medium, high
 4. Quality i.e., blowing, harsh, rough, rumble
 5. Timing systolic or diastolic
 6. Finer timing i.e., early, mid or late
 7. Radiation
 8. Changes with respiration, positions, or drugs
- D. Classification (grading) of sounds and murmurs
1. Not audible for first few seconds
 2. Heard at once but faint
 3. Loud no thrust or thrill
 4. Loud with thrust or thrill
 5. Loud with thrust or thrill and audible with chest piece partially applied to chest
 6. Loud with thrust or thrill and audible with chest piece just off chest
- E. Differentiation by frequency, quality and abnormality: Frequency Quality Abnormality(ies)
1. High Blowing Regurgitations (mitral, tricuspid, aortic) and VSD
 2. Medium Harsh Stenosis (Aortic and pulmonic): Rough Pulmonic Regurgitation, ASD, Increased pulmonary outflow
 3. Low Rumble Mitral Stenosis
- F. Special Maneuvers
1. Help differentiate one murmur from another:
 - a. Valsalva:
 - (1) Patient is asked to bear down or strain.
 - (2) Increases intrathoracic pressure and decreases venous return right heart
 - (3) Most murmurs decrease during strain phase of the Valsalva
 - (4) After release of strain, right-sided events return in one to three beats; left-sided events return in four to 11 beats.
 - b. Exercise
 - (1) Increases heart rate which increases blood flow
 - (2) Murmurs may be increased in intensity
 - c. Expiration
 - (1) Changes loudness of sounds by bringing heart closer to chest wall
 - (2) Heart sounds are louder
 - (3) Faint diastolic murmurs of aortic or pulmonic regurgitation may be heard better during expiration
 - d. Hand grip
 - (1) One hand is gripped the other
 - (2) Causes increase in peripheral vascular resistance, blood pressure, heart rate and cardiac output
 - (3) Increases left-sided regurgitant murmurs (aortic and mitral regurgitation and VSD)
 - (4) Decreases left-sided ejection murmurs (aortic stenosis)
 - e. Amyl Nitrate

- (1) Causes decrease in peripheral vascular resistance
- (2) Increase in heart rate and venous return
- (3) Following inhalation of Amyl Nitrate
 - (a) Forward flow murmurs Increase (aortic stenosis, mitral stenosis, pulmonic stenosis, tricuspid stenosis)
 - (b) Left-sided regurgitant murmurs Decrease

IX. Systolic Murmurs

- A. Definition sustained noises that are audible during the time period of systole
- B. Early systolic murmur
 - 1. Begins with S1 and peaks in the first third of systole
 - 2. Modified regurgitant murmur with backward flow through an incompetent valve, septal defect or A V communication
 - 3. Common causes are small VSD or innocent murmur of childhood
 - a. Innocent systolic murmur
 - (1) Usually early systolic in type, grade 2/6 or less
 - (2) Not due to recognizable lesions of the heart
 - (3) Common in children
- C. Midsystolic murmur
 - 1. Begins shortly after S1 and peaks in mid systole and does not quite extend to the second sound
 - 2. Forward flow through a narrow or irregular valve
 - 3. Crescendo decrescendo murmur (“diamond shaped”) which builds up and decreases symmetrically
 - 4. Common causes are aortic stenosis and pulmonic stenosis
- D. Late systolic murmur
 - 1. Begins in latter one half of systole; peaks in later third of systole; and extends to the second sound
 - 2. Modified regurgitant murmur with backward flow through incompetent valve
 - 3. Common causes are papillary muscle disorders, mitral valve prolapse, and idiopathic hypertrophic sub aortic stenosis
- E. Pansystolic (Holosystolic) murmur
 - 1. Continuous throughout systole
 - 2. Begins with S1 and ends with S2 on the side it originates (i.e., left side A2; right side P2)
 - 3. Since pressure remains higher throughout systole in ejecting chamber than receiving chamber, the murmur is continuous throughout systole.
 - 4. Common causes are mitral regurgitation, tricuspid regurgitation, and VSD

X. Diastolic Murmurs

- A. Definition sustained noises that are audible during the time period of diastole
 - 1. Always organic
 - 2. Never innocent in nature
- B. Three main mechanisms causing
 - 1. Semi lunar valve incompetence

- 2. A V valve stenosis
- 3. Increase blood flow across A V valves
- C. Early diastolic murmur
 - 1. Begins with S2 and peaks in first third of diastole
 - 2. Usually regurgitant murmur with backward flow through incompetent valve
 - 3. Common causes are aortic or pulmonic regurgitation
- D. Mid diastolic murmur
 - 1. Onset delayed after second heart sound
 - 2. Peaks in mid diastole
 - 3. Usually low pitched and rumbling
 - 4. Common causes are mitral or tricuspid stenosis

XI. Pericardial Friction Rub

- A. Etiology
 - 1. Infective pericarditis (i.e., viral, pyogenic, TBc, etc.)
 - 2. Noninfective pericarditis (i.e., myocardial infarction, uremia, neoplasm, myxedema, trauma)
 - 3. Autoimmune (i.e., rheumatic fever, drugs, etc.)
- B. Pathology sign of pericardial inflammation
- C. Auscultatory signs
 - 1. One systolic sound that occurs anywhere in systole
 - 2. Two diastolic sounds at time ventricles are stretched in diastole
 - a. In early diastole near the end of the early filling wave (when S3 would occur)
 - b. At end diastole, when atrial contraction produces sudden ventricular expansion (when S4 would occur)
 - 3. Most patients have a tachycardia
- D. Sound characteristics
 - 1. Scratching, grating, or squeaking, to and fro leathery
 - 2. High pitched; best heard with diaphragm
 - 3. Louder during inspiration
 - 4. Likely to be inconsistent and transitory may disappear and reappear
 - 5. Heard most frequently during forced expiration, with patient leaning forward or with patient on hands and knees
 - 6. Heard anywhere on precordium but often loudest at LLSB

XII. Pleural Friction Rub

- A. Etiology pneumonia, viral infections, tuberculosis, pulmonary embolism
- B. Pathology sign of pleural inflammation; rubbing of visceral and parietal surfaces of pleura
- C. Auscultatory signs
 - 1. Course grating or creaking sound heard during inspiration (usually always heard) on expiration (sometimes absent)
 - 2. Common site is lower anterolateral chest wall (area of greatest thoracic mobility)
 - 3. Superficial character with definite proximity to surface
 - 4. Decreases with decreased respirations; gone when breath held

BIBLIOGRAPHY/WEBLIOGRAPHY

- Erickson BA. Heart Sounds and Murmurs Across the Lifespan. 4th ed. St. Louis, MO: CV Mosby Company, 2003.
- Erickson BA. Heart Sounds and Murmurs: A Practical Guide. 3rd ed. St. Louis, MO: CV Mosby Company, 1997.
- Erickson BA. Detecting Abnormal Heart Sounds. Nurs 86. January 1986:58-63.
- Harvey WP, Canfield DC: Clinical auscultation of the cardiovascular system: tapes with companion tests, Newton, NJ: Laënnec Publishing, 1989.
- The Auscultation Assistant. <http://www.wilkes.med.ucla.edu/inex.htm>

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Heart Sounds: Pediatric Auscultation

Barbara A. Erickson
Sponsored by 3M Health Care

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this interactive session is to provide the critical-care practitioner an opportunity for interactive learning and discussion on identifying and differentiating normal heart sounds from abnormal heart sounds and murmurs and their implications for the care of the pediatric patient. Heart sounds are replicated through the use of a heart sound simulator and stethophones. Basic recognition of normal heart sounds (S1, S2, S3, S4 and splits) are prerequisite knowledge for the session.

LEARNING OUTCOMES

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

1. Differentiate systolic murmurs that are innocent from those of a small ventricular septal defect or Tetralogy of Fallot.
2. Differentiate by their heart sounds the following abnormalities with a diastolic murmur: aortic regurgitation, pulmonic regurgitation, mitral stenosis, and tricuspid stenosis.
3. Differentiate by their heart sounds the following abnormalities with a continuous murmur: atrial septal defect, patent ductus arteriosus, cervical venous hum, and large ventricular septal defect

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Auscultation is core of pediatric cardiovascular evaluation
 - B. Meeting challenges of pediatric auscultation
- II. Pediatric anatomy and physiology of heart sounds.
 - A. Areas to auscultate
 1. LLSB
 2. Apex
 3. Base Right
 4. Base Left
 5. Back
 - B. PMI
 1. At level of 4th intercostal space (ICS) until age 7; after age 7 heard at 5th ICS
 2. Left of mid-clavicular line until age 4
 3. At mid-clavicular line between ages 4-6
 4. Moves to the right of mid-clavicular line at age 7
 - C. Heart Rate in beats /minute
 1. Fetal 120-160 (140 average)
 2. Newborn 70-170 (120 average)
 3. 1-12 months 80-160 (120 average)
 4. 1-7 years 75-130 (100 average)
 5. 8 to 12 years 70-110 (90 average)

6. 18 years or older 50-95 (75 average)
- D. S4
 1. Normal only in trained athletic child
 2. Usually not normal finding in infant or child
 3. Right ventricular origin
 - a. Birth to age 6, heard at 4th ICS at LLSB
 - b. Age 7 or older, heard at 5th ICS at LLSB
 - c. Common pathologies: Primary Pulmonary hypertension; Pulmonic stenosis; Epstein's anomaly; Tricuspid atresia; Total anomalous pulmonary venous return or complete heart block
 4. Left ventricular origin
 - a. Heard at age appropriate PMI
 - b. Common pathologies: Severe left ventricular disease; Aortic stenosis; Coarctation of the aorta
- E. Physiologic split second (splits on inspiration and is single on expiration)
 1. Not heard in newborns until after high resistance in pulmonary circulation ,which is normal in fetal life, falls.
 - a. Falls dramatically at birth
 - b. Reaches normal levels at 6 to 8 weeks old
 2. Best heard at base left with diaphragm firmly pressed
 3. Diagnostically very important
- F. Opening snap
 1. Uncommon in children
 2. High frequency sound after S2
 3. Best heard with diaphragm firmly pressed
 4. Heard at apex (mitral stenosis) or LLSB (tricuspid stenosis)
- G. S3
 1. Normal in children and young adults
 2. Males lose by late 20s
 3. Females lose by 30s
 4. Pathological S3 usually palpable; physiologic S3 not palpable
 5. Low frequency sound after S2
 6. Heard best with bell lightly held over PMI as appropriate for age
- H. Quadruple Rhythm
 1. Heard when both S3 and S4 are present
 2. Low frequency diastolic sounds
 3. Heard best with bell lightly held over PMI as appropriate for age
- III. Systolic Murmurs
 - A. Characteristics of Innocent murmurs
 1. Continuous sound that is caused by a normal

- degree of turbulence as blood moves from one area to another.
 - 2. Ejection in type
 - 3. Grade 1/6 to 2/6
 - 4. Physiologic split S2 is ALWAYS present
 - 5. 90% of children may have
 - 6. Common physical findings:
 - a. Precordial activity is normal
 - b. S1 is normal
 - c. S2 physiologic splits
 - d. Systolic murmur decreases on standing
 - B. Murmurs considered innocent:
 - 1. Still's murmur
 - a. Most common
 - b. From vibrations of pulmonic leaflets at their attachments
 - c. Auscultatory Findings:
 - (1) Early or midsystolic murmur:
 - (a) Grade 1/6 to 2/6
 - (b) Medium frequency with groaning, twanging quality
 - (c) Equally well heard with either bell or diaphragm
 - (d) Best heard over right ventricle between LLSB and apex
 - (e) Radiates toward base right
 - (f) Decreases on standing or Valsalva maneuver
 - (2) Physiologic split S2
 - d. Differential diagnosis: aortic stenosis, hypertrophic obstructive cardiomyopathy (HOCM) or small VSD.
 - 2. Innocent Pulmonary Flow murmur
 - a. From turbulence due to changes in systolic pressure from the pulmonary trunk and branches.
 - b. 15% of all innocent murmurs, especially newborn or premature infants
 - c. Disappears within few months as infant grows
 - d. Auscultatory Findings:
 - (1) Midsystolic murmur:
 - (a) Grade 1/6 to 2/6
 - (b) Medium pitched
 - (c) Heard equally well with either a bell or diaphragm
 - (d) Maximal at base left
 - (e) Does not radiate to back or neck
 - (f) Increased in high output states such as anemia or thyrotoxicosis
 - (2) No ejection sound
 - (3) Physiologic split S2
 - e. Differential diagnoses: Atrial septal defect (ASD), pulmonic stenosis, tricuspid regurgitation, and hypertrophy obstructive cardiomyopathy (HOCM)
 - 3. Cervical venous hum
 - a. Hum produced by turbulence in the blood flow in the internal jugular vein as blood flows from head and neck into thorax.
 - b. Second most common innocent murmur of toddlers to school age children
 - c. Auscultatory findings:
 - (1) Continuous sound which is heard during systole and accentuated during diastole
 - (a) Low in frequency and soft
 - (b) Best heard with bell lightly held
 - (c) Heard above clavicles on both right and left; more common on right when child is sitting
 - (d) May radiate slightly down sternum; such radiation rare
 - (2) Loudest on sitting or standing
 - (3) Hum obliterated by turning head; light pressure with finger over jugular vein; lying supine; or Valsalva maneuver
 - (4) Hum increased in conditions with increased blood flow (e.g. thyrotoxicosis or anemia)
 - d. Differential diagnosis: Patent ductus arteriosus (PDA) or AV fistula; sounds remain audible by above mentioned maneuvers.
4. Mammary Soufflé
 - a. Innocent murmur that may be heard in latter months of pregnancy or during lactation caused by increased blood flow in the internal mammary and intercostal arteries.
 - b. Auscultatory findings:
 - (1) Systolic ejection murmur with a diastolic component (latter may be present)
 - (a) High pitched
 - (b) Best heard with diaphragm
 - (c) Best heard over or just above the breasts in 2nd right or left ICS in MCL; with little radiation
 - (d) More obvious when lying flat; decreases with upright posture or pressure with stethoscope over the area.
 - c. Obliteration of this murmur with pressure permits differentiation from that of patent ductus arteriosus (PDA).
- C. Tetralogy of Fallot
 - 1. Most common congenital lesion which produces cyanosis.
 - 2. Condition has various degrees of pulmonary valvular stenosis or infundibular stenosis (most common); ventricular septal defect (VSD) with right-to-left shunt; aorta that overrides the VSD; and right ventricular hypertrophy.
 - 3. Auscultatory findings of pulmonary valvular stenosis or infundibular stenosis of Tetralogy of Fallot:
 - a. Systolic murmur; ejection or pansystolic
 - (1) Mix of high and low frequencies
 - (2) Rough quality
 - (3) Equally heard with bell or diaphragm
 - (4) Loudest in 3rd or 4th left ICS

- (5) Inverse relationship between loudness of murmur and severity of condition; loud with:
 - (a) moderate stenosis; soft to absent with very severe stenosis
 - b. P2 component of S2 very soft; S2 perceived as single; physiologic split S2 not heard.
 - c. Aortic ejection sound may be present
 - D. Blalock-Taussig operation done to correct Tetralogy of Fallot.
 - 1. This is a palliative operation:
 - a. Typically a Gore-Tex shunt is connected to the subclavian artery and right or left pulmonary artery depending in patient's anatomy.
 - b. Shunt may be removed when complete repair is done at about 3-6 months.
 - 2. Auscultatory findings of Blalock-Taussig shunt:
 - a. Causes a continuous murmur over the clavicle on the affected side or over operative scar.
 - b. Murmur heard only in systole with:
 - (1) High pulmonary vascular resistance
 - (2) Small shunt
 - c. Listen for presence of this murmur to ensure patency of shunt
 - (1) Diastolic component is lost commonly with shunt narrowing (common finding)
 - (2) Loss of diastolic component also can occur with increase pulmonary vascular resistance (in Large Shunt)
 - E. Ventricular Septal Defect (small)
 - 1. Hole in ventricular septum in most common pathology with systolic murmur in children
 - 2. Direction of blood flow through defect dependent on degree of pulmonary vascular outflow obstruction.
 - a. When low, flow is left to right
 - b. When high, flow is right to left.
 - 3. Auscultatory findings:
 - a. Murmur of VSD dependent upon its size, position, and loudness.
 - b. Systolic ejection murmur
 - (1) Small defect: high frequency; mildly harsh; best heard with diaphragm
 - (2) Loud murmur: high frequency; harsher; heard with either a bell or diaphragm
 - (3) Best heard in 3rd or 4th ICS to left of sternum
 - (4) Radiation dependent on intensity or loudness
 - (a) Very loud – hear over entire precordium and on back
 - (b) Usually not heard in neck
 - c. Physiologic split S2 may be heard
 - d. Ejection sound absent
 - e. Degree of pulmonary hypertension:
 - (1) Slight: left to right shunt becomes smaller; murmur softer
 - (2) More severe: left to right shunt decreased; murmur may disappear
 - (3) Very severe: shunt changes right to left; cyanosis is manifested; Eisenmenger's syndrome is manifested.
- #### IV. Diastolic Murmurs
- A. Aortic regurgitation
 - 1. Etiology: bicuspid aortic valve common; other congenital defects of valve; acute rheumatic fever; or association with high VSD.
 - 2. When congenital, murmur soft at birth; increases as child grows
 - 3. Auscultatory findings:
 - a. Softer than normal S1
 - b. Aortic ejection sound
 - c. Early diastolic murmur
 - (1) Blow in quality
 - (2) Best heard with diaphragm firmly pressed at Erb's point
 - (3) Radiates down the left sternal border or to Apex. May radiate to back
 - (4) Bring out murmur by having patient sit up and lean forward or lean back on elbows
 - B. Pulmonic regurgitation
 - 1. Etiology: surgical repair of Tetralogy of Fallot; congenital defect of pulmonic valve; pulmonary hypertension; or infective endocarditis.
 - 2. Auscultatory findings:
 - a. Softer than normal S1
 - b. Pulmonic ejection sound (Heard congenital and not rheumatic cause)
 - c. Early diastolic murmur
 - (1) With pulmonary hypertension: starts just after S2 and decrescendo extends to S1; medium pitched; heard equally well with either bell or diaphragm; best heard at base left and down upper left sternal border; may radiate to back between 4th – 5th thoracic vertebrae to left and right of spine.
 - (2) Without pulmonary hypertension: starts slightly after S2 and has short decrescendo; lower in pitch; best heard with bell lightly held; best heard at base left and down upper left sternal border; may not radiate to back.
 - C. Mitral stenosis
 - 1. Etiology: Acute rheumatic fever occurring in children between ages of 5 and 14 years.
 - 2. Auscultatory findings:
 - a. Louder than normal first sound
 - b. Opening snap (uncommon in children)
 - c. Mid diastolic murmur with presystolic accentuation: rumble in quality; best heard with bell lightly held; heard at apex with radiation to left axilla.
 - D. Tricuspid stenosis
 - 1. Etiology: Acute rheumatic fever and, therefore, generally associated with mitral stenosis.
 - 2. Auscultatory findings:
 - a. Louder than normal first sound

- b. Opening snap (uncommon in children)
 - c. Mid diastolic murmur with presystolic accentuation: rumble in quality;
 - d. Best heard with bell lightly held; best heard at LLSB with radiation along left sternal border.
- V. Continuous Murmurs
- A. See Blalock-Taussig operation above which may have a continuous murmur.
 - B. Atrial septal defect characteristics
 1. Congenital defect having abnormal opening between the two atria.
 2. Comprise 10 to 15% of all congenital defects
 3. More common in females
 4. Three types: Ostium primum; Ostium secundum; and sinus venosus.
 - a. Ostium Primum ASD:
 - (1) Opening low in the atrial septum, straddling atrio-ventricular valves.
 - (2) Auscultatory findings:
 - (a) Systolic ejection murmur: grade 2/6; rumble in quality; best heard with bell lightly held; heard at upper 2nd to 3rd ICS left sternal border; may radiate to back at 4th – 5th thoracic vertebrae to left and right of spine.
 - (b) Loud T1 component of first sound
 - (c) P2 component of second sound: delayed with wide fixed splitting of S2;
 - (d) High frequency; best heard with diaphragm at base left.
 - (e) Late systolic or pansystolic murmur if mitral regurgitation occurs: blow in quality; best heard diaphragm at apex with radiation to left axilla and left sternal border; may radiate to back at 4th to 5th thoracic vertebrae to left and right of spine.
 - (f) Mid diastolic murmur with large shunt: rumble in quality; best heard with bell lightly held along lower left sternal border.
 - (g) All auscultatory features present when patient stands.
 - b. Ostium Secundum ASD:
 - (1) Defect at the fossa ovalis caused by failure of development of septum secundum.
 - (2) Auscultatory findings:
 - (a) Systolic ejection murmur: grade 2/6; rumble in quality; heard best with bell lightly held at upper 2nd to 3rd ICS at left sternal border
 - (b) Short mid diastolic murmur: rumble in quality; heard best with bell lightly held at lower left sternal border
 - (c) T1 component of S1 loud
 - (d) P2 delayed and loud with wide fixed split of S2
 - (e) All auscultatory features present when patient stands
 - c. Sinus Venosus ASD:
 - (1) Defect posterior to fossa ovalis and occurs high in atrial septum due to maldevelopment of part of the sinus venosus.
 - (2) Auscultatory findings:
 - (a) Systolic ejection murmur: grade 2/6; rumble in quality; best heard with bell lightly held at upper 2nd to 3rd ICS left sternal border.
 - (b) Diastolic flow murmur from increased flow through normal tricuspid valve grade 2/6; rumble in quality; best heard with bell light held along lower left sternal border.
 - (c) Wide split S2
 - (d) All auscultatory features present when patient stands
- C. Patent ductus arteriosus
 1. Functional closure of ductus arteriosus fails to occur at birth
 2. Auscultatory hallmark is continuous “to and fro” or “machinery-like” murmur with both systolic and diastolic components: harsh in quality; equally well heard with either bell or diaphragm at middle to upper left sternal border; radiates widely to left infraclavicular area, neck, and along the left sternal border.
 3. May be absent in prematures or neonates with high pulmonary resistance
 4. Continuous murmur more pronounced after exercise
 5. Differential diagnosis include: VSD, peripheral pulmonary stenosis; and high output states.
- D. Ventricular Septal Defect (Large)
 1. Defect most often in membranous (upper portion) of ventricular septum.
 2. Defect may not be detected until 6 weeks of age when the high pulmonary vascular resistance found at birth falls and sufficient blood flows through the defect to cause a murmur.
 3. Auscultatory findings:
 - a. Pansystolic murmur: harsh in quality; heard equally well with either bell or diaphragm at 3rd to 4th ICS to left of sternum; loud murmurs may radiate over entire precordium and on back; usually not heard in neck.
 - b. Physiologic split S2 may be absent
 - c. Mid-diastolic flow murmur: rumble in quality; best heard with bell lightly held at apex
 - d. S3 in CHF present
- VI. When to Refer to a Cardiologist
- A. Patients with suspected cardiac pathology should be referred.
 - B. Patients with signs of congenital heart disease: poor feeding, failure to thrive, unexplained respiratory symptoms or cyanosis.
 - C. Unstable or acutely ill children or those with heart failure

D. Patients with innocent murmurs will not have above signs and will be asymptomatic. No need to refer.

BIBLIOGRAPHY/WEBLIOGRAPHY

Behrman RE, Kliegman RM, Jenson HB: Nelson Textbook of Pediatrics. 16th ed. Philadelphia, W.B. Saunders Company, 2000.

Erickson BA. Heart Sounds and Murmurs across the Lifespan. 4th ed. St. Louis, MO: CV Mosby Company, 2003.

Erickson BA. Heart Sounds and Murmurs: A Practical Guide. 3rd ed. St. Louis, MO: CV Mosby Company, 1997.

Erickson BA. Detecting Abnormal Heart Sounds. Nurs 86. January 1986:58-63.

Rush Children's Heart Center: Innocent heart murmurs <http://www.rchc.rush.edu/rmawebfiles/htmurmurs.htm>

The Auscultation Assistant. <http://www.wilkes.med.ucla.edu/inex.htm>

Wong DL et al: Nursing care of infants and children. 6th ed. St Louis, MO: CV Mosby Company, 1998.

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Heart Sounds: The Basics

Barbara A. Erickson
Sponsored by 3M Health Care

Level: Beginner

CONTENT DESCRIPTION

The purpose of this interactive session is to provide the critical-care practitioner an opportunity for interactive learning and discussion on identifying and differentiating normal heart sounds from common abnormal heart sounds. Heart sounds are replicated through the use of a heart sound simulator and stethophones. No prerequisite knowledge is required for the participants.

LEARNING OUTCOMES

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

1. Use basic auscultatory principles in listening to heart sounds with an understanding of the underlying cardiac area and physiology.
2. Differentiate first sound, second sound, splits of first and second sound and S4 and S3 and their clinical implications.
3. Differentiate systolic murmurs, diastolic murmurs, and pericardial friction rubs and their clinical implications

SUMMARY OF KEY POINTS

- I. Introductions
- II. Understanding the Stethoscope
 - A. Types of Scopes
 - B. Diaphragm (high frequency instrument when used with firm pressure)
 - C. Bell (low frequency instrument when used with light pressure)
 - D. Tunable Diaphragm (permits assessment of both high and low frequencies depending on pressure applied)
- III. Requirements of Auscultation
 - A. Quiet, well lit, warm room
 - B. Disrobed patient (scope placed directly on skin)
 - C. Examine in 3 positions (sitting, lying on back, turned to left lateral recumbent)
 - D. Examine from right (stretches tubing of scope and decreases extraneous sounds)
 - E. Use “Bell” and “Diaphragm” (differentiate low from high frequencies)
 - F. Selective Listening
- IV. Sequence of auscultation: Listen to all valve areas using first the “diaphragm” and then the “bell”
 - A. Left Lateral Sternal Border (LLSB):
 1. Fourth intercostal space to left of sternum
 2. Tricuspid and right heart sounds
 - B. Apex:
 1. Fifth intercostal space in mid-clavicular line
 2. Mitral and left ventricular sounds
 - C. Base Left:
 1. Second intercostal space to left of sternum
 2. Pulmonic sounds
 - D. Base Right:
 1. Second intercostal space to right of sternum
 2. Aortic sounds
 - E. Erb’s Point: (Listen here if a murmur is heard.)
 1. Third intercostal space to left of sternum
 2. Murmurs of aortic and pulmonic origin
- V. Characteristics of Sound:
 - A. Frequency: number of wave cycles generated per second by vibrating body.
 - B. Intensity: height of sound wave.
 1. High amplitude—loud sounds
 2. Low amplitude—soft sounds.
 - C. Quality: distinguishes two sounds with equal frequency and intensity but from a different source. (i.e. heart from lung)
 - D. Duration: length of time of sound
- VI. First Sound Productions: Due to closure of mitral (M1) and tricuspid (T1) valves.
 - A. M1:
 1. First audible component of first sound
 2. Occurs just after mitral valve closes
 3. Occurs 0.02 to 0.03 seconds after left ventricular pressure equals left atrial pressure.
 4. Higher intensity and frequency than T1
 5. Best heard with diaphragm
 6. Discernible over much of precordium but especially at Apex
 - B. T1:
 1. Second audible component of first sound
 2. Occurs just after tricuspid valve closes
 3. Best heard at LLSB
 4. High frequency sound
 5. Best heard with diaphragm
 - C. Split of first Sound:
 1. Both components of sound are audible
 2. Heard best at LLSB
 3. Sounds are 0.02 seconds apart (just within the ear’s ability to hear)
 4. Split is normal (common in children; only heard in about 50 percent adults)
 - D. Auscultatory Site and Intensity of First Sound :
 1. Apex: First sound louder than second sound
 2. LLSB: First sound louder than second sound
 3. Base Right: First sound softer than second sound
 4. Base Left First sound softer than second sound
 - E. Ears perception of Intensity:

1. When a loud sound is followed by a soft sound, the ear may not discern the soft sound.
 2. Loud sound followed by soft sound, perceived as coming DOWN a musical scale
 3. Soft sound followed by Loud sound, perceived as going UP a musical scale.
- F. Physiological factors affecting Intensity of First Sound:
1. Chest wall shape and thickness
 2. Rate of rise of ventricular pressure
 3. Position of mitral valve at onset of ventricular systole
 4. Stiffness of the A-V valves
- G. Using first sound to differentiate AV blocks
1. Normal Sinus Rhythms
 - a. 1st sound normal intensity with normal PR interval
 - b. Rhythm regular
 2. First Degree AV Block
 - a. 1st sound softer intensity with prolonged PR interval
 - b. Rhythm regular
 3. Second Degree AV Block
 - a. Mobitz I:
 - (1) Progressive softening of 1st sound as PR interval progressively prolongs before “dropped beat”
 - (2) Pause in rhythm
 - b. Mobitz II:
 - (1) 1st sound has same intensity with stable PR interval before “dropped beat”
 - (2) Pause in rhythm
 4. Third Degree AV Block
 - a. Varying intensity of 1st sound. No relationship of PR interval to QRS
 - b. Slow but regular rhythm
- VII. Second Sound Production: Due to closure of Aortic (A2) and Pulmonic (P2) valves; left-sided mechanical events precede right.
- A. A2 :
1. First audible component of a normal second sound
 2. Usually more energy behind its production
 3. Audible at all auscultatory sites
 4. High frequency sound which is best heard with a diaphragm
 5. Best heard at Base Right where aortic components radiate
- B. P2:
1. Second audible component of a normal second sound
 2. Softer of the two components
 3. Audible at Base Left where pulmonic components radiate
 4. High frequency sound which is best heard with diaphragm
- VIII. Physiologic Splitting of Second Sound:
- A. Both components of sound are audible
1. 0.03 seconds apart
 2. Normally aortic valve closes then pulmonic
 3. Audibility of separate closure is known as “physiological splitting”
- B. A2 Component: loudest and discernible all precordial areas
- C. P2 Component: softer; heard best at Base Left
- D. Normal split second sound is heard on INSPIRATION; Single second sound heard on EXPIRATION
- IX. Relationship of Physiologic Split Second to Inspiration:
- A. On Inspiration the following occurs:
1. Decrease in intrathoracic pressure
 2. Increase in venous return to right atrium
 3. Increased venous return to right ventricle
- B. The increased blood in right ventricle prolongs right ventricular systole; this delays pulmonic valve closure; thus making audible split
- X. Paradoxical Split of Second Sound:
- A. Aortic closure delayed:
1. Reversal of normal closure sequence
 2. Pulmonic valve closes before aortic valve
 3. On Inspiration a single second sound is heard (pulmonic closure occurs late and “fuses” with aortic closure)
 4. On Expiration a Split second sound is heard
- B. Occurs In:
1. Marked volume or pressure loads of left ventricle
 - a. Aortic stenosis
 - b. Aortic regurgitation
 - c. Large patent ductus arteriosus (PDA)
 2. Conduction defects with delayed left ventricular depolarization:
 - a. Left Bundle Branch Block (LBBB)
 - b. Some types of WPW pre-excitation
- XI. Differentiating First Sound from Second Sound:
- A. Subconscious Recognition
1. If Heart rate below 80 beats per minute.
 2. The first sound follows a pause because mechanical systole (the time between the first and second sounds) is shorter than diastole.
- B. Carotid Palpation:
1. While listening to heart sounds, gently palpate carotid.
 2. Sound heard when carotid pulsation is felt is the first hear sound.
 3. Peripheral pulses can NOT be used due to the time lag that occurs between sound and pulse
- C. Listen at Base:
1. Second sound is always loudest at base.
 2. Go to base, identify loudest sound
 3. “Inch” back down to LLSB
- D. Precordial Pulsation:
1. Listen and look at point of maximum impulse

- (PMI) or Apex
 - 2. First sound simultaneous with PMI
- XII. Fourth Heart Sound – S4:
- A. Nomenclature: Atrial gallop; Presystolic gallop; S4 gallop; or S4
 - B. Physiology:
 - 1. Low frequency sound heard just before S1. Heard best with “Bell”
 - 2. Result of decreased ventricular compliance or increased volume of filling.
 - 3. Sign of ventricular stress
 - C. Heard in primary myocardial disease; coronary artery disease; hypertension (systemic and pulmonary); aortic and pulmonic stenosis (severe); delayed A-V conduction; and normal hearts (some)
 - D. If origin of S4 is Left ventricular:
 - 1. Best heard apex in supine or left lateral recumbent position
 - 2. Best heard in expiration
 - 3. Causes include:
 - a. Severe hypertension
 - b. Aortic Stenosis
 - c. Cardiomyopathies
 - d. Myocardial infarction (left heart)
 - e. Heart failure: (more likely to be heard in Diastolic dysfunction when ventricles cannot fill properly; less likely to be heard in Systolic dysfunction when heart unable to contract effectively)
 - E. If origin of S4 is Right ventricular:
 - 1. Best heard LLSB
 - 2. Accentuated with inspiration
 - 3. Causes may include:
 - a. Myocardial infarction (right heart)
 - b. Pulmonic valve obstruction
- XIII. Third Heart Sound—S3:
- A. Nomenclature: Ventricular gallop; Protodiastolic gallop; S3 gallop; S3
 - B. Physiology:
 - 1. Low frequency sound heard just after S2
 - 2. Heard best with “Bell”
 - C. Result of:
 - 1. Decreased Ventricular Compliance
 - 2. Increased Ventricular Diastolic volume
 - D. Sign of Ventricular Stress
 - E. Heard in:
 - 1. youth (normal)
 - 2. coronary artery disease
 - 3. heart failure: (first clinical sign: more likely to be heard in Systolic Dysfunction when heart unable to contract effectively; less likely to be heard in Diastolic Dysfunction when ventricles cannot fill properly)
 - 4. cardiomyopathies
 - 5. incompetent valves (mitral, tricuspid, aortic)
 - 6. left to right shunt (ventricular septal defects [VSD] or patent ductus arteriosus [PDA])
 - F. If origin of S3 is Left ventricular (most common):
 - 1. Heart best at Apex
 - 2. In supine or left lateral recumbent position
 - 3. Decreases on inspiration; increases on expiration
 - 4. Consider left ventricular failure
 - G. If origin of S3 is Right ventricular:
 - 1. Heard at LLSB or xiphoid
 - 2. Consider right ventricular failure
 - H. If S3 heard both at Apex and LLSB consider biventricular failure
 - I. S3 with Emphysema:
 - 1. All heart sounds dampened
 - 2. Heard best at xiphoid or under the rib cage
 - 3. May not be audible
 - J. Gallops and Other Considerations:
 - 1. S3 and S4 are softer on sitting or standing
 - 2. S3 and S4 are louder on exertion (coughing, rolling to left lateral recumbent)
 - 3. Gallops can be Seen, Felt, and Heard
 - 4. Gallops are found only when Searched For
- XIV. Murmurs:
- A. Definition: Murmurs are sustained noises that are audible during the time periods of systole, diastole or both.
 - B. Classification of Sounds and Murmurs: (Top number represents sound heard; bottom number represents total parts of classification system used)
 - 1. Not audible first few seconds; must “tune-in” (1/6)
 - 2. Heard at once but faint (2/6)
 - 3. Loud without thrust or thrill (3/6)
 - 4. Loud with thrust or thrill (4/6)
 - a. “thrust” when palpating precordium sensation taps hand and falls away
 - b. “thrill” when palpating precordium sensation same as placing hand on a purring cat.
 - 5. Loud with thrust or thrill and with chest piece partially applied (5/6)
 - 6. Loud with thrust or thrill and with chest piece just off chest (6/6)
- XV. Systolic Murmur: Sustained noise audible during the systolic time period
- A. Early:
 - 1. Begins with first sound and peaks in first third of systole
 - 2. Common causes are:
 - a. Small ventricular septal defect (VSD)
 - b. Innocent murmur of childhood
 - B. Mid:
 - 1. Begins shortly after the first sound and peaks in mid systole; does not extend to second sound
 - 2. Common causes are:
 - a. Aortic stenosis
 - b. Pulmonic stenosis
 - C. Late:
 - 1. Begins in latter one half of systole; peaks in later third of systole; and extends to the second sound.

2. Common causes are:
 - a. Papillary muscle disorders
 - b. Mitral valve prolapse
 - c. Idiopathic Hypertrophic Sub-aortic Stenosis (IHSS)

D. Pansystolic (Holosystolic):

1. Continuous throughout systole
2. Common causes are:
 - a. Mitral regurgitation
 - b. Tricuspid regurgitation
 - c. Ventricular Septal Defect (VSD)

XVI. Diastolic Murmurs: Sustained noises that are audible during the time period of diastole. Never Normal!

A. Early

1. Begins with second sound and peaks in first third of diastole
2. Common causes are:
 - a. Aortic regurgitation
 - b. Pulmonic regurgitation

B. Mid

1. Begins after S2 and peaks in middiastole (Both S2 and S1 are heard clearly)
2. Common causes are:
 - a. Mitral stenosis
 - b. Tricuspid stenosis

C. Late: (Also known as Presystolic)

1. Begins in latter one half of diastole; peaks in later third of diastole; extends to S1
2. Common component of:
 - a. Mitral stenosis
 - b. Tricuspid stenosis

D. Pandyastolic (Holodiastolic):

1. Begins with S2 and extends throughout diastole
2. Common cause is Patent Ductus Arteriosus (PDA)

XVII. Pericardial Friction Rub

A. Pathology: sign of pericardial inflammation

B. Auscultatory Signs

1. One systolic sound—occurs anywhere in systole
2. Two diastolic sounds—at time ventricles are stretched in diastole

C. Characteristics:

1. Scratching, grating or squeaking quality
2. High frequency which is best heard with diaphragm
3. Transitory

XVIII. Pleural Friction Rub

A. Pathology: sign of pleural inflammation; rubbing of visceral and parietal surfaces of pleura

B. Etiology:

1. Pneumonia
2. Viral infection
3. Tuberculosis
4. Pulmonary embolism

C. Auscultatory Signs:

1. One inspiratory sound
2. One expiratory sound

D. Characteristics:

1. Course grating or creaking sound
2. Common site is lower anterolateral chest wall (area of greatest thoracic mobility)
3. Superficial character with definite proximity to surface
4. Decreases with decreased respirations
5. Gone when breath is held?

BIBLIOGRAPHY/WEBLIOGRAPHY

- Erickson BA. Heart Sounds and Murmurs Across the Lifespan. 4th ed. St. Louis, MO: CV Mosby Company, 2003.
- Erickson, B.A. Identifying complete heart block in elderly patients. *American Nurse Today*. (Nov. 2006), 1(2), 16-17.
- Erickson BA. Heart Sounds and Murmurs: A Practical Guide. 3rd ed. St. Louis, MO: CV Mosby Company, 1997.
- Erickson BA. Detecting Abnormal Heart Sounds. *Nurs* 86. January 1986:58-63.
- Harvey WP, Canfield DC: Clinical auscultation of the cardiovascular system: tapes with companion tests, Newton, NJ: Laënnec Publishing, 1989.
- The Auscultation Assistant. <http://www.wilkes.med.ucla.edu/inex.htm>

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Heart Transplantation: Pearls and Pitfalls

Terri Allison Donaldson
Beth Towery Davidson

Level: Intermediate

CONTENT DESCRIPTION

Care of heart transplant recipients can be complex due to the immunosuppressive regimen required and complications that can occur. Optimal patient outcomes require a collaborative, multidisciplinary approach to ensure that the health care needs of the heart transplant recipient are met. Nurses involved in caring for patients who have undergone heart transplantation must have knowledge regarding rejection, immunosuppression and complications in order to recognize normal findings and implement actions, when appropriate, to manage adverse events.

LEARNING OUTCOMES

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

1. Identify strategies for short and long term care of the heart transplant recipient.
2. Discuss indications, complications and medication interactions related to the immunosuppressive regimen.
3. Identify complications and adverse events that may be experienced by the heart transplant recipient.

SUMMARY OF KEY POINTS

- I. Transplant Procedures
 - A. Donor allocation
 1. UNOS
 2. Matching criteria
 - B. Organ retrieval
 1. Ischemic time
 2. Organ preservation
 - C. Recipient surgical procedure
 - D. Post-operative nursing implications
 1. Heart rate
 2. Hemodynamic monitoring
 3. Inotropic support
 4. Patient education
 5. Discharge planning
- II. Short and Long Term Post-Transplant Care
 - A. Assessment for rejection
 1. Endomyocardial biopsy
 2. Echocardiogram
 - B. Infection control measures
 1. Handwashing
 2. Self-protection
 3. Vaccination
 4. Prophylactic medications

- C. Laboratory analysis
 1. Electrolytes
 2. Renal function
 3. Liver function tests
 4. Lipids
 5. Complete Blood Count
 6. Therapeutic drug levels
- III. Immunosuppression
 - A. Maintenance therapy
 1. Corticosteroids
 - a. Methylprednisolone
 - b. Prednisone
 2. Calcineurin inhibitors
 - a. Cyclosporine
 - b. Tacrolimus
 3. Antiproliferative agents
 - a. Azathioprine
 - b. Mycophenolate mofetil
 - c. Mycophenolate sodium
 4. MTOR inhibitors
 - a. Sirolimus
 - b. Everolimus
 - B. Rescue Therapy
 1. Corticosteroids
 - a. High dose intravenous methylprednisolone
 - b. Oral prednisone bump and taper
 2. Polyclonal antibodies
 - a. Antithymocyte globulin
 3. Monoclonal antibodies
 - a. Muromonab-CD3 (OKT3)
 - C. Induction Therapy
 1. Polyclonal antibodies
 2. Monoclonal antibodies
 - a. Muromonab-CD3
 - b. Basiliximab
 - c. Daclizumab
 - D. Medication interactions
 1. Grapefruit
 2. HMG CoA reductase inhibitors
 3. Calcium channel blockers
 4. Antifungal agents
 5. Macrolide antibiotics
 6. Allopurinol
 7. Phenytoin

IV. Complications and Adverse Events

- A. Rejection
- B. Infections
- C. Cardiac allograft vasculopathy
- D. Malignancy
 - 1. Post-transplant lymphoproliferative disease

V. Management of Co-Morbid Conditions

- A. Hypertension
- B. Hyperlipidemia
- C. Diabetes mellitus
- D. Renal dysfunction
- E. Rheumatology/orthopedics
 - 1. Gout
 - 2. Osteoporosis
 - 3. Avascular necrosis

BIBLIOGRAPHY/WEBLIOGRAPHY

- Baumgartner WA, Kasper E, Reitz B, Theodore J, eds. Heart and Lung Transplantation. 2nd ed. Philadelphia, PA: Saunders; 2002.
- Cupples SA, Ohler, L, eds. Transplantation Nursing Secrets. Philadelphia, PA: Hanley & Belfus; 2003.
- Kirklin JK, Young JB, McGiffin DC. Heart Transplantation. New York, NY: Churchill Livingstone; 2002.
- Lancaster LE. Physiology of inflammation and immunity. In: Lancaster, LE, ed. Core Curriculum for Nephrology Nursing. 4th ed. Pitman, NJ: American Nephrology Nurses' Association; 2001: 33-55.
- Ohler L, Cupples SA, eds. Core Curriculum for Transplant Nurses. St. Louis, MO: Mosby; 2008.
- Taylor AL, Watson CJE, Bradley JA. Immunosuppressive agents in solid organ transplantation: Mechanisms of action and therapeutic efficacy. *Critical Reviews in Oncology/Hematology*. 2005;56: 23-46.
- United Network for Organ Sharing. Policy 3.7: Allocation of Thoracic Organs. Available at: <http://www.unos.org/qa.asp#matchFactors>. Accessed October 6, 2007.

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The Heart-Kidney Connection: Complexities of Cardiorenal Syndrome

Peggy L. Kirkwood

Level: Expert

CONTENT DESCRIPTION

The purpose of this session is to prepare acute and critical care staff and advance practice nurses to care for acutely decompensated heart failure (HF) patients with renal insufficiency. Acute decompensated HF is the most common reason for hospital admission among patients over age 65. The majority of these patients are admitted with fluid overload. A worsening of their clinical picture is seen when their HF is complicated by renal insufficiency. Renal dysfunction in the setting of HF has been identified as “cardiorenal syndrome”, and is seen in 80% of acutely decompensated HF patients. The syndrome is characterized by fluid overload and diuretic resistance which further worsens their HF. The pathophysiology is complex and involves moving beyond our traditional explanations of renal failure in the HF patient to an intricate understanding of the connections between the heart, renin-angiotensin system, sympathetic nervous system, and inflammation. The presentation will review the prevalence, etiologies, prognostic factors, and pathophysiology of cardiorenal syndrome. Specific evidence-based interventions will be presented. A working understanding of basic renal and cardiac function is necessary for participants to have maximum understanding of this presentation. After attending this session, nurses will have an improved understanding of the connection between renal and the cardiac physiology, factors contributing to hospital admissions, evidence-based treatments available, and interventions the nurse can implement to decrease adverse outcomes in this complex patient population.

LEARNING OUTCOMES

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

1. Describe the interrelationships between cardiac and renal physiology and their influence on heart failure outcomes.
2. Discuss etiologies and symptoms of renal insufficiency in heart failure patients
3. Outline management strategies for cardiorenal syndrome in the heart failure patient

SUMMARY OF KEY POINTS

- I. Describe the interrelationships between cardiac and renal physiology and their influence on heart failure outcomes
 - A. Definitions:
 1. Heart failure: inability of the heart to pump blood at a rate commensurate with metabolic requirements; manifested as dyspnea, fatigue, and fluid retention; chronic, progressive disease characterized by frequent hospital admissions and high mortality rates.

2. Renal insufficiency: abrupt reduction of renal function with the progressive retention of nitrogenous water compounds; usually, though not always, reversible and accompanied by oliguria.
 3. “Cardiorenal syndrome is defined as moderate or greater renal dysfunction that exists or develops in a patient with heart failure (either systolic or with preserved systolic function) during treatment” (Heywood, 2004)
- B. Renal Physiology
 1. Renal blood flow control
 - a. Autoregulation: General principles
 - b. Neural control
 - c. Hormonal control
 - (1) Renin-angiotensin-aldosterone
 - (2) Antidiuretic hormone
 - d. Pharmacological effects
 2. Excretion of metabolic wastes
 3. Fluid regulation
 4. Blood pressure control
 5. Red blood cell synthesis and maturation
 - C. Cardiac physiology and the connections between renal function and heart failure
 1. Decreased ventricular function
 2. Decreased cardiac output
 3. Decreased tissue and organ perfusion
 4. Increased sympathetic activity (neural and hormonal abnormalities)
 5. Increased vascular resistance
 6. Worsening valve dysfunction – MR or TR
 7. Effects of RAAS on fluid retention
 8. Decreased ventricular function
 9. Role of inflammation / oxidative stress
- II. Discuss etiologies and symptoms of renal insufficiency in heart failure patients
 - A. Hypovolemia (decreased preload) – Prerenal
 1. Prerenal
 - a. Pathophysiology
 - (1) Decreased renal perfusion caused by decreased renal blood flow without damage to the renal tubules
 - (2) Renal function is completely normal: kidneys are unable to filter
 - (3) If the compromised perfusion persists, it can lead to irreversible ischemia
 - b. Etiologies
 - (1) Decreased circulating volume
 - (2) Decreased cardiac output
 - (3) Decreased vascular resistance

- c. Assessment
 - (1) Decreased circulating volume / decreased peripheral vascular resistance: Tachycardia, hypotension, orthostatic BP, dry mucous membranes, CVP less than 5, PAWP less than 10, flat neck veins, coma
 - (2) Cardiac failure: decreased cardiac output, hypotension, tachycardia, cool, clammy skin, PAWP greater than 18, JVD, friction rub
 - (3) Laboratory findings
 - (a) Oliguria (less than 400 cc/24 hours)
 - (b) Urine Na+ less than 10-15 mEq
 - (c) SG greater than 1.015
 - (d) BUN elevated
 - (e) Creatinine slightly elevated
 - (f) Urine osmolality greater than 500 mOsm / kg
 - (g) Urine Na+ and SG important
 - (h) BUN: creatinine ratio greater than 20:1
- B. Neurohormonally mediated vasoconstriction (increased afterload)
- C. Hypotension with preserved cardiac output (vasodilatory shock)
- D. Low-output syndrome
- E. Other
 - 1. Renal vascular disease
 - 2. Decreased GFR induced by certain classes of drugs
 - a. NSAIDs
 - b. Cyclosporine
 - c. ACE inhibitors and ARBs
 - 3. Intrinsic renal disease due to long-standing hypertension and/or diabetes
 - a. Intrarenal
 - (1) Pathophysiology
 - (a) Result of disease or injury to the nephron
 - (b) Initial insult leads to vascular swelling and self perpetuating ischemia which leads to actual damage to the glomerular filtration process
 - i. Ischemic
 - ii. Nephrotoxic
 - (c) Assessment
 - i. Same as prerenal-decreased cardiac output, hypovolemia
 - ii. Don't forget extrarenal losses: vomiting, diarrhea, hemorrhage, insensible losses (febrile)
 - iv Laboratory Findings
 - Oliguria / nonoliguria (most common with nephrotoxic)
 - SG less than 1.015
 - Osmolality : urine less than 350 mOsm

Urine Na+ greater than 30 mEq
 BUN greater than 30 mg
 Creatinine-elevated
 Acidosis
 Protein in urine

- III. Outline management strategies for cardiorenal syndrome in the heart failure patient (25 minutes)
 - A. Determine fluid status: Goal to achieve and maintain near euvolemic state
 - 1. Hypovolemia
 - a. Fluid administration
 - b. Stop diuretics
 - 2. Hypervolemia
 - a. Diuresis
 - b. Ultrafiltration
 - B. Manipulate Cardiac Output and SVR
 - 1. Vasopressor Medications
 - 2. Inotropic Medications – Short term only
 - 3. ACE Inhibitors – stop or start (Attempt to maintain RAAS blockade if possible)
 - 4. Nesiritide
 - 5. Erythropoietin
 - 6. Vasopressin antagonists
 - 7. IABP
 - 8. LVAD
 - C. Intervention via Renal Replacement
 - 1. Renal Replacement
 - a. Indications for renal replacement
 - (1) Volume overload
 - (2) Uncontrolled hyperkalemia
 - (3) Symptomatic uremia
 - (4) Pericarditis
 - (5) BUN greater than 100 mg / dL
 - b. Methods of renal replacement
 - (1) CVVHDF
 - (2) CVVHD

BIBLIOGRAPHY/WEBLIOGRAPHY

- Geisberg, C & Butler, J. Addressing the challenges of cardiorenal syndrome. *Cleveland Clinic journal of Medicine*, 2006, 73,5:485-491.
- Heywood, JT. The cardiorenal syndrome: Lessons from the ADHERE database and treatment options. *Heart Failure Reviews*, 2004, 9, 195-201.
- Francis, G. Acute decompensated heart failure: the cardiorenal syndrome. *Cleveland Clinic Journal of Medicine*, 2006, 72, supplement 2, S8-S13.
- Shlipak, M.G. & Massie, B. M. The clinical challenge of cardiorenal syndrome. *Circulation*;2007;110:1514-1517.
- Chittineni, H. Mayawaki, N., Gulipelli, S., Fishbane, S. Risk for acute renal failure in patients hospitalized for decompensated congestive heart failure. *Am J Nephrol* 2007;27:55-62.

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Hematology Assessment: The CBC with Diff and Coagulation Profiles

Carol A. Rauen

Level: Intermediate

CONTENT DESCRIPTION

The hematopoietic system is one of the most sophisticated systems in the human body. An evaluation of the complete blood count (CBC) and coagulation studies are common assessment strategies in critical care and are utilized for a variety of purposes. The hemoglobin and hematocrit and the white blood cell count are the commonly assessed parameters but so much more information can be gleaned from the CBC. The differential of the white blood cell count helps to identify pathology as well as evaluate treatment modalities. This session will discuss, in cellular detail, the many parameters and how they can be useful in diagnosis, evaluation and treatment. Clinical situations, specific disorders and case studies will be used to integrate the science to practice and demonstrate application of this information to patient assessment. The coagulation studies will also be discussed with evaluation parameters and common problems outlined. This session will review the blood tests that make up the CBC and coagulation profiles. This lecture will be useful for all specialty areas and all levels of practice from the bedside to advance practice, pediatrics to geriatrics. The physiology and pathophysiology of the hematopoietic system as they relate to lab value assessment will be discussed and clinical application and evaluation principles will be outlined.

LEARNING OUTCOMES

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

1. Identify the normal values for erythrocytes, leukocytes and coagulation profiles.
2. Integrate evaluating the CBC with differential and coagulation profiles into patient assessment and diagnostic strategies.

SUMMARY OF KEY POINTS

- I. Introduction
- II. ERYTHROCYTE (red blood cells) RELATED STUDIES
 - A. Red Blood Cell: Normal Count Male: 4.6 – 6.0 million/mm³, Female: 4.0 – 5.0 million/mm³

The erythrocyte is a soft and pliable biconcave disk that transports oxygen and carbon dioxide as well as functions as an intracellular buffer in the acid-base balance within the body. Erythropoiesis (forming of RBCs) takes place in the bone marrow when a stem cell differentiates into an erythrocyte. This process is regulated by the hormone erythropoietin, which is secreted by the kidney in response to hypoxia. The immature RBC is called reticulocyte and it normally takes 11 days for a reticulocyte to mature into a fully function-

ing erythrocyte. To make a healthy RBC the bone marrow needs Vit B12, folic acid, proteins, enzymes, iron and copper. The hemoglobin is made up of globin a polypeptide and four heme groups capable of carrying one molecule of oxygen each. The average life of an erythrocyte is 120 days and the body is consistently creating new cells and excreting old ones. The iron portion of the dying RBC is recycled and used again and the remainder is broken down to form bilirubin and excreted in the bile.

1. Elevated Red Blood Cell Count:

Polycythemia or Erythrocytosis Male: > 6.0 million/mm³, Female: > 5.0 million/mm³

Elevated levels of RBCs can be from primary or secondary causes. Polycythemia vera is a primary elevation in which the cause of the overproduction is not known. Secondary polycythemia is common in hypoxic states such as high altitude environments, increased physical training with athletes, chronic lung diseases and congenital heart defects. The concern with this disorder is that high levels of RBCs increase the individual's risk of venous thrombosis and this potential is exacerbated if the patient is in a dehydrated state. One tx for polycythemia is to keep the patient well hydrated if not over hydrated. Historically the tx for primary polycythemia was radioactive phosphorus to slow down the overactive bone marrow. Current tx is phlebotomy. The tx for secondary polycythemia is to treat the underlying cause of the hypoxia.

2. Low Red Blood Cell Count: Male: < 4.6 million/mm³, Female: < 4.0 million/mm³

Anemia is a nonspecific term for a low RBC count. To identify the cause and treatment the red blood cell indices must be evaluated. Potential causes are blood loss, microcytic anemia or macrocytic anemia. Treatment includes blood transfusion, erythropoietin administration and/or finding and treating the cause of the anemia

3. Red Blood Cell Indices: Analysis of the RBC count, size, weight, and hemoglobin concentration. Helpful in evaluating the presence of anemias and identifying the type.

a. Mean Corpuscular Volume (MCV):

Men: 78–100 cubic micrometers, Female: 78–102

MCV indicates the mean size of the erythrocytes. Microcytic erythrocytes are smaller than normal. Causes of microcytic anemia are iron deficiency, malignancy, rheumatoid arthritis, lead poisoning and radiation therapy. The hemoglobinopathies that cause microcytic anemia include Thalassemia anemia, Sickle cell anemia and Hemoglobin C anemia. Macrocytic erythrocytes are larger than normal. Causes of macrocytic anemia include aplastic, hemolytic and pernicious anemia, chronic liver disease, vit B12 deficiency, hypothyroidism and some medications. Macrocytosis can also be used as a marker for recent alcohol intake.

b. Mean Corpuscular Hemoglobin (MCH) & Mean Corpuscular Hemoglobin concentration (MCHC) MCH: 25–35 pg, MCHC: 31–37%

MCH is the amount of hemoglobin present in one cell weighed in picograms (pg). MCHC is the proportion of the cell occupied by hemoglobin. Both of these measurements are looking at the weight of the RBC relative to hemoglobin. These are both decreased in microcytic anemias and elevated in macrocytic anemias. Hypochromic describes a low hemoglobin complement and hyperchromic describes a high hemoglobin complement with normochromic being normal.

c. RBC Distribution Width (RDW): 11.5%–14.5%

RDW is a reflection of the size (width) of the erythrocytes. It is useful in predicting anemias early, before changes in the other indices occur or even physiologic symptoms from the anemia. Elevated RDW is the important value. Iron def, folic acid def, pernicious anemia and hemoglobinopathies can all elevate the RDW.

4. Erythrocyte Sedimentation Rate (Sed Rate):

Male: 0 - 17mm/hr, Female: 1 - 25mm/hr

The sed rate is a nonspecific test that is reflective of the time (speed) at which the red blood cells fall and settle to the bottom of the blood tube. The sed rate will increase when there is inflammation present in the body. Infections, cancer, inflammatory processes or diseases will all cause an increase sed rate. Trends can also be followed to identify if the anti-inflammatory therapies are working. A low sed rate has no clinical significance.

5. Reticulocyte Count: 0.5 - 2.5% of total RBC count

Reticulocytes are immature RBCs. An elevated level indicates increased production of RBCs from the bone marrow and occurs in sickle cell disease, thrombotic thrombocytopenic purpura, malaria and after blood

loss. Decreased levels are seen in most anemic states, radiation therapy, and alcoholism. Some drugs can also decrease levels.

6. Hematocrit (Hct): Male: 37–49% Female: 36–46%

The Hct represents the percentage of plasma that is red blood cells. It is a reflection of the RBC count. It is the volume measured in ml of RBCs found in 1dL (100ml) of plasma. A low Hct is present in acute and chronic blood loss, anemias, leukemias, cirrhosis, protein malnutrition, chronic renal failure, SLE, and also overhydration. An elevated Hct is found in dehydration, DKA, burns, severe diarrhea, emphysema and eclampsia. A false positive low value could result from drawing venous blood from an arm with an IV infusing.

7. Hemoglobin (Hgb): Male: 13–18 g/100ml Female: 12–16 g/100ml

Hgb is the protein substance found on the erythrocytes. It contains heme (iron) and globin (protein) and carries oxygen and carbon dioxide. Hgb levels will drop for all the same reasons the RBC or Hct levels drop. Hgb levels are also influenced by medications and will decrease with antibiotics, aspirin, chemotherapy agents, hydralazine, indomethacin, MAO inhibitors, and sulfonamides. Hemoglobinopathies (discussed below) will cause low Hgb levels. High Hgb levels can be found in dehydration, and those indications listed above for high Hct. Gentamicin and aldomet administration can increase the Hgb. Leaving a tourniquet on for > 1 min may cause homeostasis which could result in a falsely elevated Hgb level.

a. Hemoglobin Electrophoresis:

Hgb A1 = 95–98%, Hgb A2 = 1.5%, Hgb F < 2%, Hgb F, Hgb C, Hgb S are abnormal

b. Methemoglobin: < 1% of total Hemoglobin

Methemoglobin is formed when heme is oxidized to the ferric state in the blood and enzymes reduce it back to Hgb. The significance of methemoglobin is that it cannot carry oxygen. Cyanosis will occur if the level exceeds 15% of the Hgb. It is a greater problem in neonates because they lack the reducing enzymes.

III. LEUKOCYTE (white blood cell) DIFFERENTIAL

A. White Blood Cells: 4,500 - 11,000/mm³

The leukocytes are the body's physiologic defense from foreign pathogens. There are two major groups of white blood cells; the polymorphonuclear and mononuclear cells. Each group has distinct visible characteristic and functions in the defense & immune response.

1. Polymorphonuclear (PMN) Granulocyte Leukocytes

- a. Neutrophils: are the most abundant WBC type and the body's first line of defense against bacterial infection and severe stress. Neutrophils initiate phagocytosis and assist in the removal of foreign organisms, dead cells, and cellular debris. The cell nucleus of the mature neutrophil has a characteristic segment and the cells are frequently called segs. The immature neutrophil does not have this segment but has a band – and are called bands. Usually less than 5% of the WBC count are bands. An increased number of bands signify either a pathology or a stimulation of the immune system. This is called a “shift to the left” in the differential. When the WBC counts were written by hand the bands were counted first and therefore written on the left side of the page.
- b. Eosinophils: associated with allergic (antigen-antibody) reactions and parasitic infestations. Eosinophilia occurs with allergies, asthma, emphysema, renal disease, phlebitis and many cancers. The count will decrease with stress, elevated steroid (endogenous and exogenous) levels, hyperthyroidism and shock.
- c. Basophils: the function of these cells is not clearly understood. They appear to play a role in the healing process and hypersensitivity reactions. The count increases with leukemia, inflammation, and hemolytic anemia and decreases with stress and hypothyroidism. The absolute number is such a low % of the WBC count that is difficult to really identify a physiologically significant low volume of basophils.

2. Mononuclear Agranulocyte Leukocytes:

- a. Lymphocytes: Humoral (B Lymphocytes) and cellular (T Lymphocytes) regulate the immune response for the body. They provide recognition and eradication of foreign substances. Lymphocytosis occurs in viral and bacterial infections, most leukemias. Lymphopenia occurs in HIV+, AIDS, autoimmune diseases, SLE, MS, cancers, renal failure, severe malnutrition. Lymphocyte Immunophenotyping: the subsets of lymphocytes are classified by their clusters of differentiation (CD).
- b. Monocytes: the second line of defense against infection, have a strong phagocytes in chronic inflammatory disorders. Increased levels will be seen in protozoan infections (malaria), monocytic leukemias, chronic inflammatory diseases like colitis and enteritis, many cancers and sickle cell and hemolytic anemia. Decreased levels are

seen with aplastic anemia and lymphocytic leukemia.

IV. COAGULATION PROFILES

A. Introduction:

The clotting mechanics involve 12 clotting factors, the intrinsic and extrinsic pathways and many other physiological functions (ie liver function and healthy platelets) to be in balance for the patient not to bleed excessively or be hypercoagulable. We measure many of these parameters to determine if there is an impending problem or to discover mechanism is not functioning appropriately and therefore help to direct therapy.

B. Coagulation Studies

1. Platelet Count: 150,000 – 400,000/mm³

Thrombocytes (platelets) originate from stem cells in the bone marrow and are non-nucleated cells whose major function is to promote coagulation. The quantity of platelets is reflected in the count. The quality of the platelets or functional ability is another concern. If the platelets cannot function appropriately (in vonWillabrand's disease for example) there is a risk of bleeding despite the fact of a normal thrombocyte count.

a. Thrombocytosis: Platelets > 400,000/mm³

Causes for increased platelet count include leukemia, lymphoma, solid tumors (colon), metastatic carcinoma, PE, TB, and polycythemia vera. Some clinical situations that can cause an elevation in thrombocytes are trauma, postsplenectomy, after acute blood loss and with some medications (epinephrine). Thrombocythemia a count > 1,000,000 can occur with spontaneous thrombocytosis associated with malignancy.

b. Thrombocytopenia: Platelets < 150,000/mm³

Idiopathic thrombocytopenic purpura (ITP) is a condition of low platelet count of unknown origin. Most thrombocytopenia is easily explained by a medical condition or pathology. Common causes include: autoimmune diseases (SLE), AIDS, depressed bone marrow (medications, chemotherapy, radiation), DIC, most leukemias and anemias, liver disease, renal disease, eclampsia, HELLP syndrome, white clot syndrome, and hemorrhage. The fragile platelets can also be destroyed by an extracorporeal circulation mechanism like the cardiopulmonary bypass machine or continuous veno-venous hemofiltration (CVVH) circuit and medications like antibiotics, aspirin, and many diuretics. An artificial cardiac valve also can destroy platelets. The count is also lower in hemodilutional states. Thrombopoietin or Oprelvekin (neumega), a colony-stimulating factor, increases platelet stimulation and production. Platelet

- administration is another treatment option.
2. Prothrombin Time (PT): 11 – 15 seconds
 Factor II, Prothrombin, is a vitamin K dependent – liver synthesized clotting factor. Because of the inter-relatedness of all the clotting factors the PT is measuring more than just factor II function, it reflects factors I, V, VII, and X. The PT, and these factors, are most reflective of the extrinsic arm of the clotting cascade. This test is used clinically to determine the patient's ability to clot and also evaluate the therapeutic level of the anticoagulant Warfarin (Coumadin). The result is interpreted in relation to the control value for the lab in which the test was performed. Due to environmental variables and reagent utilized the result can be different from lab to lab. To combat this problem the patient's blood is compared to a "control blood" and the ratio is compared. A control value is always given with the patient's result.
 3. International Normalized Ratio (INR): 0.7 – 1.8
 The INR is not a blood test. It is a way of reporting the PT. Due to the fact that the PT normal range varied depending on the reagent and the lab the results were difficult to interpret and standardize. Using an international sensitivity index (ISI) and a table the PT can be reported as the INR and interpreted consistently regardless of the reagent used in any particular lab. The ISI is determined by the manufacture of the reagent and printed on the label. The lab performs the PT and then converts the result, using the table, to an INR.
 4. Activated Partial Thromboplastin Time: APTT 20-35 sec, Partial Thromboplastin Time: PTT 60–70
 The PTT is a nonspecific test, which reflects the function of the intrinsic clotting system and most clotting factors (not VII). It is a better screening test to detect deficiencies of clotting factors than the PT. The aPTT is a more sensitive test than PTT because of the reagent used. Heparin therapy is monitored with the PTT.
 - a. Increased PTT
 The PTT will elevate in Hemophilia A & B, DIC, factor IX deficiency, blood transfusions, liver failure, vonWillebrand's disease, malaria, most leukemias, and heparin therapy.
 - b. Decreased PTT: The PTT will decrease in pregnancy and extensive cancer states.
 5. Anti-Factor Xa: Normal 0U/ml LMWH
 Monitoring: DVT Treatment 0.4-1.1U/ml
 DVT Prophylaxis < 0.45U/ml
 Anti-Factor Xa is used to monitor the effectiveness of low molecular weight heparin (LMWH). These agents function as anticoagulants in the coagulation cascade at Xa (common pathway). Because of this the PT and PTT can not be utilized to evaluate the effectiveness of these agents. LMWHs have relatively predictable bioavailability (unlike unfractionated heparin) and therefore the effect is predictable. Routine monitoring is not necessary or recommended as long as the recommended dose is being administered.
 6. Bleeding Time: Ivy 1 - 8 min, Duke 1 - 3min
 The bleeding time is a direct method of measuring the functional ability of the vascular and platelet factors associated with hemostasis. Qualitative as well as quantitative abnormalities will be revealed. The test is accomplished by puncturing the arm (Ivy) or ear lobe (duke) and monitoring the time it takes for bleeding to stop. Platelet function and vascular response are not affected by the intrinsic or extrinsic systems so a patient may have normal coagulation studies (listed above) and still have an abnormal bleeding time.
 7. Activated Clotting Time: 70 – 120 seconds
 A bedside (OR or Cath lab) test for clotting time. Used to screen for coagulation problems or to regulate and evaluate anticoagulation therapy (heparin). It is reflective of the PTT and all information for PTT applies to the ACT. Whole blood is placed in a special blood tube, the tube is rotated (by hand or machine) and the time for clot formation is measured (by eye or machine). The therapeutic goal depends on the reason for the anticoagulation therapy.
 8. Fibrinogen: 200 – 400mg/dl
 Factor I (fibrinogen) is a non vit. K dependent plasma protein synthesized in the liver. In the clotting cascade thrombin splits fibrinogen to produce fibrin strands which are the end product of blood clotting. Low fibrinogen levels are seen in DIC, liver disease, leukemia and many obstetric complications. Prolonged PT, APTT, PTT and/or ACT may suggest a fibrinogen deficiency. Elevated levels are seen in acute infections, sepsis, inflammatory diseases, collagen diseases and hepatitis. Hormonal therapies and heparin can also increase the fibrinogen level.
 9. Thrombin Time (TT): 14 - 16 seconds
 The TT measures the quality of the functional fibrinogen. If a clot does not form immediately when thrombin is added to the patient's blood sample a fibrinogen deficiency is present. A different reagent

can be used if the patient is on heparin. The TT is commonly used to evaluate patient's receiving fibrinolytic agents.

10. Fibrin Degradation (Split) Products (FDP):
2–10 mcg/ml

FDPs are the byproduct of fibrinolytic activity (breakdown of clot). Thrombin, which initially enhanced clotting, promotes the conversion of plasminogen to plasmin. Plasmin breaks fibrinogen and fibrin to create FDP. These split products function as anticoagulants. Elevated FDP are seen in DIC, shock, massive tissue damage, sepsis, obstetric complications, acute MI, PE, liver and renal disease, burns and acute leukemia. Increases can also be seen in fibrinolytic therapy (streptokinase, urokinase, TPA).

11. D-Dimer: < 2.5 mcg/ml

The D-Dimer is the product of the degradation of cross-linked fibrin. Its presence represents fibrinolysis that is specific to DIC, PE, DVT and arterial thrombosis. It may also be present in sickle cell crisis and some cancers.

12. Protein C & Protein S: 70-140% (each)

Protein C & Protein S are naturally occurring anticoagulants. Their role in the clotting process is to limit the size of the clot. When these proteins are absent (congenital or acquired) or not activated there is an increased risk of vascular thrombosis.

V. SUMMARY/DISCUSSION

BIBLIOGRAPHY/WEBLIOGRAPHY

- DeLoughery, T. (2005). Critical care clotting catastrophes. *Crit Care Clin*, 21, 531-562.
- Dressler, D. (2006). Hematology and Immunology systems. *AACN Essentials of Critical Care Nursing*. McGraw Hill: New York; 305-315.
- Kee, J. (2005). Laboratory diagnostic test with nursing implications, 7th ed. Appleton & Lange: Connecticut.
- Munro, N. (2004). Hematopoiesis, coagulation and bleeding. In Morton & Fontaine: *Critical Care Nursing: A Holistic Approach*. 8th ed. Philadelphia: Lippincott, Williams and Wilkins.
- Pearl, R., Pohlman, A. (2002). Understanding and managing anemia in critically ill patients. *Crit Care Nsg*, supp. Dec
- Rampher, K., Little J. (2004). Assessment of red blood cell and coagulation laboratory data. *AACN Clin Issues*. 15, 622-637.
- Reid, M., Calhoun, L., Petz, L. (2006). Erythrocyte antigens and antibodies, chpt 128 In Lichtman M., et al Eds. *Williams Hematology*, 7th ed. New York: McGraw Hill Medical. 2119-2136.
- Sullivan, K., Kipps, T. (2006). Human leukocyte and platelet antigens. Chpt 129 In Lichtman M., et al Eds. *Williams Hematology*, 7th ed. New York: McGraw Hill Medical. 2137-2150.
- Vincent-Corbett, J. (2004). *Laboratory Tests and Diagnostic Procedures with Nursing Diagnoses*, 6th ed. Prentice Hall: New Jersey.

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Hepatic Failure, Comorbidity and Neurologic Complications

Richard Arbour

Level: Expert

CONTENT DESCRIPTION

Primary session purpose is to analyze and evaluate physiological consequences of hepatic failure related to effects on central nervous system (CNS) stability, specifically brain function. Secondary session purpose is correlating pathophysiology of hepatic failure and neurologic consequences such as intracranial hypertension and encephalopathy with management strategies. Aggressive, mechanism-based intervention is evaluated within the framework of serving as a bridge to orthotopic liver transplantation.

Session scope includes clinical, laboratory and radiographic and electrophysiologic findings specific for neurologic consequences of hepatic failure. Consequences such as blood-brain barrier disruption, brain edema, intracranial hemorrhage, intracranial hypertension and hepatic encephalopathy are analyzed within the context of patient safety and mechanism-based therapies. Lactulose and mechanisms of action for hepatic encephalopathy is reviewed. Interventions for brain edema including ICP monitoring, mechanism-based ICP reduction and drug-induced coma utilizing clinical and electrophysiologic monitoring are illustrated by case study. Hematological complications including thrombocytopenia, bleeding risk and coagulopathies as related to intracranial complications are correlated with interventions such as plasma volume replacement, blood and blood product resuscitation in minimizing risk of invasive procedures. Optimal clinical management is appraised as integral to optimal neurologic recovery, successful transplant outcome and minimizing ICU length of stay.

Target audience includes clinicians, educators and advanced practitioners managing hepatic failure. Content application optimizes management of neurologic consequences of hepatic failure, improves neurologic outcomes and decreases ICU length of stay. Participants possessing basic knowledge of intracranial physiology and liver dysfunction benefit most from session content. Session contains 75 % pharmacology content.

LEARNING OUTCOMES

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

1. Analyze physiological consequences of hepatic failure in context of consequences affecting multiple body systems.
2. Appraise neurologic consequences of hepatic failure in context of risk factors and comorbidities
3. Analyze plan of care for optimal clinical management of neurologic consequences of hepatic failure.

SUMMARY OF KEY POINTS

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

severe acute liver injury in patients with previously normal liver function: Multisystem consequences.

1. Impaired synthetic function.
2. Coagulopathy.
3. Encephalopathy.
4. Impaired membrane physiology
5. Brain edema.

- B. Etiologies: Viral, Drug/toxin causes (Acetaminophen), herbal products, halothane, analgesics.

II. Neurologic consequences: MOST COMMON CAUSE OF DEATH IN HEPATIC FAILURE!!!

- A. Hepatic encephalopathy: Reversible impairment of brain function in hepatic failure.

1. Serum ammonia: Direct neurotoxicity (primary event).
 - a. GI sources: Bleeding, consequent to liver impairment.
2. Secondary consequences: Neurotransmitter dysfunction.
 - a. Impaired blood to brain transport of amino acids.
 - b. Increased brain uptake of neutral amino acids.
 - c. Altered synthesis of neurotransmitters dopamine, norepinephrine and serotonin.
3. Increased intracellular osmolality in astrocytes: Functional changes.
 - a. Altered neuronal electrical activity: Inhibited excitatory and inhibitory impulses.
 - b. Impaired brain energy metabolism/ Neurotransmission:
 - c. Activation of astrocytic benzodiazepine receptors.
 - d. Altered blood-brain barrier.
 - e. Brain edema.

III. Management of hepatic encephalopathy:

- A. AGGRESSIVE management of causes.
 1. Reduction of ammonia levels.
 2. GI elimination, increased stool volume.
 3. Lactulose, increased fecal nitrogen elimination.
 4. Oral antibiotics: Rifaximin.

IV. Intracranial hypertension: Multifactorial consequence of hepatic failure.

- A. Brain edema: Increased brain bulk/poor intracranial compliance.
 1. Astrocyte swelling
 2. Altered blood-brain barrier
 3. Osmotic disturbance: Membrane failure.
 4. Increased brain water content.

- B. Intracranial hemorrhage: Risk factors.
 1. Coagulopathy.
 2. Cerebral hemodynamic stress.
 3. Invasive procedures.
- C. Invasive monitoring:
 1. ICP monitoring technology: Potential bridge to transplantation.
 - a. Correction of coagulopathy/Goal-directed therapy.
 - b. Assessment of intracranial compliance.
 - c. Titrating lung ventilation/osmotic diuresis/metabolic suppression therapy.
- D. Brain tissue approx. 80 % total intracranial volume.
 1. Hyperosmolar agent: Mannitol.
 - a. 0.25-1.0 g/kg body weight: Low-dose versus high-dose protocols.
 - b. Risks: Hypovolemia, electrolyte depletion, renal failure, ICP/edema rebound.
 - c. Evidence-based practice issues:
 - d. Outcomes, blood-brain barrier, administration issues, CBF augmentation.
 2. Hypertonic saline:
 - a. Fluid loss from swollen brain tissue: Concentration gradient.
 - b. Systemic/hemodynamic, CPP benefits.
 - c. CBF augmentation.
 - d. Evidence-based practice issues.
 - e. Variable concentrations utilized: 2% through 23% saline.
- E. Altered cerebral blood flow state: Generalized vasodilatation >>>> cerebral hyperemia.
 1. CBF autoregulatory failure.
 2. Poor intracranial compliance.
- F. Hyperemic cerebral blood flow states.
 1. Blood volume approximately 10 % intracranial volume (arterial and venous).
 2. Lung ventilation: PaCO₂ goal between 35-40 mm Hg; avoid hypercapnia.
 - a. SHORT-TERM, PaCO₂ between 25-30 mm Hg, prior to maximizing other interventions: Delay onset of brain herniation.
 - b. Real-time titration if CBF monitoring (direct or indirect) available.
- V. Metabolic suppression therapy:
 - A. Opioid analgesia: Morphine/Fentanyl
 1. CMRO₂/CBF
 2. ICP
 - B. Benzodiazepine sedation: Lorazepam/Midazolam - Liver oxidative stress
 1. Cerebrophysiologic effects:
 - a. CMRO₂/CBF
 - b. ICP
 - c. Increase seizure threshold.
 - C. Propofol
 1. High lipid solubility: Rapid onset/termination.
 2. Infusion only: Titration to therapeutic goals.
 3. Systemic/Cardiopulmonary effects:
- 4. Cerebrophysiological effects:
 - a. CMRO₂/CBF
 - b. ICP
 - c. MAP/ CPP
 - d. Neuroprotection
 - e. Free radical scavenging
- D. Barbiturates: Thiopental/pentobarbital.
 1. Lipid soluble agents, rapid traverse of blood-brain barrier
 2. Kinetics determined by multiple variables.
 3. Systemic effects: Myocardial depression, vasomotor loss (dose related).
 4. Monitoring issues:
 - a. Hemodynamic consequences/brain stem depression.
 - b. EEG: Burst suppression pattern/maximal reduction in cerebral metabolism.
- E. Hypothermia:
 1. Mechanism of action:
 - a. Reduction in cerebral blood flow/CMRO₂ and cerebral metabolic rate of glucose.
 - b. Decrease in CO₂, high-energy phosphates and lactate production.
 - c. Membrane stability, decreased ion exchange.
 2. Mild vs. moderate hypothermia:
 - a. Risk/benefit analysis.
 - b. Systemic complications
 - c. Patient selection/concurrent therapies
 - d. Outcomes.
 - e. Altered pharmacokinetics.
 - f. EEG/EEG-derived monitoring.
- VI. Case study one:
 - A. Mr. V. G 56 y/o patient: Medical ICU admission for evaluation/management of liver disease.
 1. Cirrhosis consequent to occupational toxin exposure/ Progressive decline in hepatic function.
 - B. Clinical issues: 2 days post-admission.
 1. Hypoxemia.
 2. Oliguria.
 3. Bleeding risk.
 4. Hemodynamic instability
 5. Decline in LOC, grade 3 encephalopathy.
 - C. Clinical management:
 1. Intubation/controlled ventilation.
 2. Dialysis catheter insertion:
 - a. Emergent dialysis.
 - b. CRRT q day X 8 hours.
 - c. Large volume plasmapheresis.
 - d. Replacement with FFP/clotting factors.
 - D. Pulmonary artery catheterization: Low SVR/PVR, high cardiac output state.
 - E. Progression to stage 4 encephalopathy/probable ICP elevation: Priority listing for transplant.
 1. ICP monitoring >>>> fiberoptic catheter: Intracranial hypertension.
 2. Urgent head CT: Optimal device placement, no bleeding.

3. Serial lab monitoring: CBC/electrolytes/Pt/Ptt/INR, fibrinogen/FSP.
- F. Further measures for ICP management:
Mechanism-based.
1. Short-term hypocapnia.
 2. Drug-induced coma.
 3. Therapeutic hypothermia.
 4. EEG-based monitoring: Barbiturate titration in real-time.
 - a. Effective cerebral metabolic control.
- G. ICU day 4- "Yellow alert" followed by "red alert":
OR for liver transplantation.
1. Good clinical outcome: Extubation POD 3, ICU discharge POD 5.
- VII. Case study two:
- A. Mr. D. P. 49 year-old with end-stage liver disease (ESLD): Admitted to ICU for transplant evaluation.
1. Priority listing for orthotopic liver transplantation.
 2. Baseline head CT.
- B. Clinical issues:
1. Coagulopathy.
 2. Hepatic encephalopathy.
 3. Respiratory failure/ventilator dys-synchrony.
- C. Clinical management:
1. Controlled ventilation: Sedation (propofol)/analgesia.
 2. Serial coagulation studies/INR.
 3. Lactulose.
- D. Daily "drug holiday" as clinically tolerated.
- E. Neurological assessment:
1. Minimal response to stimulation.
 2. Brainstem function marginal.
- F. Follow-up head CT.
1. Multiple intracerebral bleeds.

2. Family meeting:
3. DNR/Withdrawal of ventilation.
4. Comfort measures only.

BIBLIOGRAPHY/WEBLIOGRAPHY

- Han MK, Hyzy R. Advances in critical care management of hepatic failure and insufficiency. *Crit Care Med.* 2006; 34(9 Suppl): S225-S231.
- Gagliardi G, Laccania G, Boscolo A, La Guardia P, Arrigoni M, Michielan F. Intensive care unit management of fulminant hepatic failure. *Transplantation Proceedings* 2006; 38: 1389-1393.
- Norenberg MD, Rao KV, Jayakumar AR. Mechanisms of ammonia-induced astrocyte swelling. *Metab Brain Dis.* 2005; 20 (4): 303-318.
- Vaquero J, Rose C, Butterworth RF. Keeping cool in acute liver failure: Rationale for the use of mild hypothermia. *J Hepatol.* 2005; 43 (6): 1067-1077.
- Tofteng F, Hauerberg J, Hansen BA, et al. Persistent arterial hyperammonemia increases the concentration of glutamine and alanine in the brain and correlates with intracranial pressure in patients with fulminant hepatic failure. *J Cereb Blood Flow Metab.* 2006; 26 (1): 21-27.
- Rasian A, Bhardwag A. Medical management of cerebral edema. *Neurosurg focus.* 2007; 22: E12. Accessed 8-15-07 at <http://www.medscape.com/viewarticle/559004>.
- Jalan R, Damink SW, Deutz NE, Hayes PC, Lee A. Moderate hypothermia in patients with acute liver failure and uncontrolled intracranial hypertension. *Gastroenterology.* 2004; 127:1338-1346.
- Vaquero J, Blei AT. Mild hypothermia for acute liver failure: A review of mechanisms of action. *Clin Gastroenterol* 2005; 39(Suppl 2): s147-s157.

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Herbals and Medications: Common Interactions

Julia Baldwin

Level: Intermediate

CONTENT DESCRIPTION

Healthcare professionals in any patient setting need a working knowledge of the interactions between commonly used dietary supplements and prescription medications. This session will explore typical indications for use of herbal supplements and adverse reactions when combining commonly used herbal products with traditional OTC and prescription medications.

This session will address the 20 most commonly used herbal products, their active ingredients, and their interactions with commonly used traditional medications. The discussion will include a brief overview of herbal product terminology. Reasons for the increase in the use of herbal products and the rationale for not disclosing their use to healthcare professionals will be discussed.

LEARNING OUTCOMES

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

1. Describe the demographics of the typical herbal product user
2. Discuss the regulation and Safety of Herbal products
3. Discuss the origins, common uses, and typical medication interactions of 21 of the most commonly used herbal products

SUMMARY OF KEY POINTS

- I. Introduction / Nomenclature
 - A. Phytopharmaceuticals
 - B. Nutraceuticals
 - C. Alternative Medicines
 - D. Natural Medicines
 - E. Complementary and Alternative Medicine (CAM)
- II. Demographics and history of usage – Who has been and are using supplements?
 - A. Ancient to modern times
 - B. Older and younger population
 - C. United States compared to other countries
- III. Regulation and Safety Issues – How safe are they?
 - A. USP
 - B. German Monographs
 - C. Preparation and shipping
- IV. Origins and Common Uses of Selected or Most Popular Natural Medicines and their interactions with medications
 - A. Pure versus extracted components/ Definitions
 1. Aril – seed coating
 2. Bitters – stimulate salivary and digestive juices
 3. Coumarins – blood thinner, tanning skins, smooth muscle relaxant
 4. Flavonoid – blue to red pigments, antioxidants

5. Infusion – liquid preparation made by pouring water over it
6. Oils – essential – aromatic volatile oils extracted, may be antiseptic or insect repellents
7. Oils - infused – volatile oil plus another oil used in extraction
8. Phenols – varied group of constituents from acids to sugar based. Antioxidants, anti-inflammatories, antivirals.
9. Poultice – plant material wrapped in gauze and applied topically
10. Rhizome – underground plant stem, usually grows horizontally with roots on underside
11. Tannin – promotes skin tanning, used to tighten tissues, may stop bleeding
12. Tincture – extract made by soaking herbs in liquid, straining, then discarding plant material
13. Wort - plant
- B. Popular “natural” medicines – uses and medication interactions
 1. Bilberry
 2. Black Cohosh
 3. Chamomile
 4. DHEA
 5. Echinacea
 6. Ephedra
 7. Evening primrose
 8. Feverfew
 9. Garlic
 10. Ginger
 11. Ginkgo biloba
 12. Ginseng
 13. Glucosamine
 14. Green tea
 15. Guarana
 16. Hoodia
 17. Horehound
 18. Kava
 19. Mate
 20. Melatonin
 21. Milk thistle
 22. Red clover
 23. Red yeast rice
 24. Royal Jelly
 25. Salvia divinorum
 26. Saw Palmetto
 27. St. John’s Wort
 28. Yohimbe
 29. Valerian
- V. Patient / family education / Information Sources
 - A. Online sources

- B. Reference texts
 - C. National Goals
- VI. Summary

BIBLIOGRAPHY/WEBLIOGRAPHY

- Chevallier, A. (2000). *Natural Health Encyclopedia of Herbal Medicine*. 2nd ed. New York, New York: Dorling Kindersley.
- Kelly J, Kaufman D, Kelley K, et al. (2005). Recent trends in use of herbal and other natural products. *Archives of Internal Medicine*. 165: 281-286.
- Law, M. (2007). History of food and drug regulation in the united states. *EH. Net Encyclopedia*. Retrieved May 29, 2007, from <http://eh.net/encyclopedia/articl/Law.Food.and.Drug.Regulation>.
- Long C. Great online links: Complementary and alternative medicine. *Nursing* 2004; 34: 78.
- National Center for Complementary and Alternative Medicine. (2007). The use of complementary and alternative medicine in the United States. Retrieved May 28, 2007 from http://nccam.nih.gov/news/camsurvey_fs1.gtm.
- Natural medicines: Comprehensive database. (2005). Compiled by the Editors of *Pharmacist's Letter* and *Prescriber's Letter*. Stockton, California: Therapeutic Research Facility. <http://www.NaturalDatabase.com>.
- Skidmore-Roth, L. (2006) *Mosby's handbook of herbs and natural supplements*. 3rd .ed. St. Louis, Missouri: Mosby, Inc.
- U.S. Food and Drug Administration. Regulatory initiatives for dietary supplements. *FDA Consumer Magazine* [serial online]. January – February 2005.

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Historic Trauma Cases: Would They Survive Today?

Suzanne Frey Sherwood

Level: Beginner

CONTENT DESCRIPTION

Participants will journey into the past, discovering a unique perspective of how the treatment of trauma patients evolved into the current practice of trauma care today.

This presentation will utilize a case study approach of the medical and nursing care of trauma patients throughout history. From the era of the American Civil War, where many medical and surgical practices were developed as last hope efforts to save a life, to the recent medical and surgical practices utilized in the trauma setting today. Blunt and penetrating traumatic injuries will be discussed from a historic point of view. Trauma care of the past will then be compared with trauma care of today as well as where the future of trauma care will lead us.

Throughout history, health care professionals have looked to the past to create a future where evidenced based practice may be incorporated into how we care for trauma patients.

LEARNING OUTCOMES

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

1. Describe past medical/nursing care of the traumatically injured patient.
2. Identify current resuscitation practices utilized in trauma care today.
3. Discuss current/future trends of medical/nursing care of trauma patients.

SUMMARY OF KEY POINTS

I. Introduction

II. Who's who in trauma: General Thomas "Stonewall" Jackson

- A. Personal
- B. Professional
- C. Mechanism of injury
- D. Assessment
 1. Past 1863
 2. Present 2008
- E. Medical management
 1. Past 1863
 2. Present 2008

III. Who's who in trauma: President James A. Garfield

- A. Personal
- B. Professional
- C. Mechanism of injury
- D. Assessment
 1. Past 1881
 2. Present 2008

E. Medical management

1. Past 1881
2. Present 2008

IV. Who's who in trauma: Present William McKinley

- A. Personal
- B. Professional
- C. Mechanism of injury
- D. Assessment
 1. Past 1901
 2. Present 2008
- E. Medical management
 1. Past 1901
 2. Present 2008

V. Who's who in trauma: Princess Grace

- A. Personal
- B. Professional
- C. Mechanism of injury
- D. Assessment
 1. Past 1982
 2. Present 2008
- E. Medical management
 1. Past 1982
 2. Present 2008

VI. Who's who in trauma: Princess Diana

- A. Personal
- B. Professional
- C. Mechanism of injury
- D. Assessment
 1. Past France/1998
 2. Present USA/2008

VII. What's new on the horizon?

- A. Penetrating trauma
- B. Hemorrhagic shock
- C. Glycemic control in trauma patients
- D. Sepsis
- E. Non-invasive to damage control management of trauma

VIII. Conclusion

BIBLIOGRAPHY/WEBLIOGRAPHY

- Anderson C. The Day Diana Died. William Morrow and Co. Inc, New York, 1998.
- Beller, S. Medical Practices in the Civil War. Betterway Books, F & W Publications Cincinnati, Ohio 1992.
- Fakhry S, Trask A, Waller M et al. (2004). Management of brain-injured patients by evidence-based medicine protocol improves outcomes and decreases hospital charges. *J Trauma* 56(3) 492-499.

- Farwell B. *Stonewall: A Biography of General Thomas J. Jackson*. W.W. Norton Company, New York 1992
- Gordon RF. *The Alarming History of Medicine*. Sinclair-Stevenson Limited, Great Britain, 1993.
- Keel M & Meier C. (2007). Chest injuries- what is new? *Current Opinion in Critical Care* 13(6) 674-679.
- Kleinpell RM, Graves BT & Ackerman MH. (2006). Incidence, pathogenesis, and management of sepsis: an overview. *AACN Advanced Critical Care* 17(4) 385-393.
- Mitra B, Alfredo M, Cameron PA, Fitzgerald M et al. (2007). Massive blood transfusion and trauma resuscitation. *Injury, Int. J Care Injured* 38, 1023-1029.
- Porter, R. *The Greatest Benefit to Mankind*. W.W. Norton and Co., New York London, 1997.
- Potter WW. (1901). The assassination of President McKinley. *Buffalo Medical Journal* October, XLI (3) 226-233.
- Sherwood SF, Hartsock RL. *Thoracic Injuries in Trauma Nursing from Resuscitation Through Rehabilitation*, eds McQuillan, VonRueden, Hartsock, Flynn, Whalen. WB Saunders. 2002
- Soreide K, Petrone P & Asensio JA. (2007). Emergency thoracotomy in trauma: rationale, risks, and realities. *Scand J Surg* 96(1) 4-10.
- Sperry JR, Frankel HL, Vanek SL, Nathens, et al (2007). Early hyperglycemic predicts multiple organ failure and mortality but not infection. *J Trauma* 63(3). 487-494.
- Straubing HE, *Hospital and Camp: The Civil War through the Eyes of its Doctors and Nurses*. Stackhouse Books, USA, 1994
- Taraborrelli JR. *Once Upon a Time: Behind the Fairy Tale of Princess Grace and Prince Ranier*. WarnerBooks, New York, 2003.
- Weninger P & Hertz H. (2007). Factors influencing the injury pattern and injury severity after high speed motor vehicle accident-a retrospective study. *Resuscitation* 75, 35-41.
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Hold'em or Fold'em? Creating a NP Position in Acute Care

Kathy Austin

Level: Advanced Practice

CONTENT DESCRIPTION

Establishing the Nurse Practitioner (NP) role in acute care is no easy task, yet a much-needed endeavor. The purpose of this presentation is to offer some insight into overcoming the obstacles encountered in creating an NP position in the acute care setting. In addition, the presentation will evaluate how to identify when the encountered obstacles become insurmountable.

LEARNING OUTCOMES

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

1. Identify desirable qualities of an acute area in which to establish and NP role.
2. Identify potential obstacles to implementing a NP role in the acute care setting.
3. Identify 3 ways to evaluate effectiveness of the NP role in the implemented role.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. Good news – the need for NPs in acute care is growing
 - B. Bad news – implementing the role into actual practice is not easy
- II. Overview
 - A. Identifying a practice area
 - B. Assessing the need for an NP in a given area
 - C. Identifying potential obstacles
 - D. Setting a time frame for the implementation of the position
 - E. Evaluation of the effectiveness of the endeavor
 - F. Avoiding sacrificing the role for the job
- III. Hold 'em or Fold 'em
 - A. Identifying an area to practice
 1. Acute Care
 2. Intensive Care Areas
 3. Medical Surgical Areas
 4. Progressive Care
 5. Diagnostic Area
 - B. Assessing the need for an NP in a given area
 1. Is there a physician shortage?
 2. Is there a fragmentation in patient care?
 - C. Identifying potential obstacles
 1. Is there a clear geographic location in which to practice?
 2. Absence of clearly defined leadership
 3. Poor introduction of the NP role
 4. Collaborating MD lacks knowledge of the NP role

5. Billing issues
- D. Setting a time frame
 1. Individualized
 2. Be clear at the time the role is implemented what the desired time frame is
 3. Know that this can be re-negotiated
- E. Evaluation of Effectiveness
 1. Positive feedback from the medical and nursing staff.
 2. Are you accessible?
 3. Are you approachable?
 4. Commitment to high-quality care
 5. Improved patient outcomes
 6. Decreased length of patient's stay
 7. Communicates clearly with all disciplines in assigned area
 8. Improvement in core measures
 - a. medication reconciliation
 - b. increased compliance with DVT and GI prophylaxis
 - c. decreased pneumonia rate
- F. Avoiding sacrificing the role for the job
 1. Is this what you as an NP wanted?
 2. Does the job fit your description of your role?
 3. Do you feel effective?
- IV. Case Study #1
 - A. Overview of case
 - B. Audience brainstorming
 - C. Review of solution
- V. Case Study #2
 - A. Overview of case
 - B. Audience brainstorming
 - C. Review of solution
- VI. Summary

BIBLIOGRAPHY/WEBLIOGRAPHY

- Hoffman L, Happ MB, Scharfenberg C, DiVirgilio-Thomas D, Tasota F. Perceptions of physicians, nurses, and respiratory therapists about the role of acute care nurse practitioners. *American Journal of Critical Care*. 2004; 13: 480-488.
- Kleinpell R. Acute care nurse practitioner practice: results of a 5-year longitudinal study. *American Journal of Critical Care*. 2005; 14: 211-220.
- Shapiro D, Rosenberg N. Acute care nurse practitioner collaborative practice negotiation. *AACN Clinical Issues*. 2002; 13: 470-478.

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How do you Spell Relief? A Pressure Ulcer Prevention Bundle

Perrilynn Baldelli
Mary Paciella

Level: Beginner

CONTENT DESCRIPTION

Pressure ulcers continue to be a major health care problem. The prevalence and incidence rates of pressure ulcers and the cost of treatment, constitutes a substantial burden for our health care system and concern for health care providers. The purpose of this session is to describe the development of an evidence based pressure ulcer prevention bundle that is the central focus and driving force of a successful pressure ulcer prevention program. This program has decreased pressure ulcer prevalence and incidence rates in one institution. This program was developed based on the recommendations of the Wound Ostomy Continence Nurse's Society, Agency for Health Care Research and Quality, as well as experts such as Ayello and Braden. This session is designed for acute and critical care nurses, educators, and advanced practice nurses interested in reducing pressure ulcer development in hospitalized patients. There is no pre-requisite knowledge needed for this session. Prevalence and incidence definitions and data will be reviewed. This session will describe strategies used to develop, implement, and evaluate a comprehensive pressure ulcer prevention program. The participants will identify that the reduction of pressure ulcer rates requires education, the incorporation of evidenced based practice, improved communication, administrative support and ultimately, a culture change. The reduction of pressure ulcers is a way of "Reclaiming Our Priorities" to improve our patient outcomes as well as our quality of care.

LEARNING OUTCOMES

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

1. Verbalize an understanding of the incidence and prevalence of pressure ulcers and pressure ulcer rates.
2. Describe the components of the pressure ulcer prevention program.
3. Identify the elements of the pressure ulcer prevention bundle.

SUMMARY OF KEY POINTS

- I. Prevalence and Incidence
 - A. Definitions
 1. Prevalence
 - a. "Snap-shot" of one hospital day in which every patient's skin is assessed for pressure ulcers
 - b. Provides the percentage of pressure ulcers in a patient population at a given period of time
 - c. Includes facility acquired and community acquired pressure ulcers
 2. Incidence
 - a. Rate of nosocomial pressure ulcers that develop over a specific length of time

- B. Data review
- C. Benchmarking
 1. KCI USA, Inc/Novation
 2. NDNQI
- II. Pressure Ulcer Prevention Program
 - A. Development
 - B. Evidence Based Practice
 1. Pressure Ulcer Prevention Bundle
 - a. IHI "Bundle" Concept
 - (1) Bundle elements features/definition
 - b. Skin Assessment
 - c. Risk assessment
 - d. Turning and positioning
 - e. Heel elevation
 - f. Head of bed less than 30 degrees
 - g. Incontinence skin care regime
 - h. Pressure relief surfaces
 - i. Nutritional assessment/protocol
 - C. Campaign Theme
 1. "Check, Rock, and Roll Around the Clock"
 2. Education Program
 3. Communication
 - a. Posters
 - b. "Flat" Stanley
 - D. Evaluation Process
 1. Audits
 2. Unit pressure ulcer reports
 3. Prevalence and incidence studies
- III. Program Outcomes
- IV. Future Directions
- V. Questions and Discussion

BIBLIOGRAPHY/WEBLIOGRAPHY

- Lyder C. Pressure Ulcer Prevention and Management. *The Journal of the American Medical Association*. 2003; 289(2): 223- 226.
- Lyder C, Yu C, Stevenson D, et al. Validating the Braden Scale for the prediction of pressure ulcer risk in blacks and Latino/Hispanic elders: a pilot study. *Ostomy Wound Manage*. 1998;44(suppl3A):42S-49S.
- Novation/KCI National Prevalence & Incidence Study Report. KCI USA, Inc. 2006
- Ratliff CR, Bryant DE. *Guideline for Prevention and Management of Pressure Ulcers*. Glenview, IL: Wound, Ostomy, and Continence Nurses Society; 2003
- Reddy M, Gill S, Rochon P. Preventing Pressure Ulcers: A Systemic Review. *The Journal of the American Medical Association*. 2006; 296(8): 974-984.

Whittington, K, Briones, R. National Prevalence and Incidence Study: 6-Year Sequential Acute Care Data. *Advances in Skin & Wound Care*. 2004; 17 (9):490 – 494.

The Agency for Health Care Research and Quality (AHRQ).
The AHRQ National Guidelines Clearinghouse page.
Available at: <http://guidelines.gov>

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How to be an AACN Speaker

NTI Work Group

CONTENT DESCRIPTION

Presenting at AACN national meetings is an exciting professional opportunity. This session will provide the participant with an overview of AACN's Speaker Resource Network, as well as provide practical information on how to prepare an abstract for presentation. The selection process for topics included in national AACN programs such as the NTI and API will also be discussed.

LEARNING OUTCOMES

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

1. Identify the topic and format of presentations sought for AACN programs.
2. Describe the selection process for NTI/API sessions.

SUMMARY OF KEY POINTS

- I. AACN's Programming
 - A. NTI
 - B. API
 - C. Other
- II. Topic Areas
 - A. Clinical - Adult, Pharmacology, Pediatric, and Neonatal
 - B. Influencing Skills, Management, Emergency Department, Leadership Development
 - C. Education
 - D. Research
 - E. Professional Enrichment
- III. Characteristics of AACN Programs
 - A. Supports vision
 - B. Supports mission
 1. Clinical mastery
 2. Role mastery
 3. Systems mastery
 - C. Supports values
 - D. Systems-thinking perspective

- E. High 'take home and use' value
 - F. Addresses the continuum of care
 - G. Promotes critical thinking and decision-making skills
 - H. Incorporates research
 - I. Builds knowledge about practice of critical care
 - J. Reflects multidisciplinary approach
- IV. "Powered by Insight"
- A. Plan early!!
 - B. Consider options to increase newcomer experience – Learning Connection Mentor Sessions
 - C. Investigate a topic
 - D. Decide on Scope – not too big, not too little, not too specific, not too general
 - E. Identify your target audience
 - F. Use a unique approach to delivery your message
 - G. Organize abstract according to established formats (Resource information available at <http://www.aacn.org>)
 - H. Nurture your abstract
 - I. Show your spark! - be knowledgeable, passionate, inspired, and creative
 - J. Expect reference checks
 - K. Revise and finalize
 - L. Always read the Call for Abstracts instructions and follow completely
 - M. Submit ONLINE and Wait!
- V. Selection Process for NTI/API
- A. Building blocks and template
 - B. Assessment of current hot topics and needs
 - C. Curriculum development
 - D. Deadlines, deadlines, deadlines – know those dates
 - E. NTI Work Group
 - F. Research Work Group
 - G. Advanced Practice Work Group

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I Can't Believe It's Not Real: Using Human Patient Simulation

Sherri Harkless
Paula Garvey

Level: Beginner

CONTENT DESCRIPTION

In order to prepare nurses to make decisions in a health care climate that demands expertise in clinical areas, innovative approaches to teaching and learning in a collaborative model for acquisition of skills is important. An alternative to more standard approaches has been the increasingly popular introduction of high fidelity human patient simulators to hospital-based education departments. This session is designed to introduce the critical care nurse to Human Patient Simulation (HPS) and become familiar with implementing this type of teaching modality. One of simulation's many benefits is that nurses can gain clinical experience in a safe, non-threatening environment. From simulation, they have gained a foundation for clinical judgment, reasoning and critical thinking. Simulation allows the learning to be participant driven. The instructor is there simply as a facilitator and must make the situation applicable to the participants and the environment they are accustomed to. Doing this enables the participant to put theory into practice and leave the session with a positive feeling of accomplishment. Simulation is applicable for many types of situations such as code blue readiness, ACLS, problematic disease processes, collaborative team approaches and critical care equipment training. The desired outcome of this session is to increase the participant's knowledge, understanding, and applicability of simulation which will include participation in a simulation scenario. This presentation is directed towards educators, clinical nurse specialists, and registered nurses interested in using simulation. No prerequisite knowledge is required for this session.

LEARNING OUTCOMES

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

1. Explore the benefits of HPS as a teaching modality for critical care nurses.
2. Examine reasons to implement simulation as a teaching modality in a hospital-based staff development department.
3. Demonstrate the care of a patient utilizing the HPS.

SUMMARY OF KEY POINTS

- I. Benefits of HPS as a Teaching Modality
 - A. Simulation background
 - B. Gain skills in a safe, non-threatening environment
 - C. Gain a foundation for critical thinking
 - D. Explore different treatment regimens and see different consequences without harm to a real patient
 - E. See classroom theory put into practice

- II. Implementation of Simulation in Course Development
 - A. Failure to Rescue
 - B. ACLS
 - C. Code Blue Readiness
 - D. Unit-based issues such as relationship between RN-PCA
 - E. RN-MD communication and collaboration
 - F. ECG Class
 - G. Central Nurse Orientation
- III. Patient Care management
 - A. Management of Chest Pain
 - B. Management of Respiratory distress
 - C. Management of Cardiac Arrest

BIBLIOGRAPHY/WEBLIOGRAPHY

- Beyea SC, von Reyn L, Slattery MJ. A nurse residency program for competency development using human patient simulation. *Journal for Nurses in Staff Development*. 2007;23(2):77-82.
- Bush MC, Jankouskas TS, Sinz EH, Rudy S, Henry J, Murray WB. A method for designing symmetrical simulation scenarios for evaluation of behavioral skills. *Simulation in Healthcare: The Journal of the Society for Simulation in Healthcare*. 2007;2(2):102-109.
- Day L. Simulation and the teaching and learning of practice in critical care units. *American Journal of Critical Care*. 2007;16(5):504-507.
- Glavin RJ. Simulation: An agenda for the 21st Century. *Simulation in Healthcare: The Journal of the Society for Simulation in Healthcare*. 2007;2(2):83-85.
- Jankouskas T, Bush MC, Murray B, et al. Crisis resource management: evaluating outcomes of a multidisciplinary team. *Simulation in Healthcare: The Journal of the Society for Simulation in Healthcare*. 2007;2(2):96-101.
- Jeffries P. A framework for designing, implementing, and evaluating simulations used as teaching strategies in nursing. *Nursing Education Perspectives*. 2005;26(2):93-103.
- Nehring W, Lashley F. Current use and opinions regarding human patient simulators in nursing education: an international survey. *Nursing Education Perspectives*. 2004;25(5):244-248.
- Winslow S, Dunn P, Rowlands A. Establishment of a hospital-based simulation skills laboratory. *Journal for Nurses in Staff Development*. 2005;21(2):62-65.

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If It's Not Tamponade . . . Rarer Complications of Cardiac Surgery

Elizabeth A. Mattox

Level: Intermediate

CONTENT DESCRIPTION

Over 600,000 open-heart procedures are performed annually in the U.S. While post-operative hemorrhage, cardiac tamponade and rhythm disturbances are quickly identified, rarer complications exist that also require early recognition at the bedside. Delay in diagnosis and treatment of these complications contributes to increased morbidity and mortality. Adding these complications to the realm of the "possible" allows for enhanced nursing surveillance, timely identification, focused assessment and appropriate nursing and medical management.

In the early post-operative period, vasoplegia syndrome presents as hypotension refractory to catecholamines. Other patients have severe pulmonary dysfunction due to a systemic inflammatory response. Gastrointestinal complications occur in less than 5% of patients but have an overall associated mortality of 20 - 60%. All three of these early complications can result in profoundly ill and unstable patients. The neuropsychological effects of cardiac surgery have been identified in the literature, although little is known about the cause. An estimated 30-79% of patients describe post-op cognitive dysfunction ("pump head"), which can interrupt executive level functioning and may be persistent. Other patients experience anxiety, depression and post-traumatic stress disorder. Neuropathies are serious complications.

Phrenic neuropathy may prolong dependence on mechanical ventilation. Optic and upper extremity neuropathies can be particularly distressing to patients and may limit their ability to return to work or significantly impact quality of life.

Lastly, chest wall complications present later in recovery and contribute to permanent disability. Sternal wound infection and mediastinitis are linked to increased mortality and morbidity, as well as huge social and financial costs. Sternal avascular necrosis, mechanical dehiscence and chronic chest wall pain will also be discussed.

The focus will be on rapid identification and treatment of each syndrome. Assessment findings, appropriate diagnostics, risk factor identification and prevention strategies will be described. Treatment strategies including pharmacological and non pharmacological will be discussed. Photographs and diagnostic imaging will be shown.

LEARNING OUTCOMES

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

1. Participate in the early recognition of "rarer" complications following cardiac surgery.
2. Identify patients at higher risk for "rarer" complications of cardiac surgery.
3. Expand general knowledge about the spectrum of surgical complications.

SUMMARY OF KEY POINTS

- I. Vasoplegic Syndrome
 - A. Criteria: hypotension, tachycardia, low filling pressures, high/normal CI/CO, low SVR and POOR RESPONSE to volume and catecholamines
 - B. Risk factors include certain (common) pre operative medications and prolonged CPB time
 - C. Treatment strategies: methylene blue, vasopressin
 - D. Comments on Trendelenberg, propofol infusion syndrome and adrenal insufficiency
- II. Pulmonary Dysfunction following Cardiac Surgery
 - A. ARDS
 - B. TRALI
 - C. Pulmonary Emboli
- III. Gastrointestinal Complications
 - A. Mesenteric ischemia
 - B. Acute pancreatitis
 - C. Complications following transesophageal echocardiogram
- IV. Neurological/Cognitive/Psychological Complications
 - A. Neurocognitive decline following cardiac surgery
 - B. Depression, anxiety and PTSD
 - C. Peripheral neuropathies
 1. Phrenic nerve damage/dysfunction
 2. Upper Extremity neuropathies
 3. Ophthalmologic complications including POIN
- V. Chest Wall Complications
 - A. Sternal wound infections (superficial & deep)
 - B. Avascular sternal necrosis
 - C. Mechanical/non infectious dehiscence
 - D. Chronic post operative pain syndromes
 - E. Note on closure techniques

BIBLIOGRAPHY/WEBLIOGRAPHY

- Baumgartner, FG (2004) Cardiothoracic Surgery. 3rd Edition. Landes Bioscience, Georgetown, TX.
- Bojar, RM (2004) Manual of Perioperative Care in Cardiac Surgery, 4th Edition. Blackwell Publishing, Malden, MA.
- Gomes WJ; Carvalho AC; Palma JH; Teles CA; Branco JN; Silas MG; Buffolo E. Vasoplegic syndrome after open heart surgery. *J Cardiovasc Surg (Torino)*. 1998 Oct; 39(5):619-23.
- Jolles H; Henry DA; Roberson JP; Cole TJ; Spratt JA. (1996) Mediastinitis following median sternotomy: CT findings. *Radiology* Nov;201(2):463-6.
- Loop FD; Lytle BW; Cosgrove DM; Mahfood S; McHenry MC; Goormastic M; Stewart RW; Golding LA; Taylor PC. Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care. *Ann Thorac Surg* 1990 Feb;49(2):179-86; discussion 186-7.

- Olbrecht VA; Barreiro CJ; Bonde PN; Williams JA;
Baumgartner WA; Gott VL; Conte JV. Clinical outcomes of
non infectious sternal dehiscence after median sternotomy.
Annals thoracic surgery 82(3): 26: 902-907.
- Shaw PJ; Bates D; Cartlidge NE; Heaviside D; French JM;
Julian DG; Shaw DA. Neuro-ophthalmological complications
of coronary artery bypass graft surgery. Acta Neurol Scand
1987 Jul;76(1):1-7.
- Weissman, C. (2004) Pulmonary complications after cardiac
surgery. Seminars in Cardiothoracic and Vascular Anesthesia.
8(3): 185.

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Immunity to Change

John Forman

Level: Intermediate

CONTENT DESCRIPTION

The “Immunity to Change” process was originally designed by Robert Kegan, professor of Adult Learning and Professional Development at the Harvard University Graduate School of Education. The process helps people to surface internal contradictions and personal “assumptions-taken-as-truths.” It creates a simple, but elegant map or diagnostic of a dynamic we call the “immunity to change.” The process is built on the discovery of a set of new ways of talking about our situations that honor the complex nature of every person. Each new form of discourse represents the transformation of a more familiar mode. The exercise is remarkably powerful as a stand-alone workshop, but participants tend to find additional momentum by taking what they have learned back into the workplace. Consulting assistance is available to anyone interested in a specific follow-up. In the workshop, participants will be helped to create their own custom-designed version of what is usually a highly powerful personal technology for transformational learning. Participants are asked a sequence of reflective questions – each designed to provoke a distinctly different form of discourse with a debriefing partner. The process does invite people out into deep waters, but it is always left to the individual participant to set the depths to which they choose to go. By the end, each person will have an actionable, new path for learning their way through intractable problems, enabling them to make sustainable change, not just temporary or apparent change.

While individual participants will benefit from this workshop, the process is especially useful for nurse executives, supervisors, charge nurses or any other team leader interested in the pursuit of lasting change.

LEARNING OUTCOMES

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

1. Understand themselves and the nature of “non-change” better.
2. Take new actions toward a desired outcome
3. Inspire new commitment and energy in their workplaces
4. See opportunities for effecting lasting change in small or large groups

SUMMARY OF KEY POINTS

Each workplace is, among other things, a “language community.” The way that we think and talk about our workplaces, and the associated emotions that arise, influence what we are able to see and do. Consequently, one of the more powerful avenues to coping with change (both desired and imposed) is to find new forms of discourse.

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Implementation of Remotely Monitored ICUs

Level: Beginner

CONTENT DESCRIPTION

The use of remotely monitored intensive care units (ICUs), or telemedicine, is a solution that has emerged to bridge the gap between the increasing size of the critical care population and a shortage of intensivists. Approximately 6 million critically ill adults are admitted to ICUs each year in the United States. Hospitals are increasing their focus on ICU patient safety and care standards, and they also are facing mounting pressure to reduce total length of stay in the face of increasing case complexity and volume. Meanwhile, facilities constantly struggle to recruit and retain medical team members.

This presentation details the key components, operational implications and likely benefits of these innovative systems. Participants will explore potential business models and a detailed implementation plan. Emphasis will be placed on the benefits of remotely monitored ICUs including improved quality of care, increased physician productivity, increased patient satisfactions and potential profitability despite a significant capital investment. describe the key components, operational implications and likely benefits of remotely monitored ICUs.

LEARNING OUTCOMES

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

1. Discuss specific quality and safety, productivity, satisfaction and profitability implications.
2. Review the benefits and challenges of 3 business models for remotely monitored ICUs
3. Explore guidelines and tools to initiate the evaluation, planning and implementation process.

SUMMARY OF KEY POINTS

The use of remotely monitored intensive care units (ICUs), or telemedicine, is a solution that has emerged to bridge the gap between the increasing size of the critical care population and a shortage of intensivists. Approximately 6 million critically ill adults are admitted to ICUs each year in the United States. Hospitals are increasing their focus on ICU patient safety and care standards, and they also are facing mounting pressure to reduce total length of stay in the face of increasing case complexity and volume. Meanwhile, facilities constantly struggle to recruit and retain medical team members.

Remotely monitored ICU systems can monitor the physiological signs of multiple ICU patients and engage on-site clinical staff when a patient's status changes. Standardization of care is possible since the remote command center observes patients at not just one ICU, but across multiple ICUs and potentially across multiple facilities.

Today, remote monitoring is focused on the ICU. However the platform technology is versatile enough to expand to other care settings, such as the emergency department. Staffing shortages, consumer demands and quality expectations will require hospitals to rethink how they engage patients. Early adopters of remote monitoring competencies will be well positioned to expand services as the technology evolves.

This presentation details the key components, operational implications and likely benefits of these innovative systems. Participants will explore potential business models and a detailed implementation plan. Emphasis will be placed on the benefits of remotely monitored ICUs including improved quality of care, increased physician productivity, increased patient satisfactions and potential profitability despite a significant capital investment.

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Implications and Applications of Noninvasive Positive Pressure Ventilation

Mark Bauman

Level: Intermediate

CONTENT DESCRIPTION

Noninvasive positive pressure ventilation (NPPV) also referred to as non invasive ventilation, is the application of positive pressure without the use of an artificial airway. The use of NPPV has been gaining wider acceptance as an alternative to invasive ventilation. It may decrease complications of intubation, in particular; nosocomial pneumonia and airway injury, and allow for earlier extubation. In addition, NPPV can be initiated, discontinued and re-initiated easily for the patient who requires intermittent support. In contrast to invasive ventilation, the upper airway remains intact with NPPV, preserving normal airway reflexes and defense mechanisms. The patient may also be able to eat, drink, verbalize, and expectorate secretions.

The purpose of this presentation is to increase the nurse's understanding of NPPV and to discuss the essential role that nurses play in optimizing the effectiveness of this modality. Key concepts to be covered include: complications of invasive ventilation, NPPV as an alternative ventilation strategy, identifying appropriate patients, devices and interfaces, indications for use, and the bedside nurse's role during implementation. This lecture is intended for novice to experienced nurses in critical and progressive care environments who desire to enhance their knowledge about this form of ventilation and its contributions to patient outcomes.

LEARNING OUTCOMES

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

1. Review potential complications of invasive mechanical ventilation including: increased need for sedation, ventilator-associated pneumonia (VAP), injury to airway structures, increased intensive care unit (ICU) stay and associated costs.
2. Appraise the conceptual and practical considerations of NPPV, including: the time course of respiratory failure, disease states and implementation on the front and back side of respiratory failure.
3. Review commonly used patient – ventilator interfaces, and the role of the bedside nurse in implementation, maintenance and optimization of NPPV.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Overview of mechanical ventilation
 - A. Associated complications and costs.
 - B. The etiology and significance of ventilator associated pneumonia (VAP)
- III. Conceptual course of respiratory failure
 - A. Definition and prevalence

- B. Delaying intubation with NPPV
- C. Disease states appropriate for NPPV
- D. Importance of spontaneous breathing for gas distribution
- E. The use of NPPV to facilitate extubation
- IV. The practical of noninvasive positive pressure ventilation (NPPV)
 - A. Modes of ventilation and patient ranges
 - B. Dedicated NPPV machines versus mechanical ventilators
 - C. The patient – machine interface: overview of masks and prongs
- V. The bedside nurse – the essential component for NPPV implementation
 - A. Application, troubleshooting, and discontinuation of NPPV
 - B. Rethinking patient assignments
 - C. A truly multi disciplinary treatment approach
 - D. Summary
- VI. Conclusions / Questions and answers

BIBLIOGRAPHY/WEBLIOGRAPHY

- Antonelli M, Pennisi A, Montini L. Clinical review: Noninvasive ventilation in the clinical setting - experience from the past 10 years. *Critical Care* 2005; 9:98-103
- Fernandez NM, Villagra A, Blanch L, Fernandez R. Non-Invasive mechanical ventilation in status asthmaticus. *Intensive Care Medicine*. 2001;27:486-92
- Ferrer M, Esquinas A, Arancibia F, Basuer Tt, Gonzalez G, Carrillo A, Rodriques-Roisin R, Torres A. Noninvasive Ventilation during Persistent Weaning Failure. A Randomized Controlled Trial. *American Journal of Respiratory Critical Care Medicine* 2003;168:70-76
- Garpestad E, Brennan J, Hill N. Noninvasive ventilation for critical care. *Chest* 2007; 132; 711-720
- Habashi N, Andrews P. Ventilator strategies for posttraumatic acute respiratory distress syndrome: airway pressure release ventilation and the role of spontaneous breathing in critically ill patients. *Current Opinion Critical care* 2004; 10(5):549-557
- Squadrons E, Frigerio P, Fogliati C, et al. Noninvasive vs invasive ventilation in COPD patients with severe acute respiratory failure deemed to require ventilatory assistance. *Intensive Care Medicine*, 2004; 30(7):1303-1310
- Warren DK, Shukla SJ, Olsen MA, Kollef MH, Hollenbeak CS, Cox CJ, Cohen MM, Fraser VJ. Outcome and attributable cost of ventilator-associated pneumonia among intensive care unit patients in a suburban medical center. *Critical Care Medicine*, 2003; 31:1312-7.

Improving Pain Management Practices for ICU Elderly

Diane J. Mick

Level: Intermediate

CONTENT DESCRIPTION

Many older patients experience chronic and persistent pain, and they bring this unrelieved pain with them to the ICU, even when they are being treated for another illness. Persistent pain in elders may not necessarily be associated with a recognizable disease process, and thus, these patients are at risk for under-recognition and under-treatment of their pain. Because the present older generation was brought up to believe that their care providers are “always right”, and because they may fear being labeled as “demanding” or “uncooperative”, older patients may be hesitant to report pain, even when it becomes severe. Everyday nursing activities, such as turning, bathing, and getting a patient out of bed, may precipitate excruciating skeletal pain that takes hours to resolve. Simple observation of an older patient’s physical presentation and responses to even minor activities will help to cue the clinician to when provision of pain relief is critical. Older patients typically respond better to around the clock dosing rather than to intermittent dosing. Certain types of persistent pain respond to adjuvant medications, such as antiarrhythmics, anti-depressants, and anticonvulsants. Some older patients may worry about the risk of opioid addiction in response to treatment of severe pain. Explanations of differences between tolerance and addiction, and reassurance that taking opioids for pain control rarely leads to addiction, will help to calm these fears. Two case examples of unrecognized/under-treated pain in hospitalized elders will be utilized to demonstrate how unrelieved pain can impede or delay a recovery trajectory. In this session targeted to both staff nurses and APNs, a review of the most recent American Geriatrics Society Panel Guideline on Persistent Pain in Older Persons will help to ground our discussion toward the goals of ensuring comfort, safety, and peace for ICU elders in our care. No prerequisite knowledge is required.

LEARNING OUTCOMES

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

1. Compare and contrast categories of pain among older patients.
2. Differentiate among effective pharmacological measures for persistent, severe, moderate, and mild pain among older patients.
3. Correlate risk of side effects and adverse reactions with opportunity for pain relief among older patients.

SUMMARY OF KEY POINTS

- I. Background on Persistent Pain in Older Adults
 - A. Modified nomenclature from “chronic pain” in 1998 Clinical Practice Guidelines to “persistent pain” in 2002 Clinical Practice Guidelines
 - B. Definitions for persistent pain
 1. Unpleasant and emotional experience

2. Complex phenomenon derived from sensory stimuli and moderated by
 - a. Individual memory
 - b. Expectations
 - c. Emotions
 - C. Is there a difference in meaning between “chronic” and “persistent”?
 1. Chronic pain
 - a. “Label” associated with negative images and stereotypes
 - (1) E.g., long-standing psychiatric problems
 - b. Treatment futility
 - c. Malingering
 - d. Drug-seeking behavior
 2. Persistent pain
 - a. Term may foster a more + attitude among both patients and clinicians
 - b. Provides hope for effective tx to alleviate suffering
 - D. ...what the patient says it is...
 1. Difficulty with identifying biological markers of presence of pain
 2. Most reliable evidence for presence and rating of intensity: patient’s description and self-report
 - E. Impact of Pain on Health and Society
 1. Primary reason people seek health care
 2. “Pain can be relieved effectively for 90% of patients, but is not relieved effectively for 80% of patients” Walco, NEJM, 1994.
 3. Harris Telephone Survey: One in five older Americans is taking analgesics regularly (several times weekly)
 - a. 63% had taken Rx pain meds for > 6 months
 - b. 45% who had taken Rx pain meds regularly had seen 3 or more providers for pain in past 5 years, 79% of whom were PCPs
- II. Practice Obligations
 - A. Beneficence (duty to benefit another)
 - B. Nonmaleficence (do no harm)
 1. Obligates all clinicians to provide effective pain management and comfort to all persons
 - a. Including those vulnerable individuals who are unable to speak for themselves (principle of Justice)
 - C. Respect for human dignity: 1st principle in the Code of Ethics for Nurses (ANA, 2001)
 - III. Common Causes and Consequences of Persistent Pain in Elders
 - A. Causes
 1. Arthritis

- 2. Bone and joint disorders
- 3. Neuropathies
- 4. Post-herpetic neuralgia
- 5. Other
 - a. Severe interstitial cystitis
 - b. Vasculopathic pain
- B. Consequences
 - 1. Depression
 - 2. Decreased socialization
 - 3. Sleep disturbance
 - 4. Impaired ambulation
 - 5. Increased health care utilization & out-of-pocket costs
- IV. Classifications of Persistent Pain
 - A. Nociceptive pain
 - 1. Arises from stimulation of pain receptors
 - 2. Tissue inflammation, mechanical deformation, ongoing injury, or destruction
 - 3. Usually responds well to common analgesic meds and non-pharm strategies
 - B. Neuropathic pain
 - 1. Results from pathophysiologic process involving peripheral NS or CNS
 - 2. Does not respond as predictably as nociceptive pain to conventional analgesics
 - 3. Does respond to unconventional analgesics such as
 - a. TCAs
 - b. Anti-convulsants
 - c. Anti-arrhythmics
 - C. Pain of mixed or undetermined pathophysiology
 - 1. Arises from mixed or unknown mechanisms
 - 2. Recurrent headaches
 - 3. Some vasculitic pain syndromes
 - 4. Treatment outcomes unpredictable
 - D. Psychologically-based pain syndromes
 - 1. Psychological factors have a role in onset, severity, exacerbation, or persistence
 - a. Psychogenic pain
 - 2. Conversion reactions
 - 3. Somatoform disorders
 - 4. Treatment: psychiatric intervention
 - 5. Traditional analgesia not indicated
- V. Assessment of Persistent Pain in Older Adults
 - A. Assess on initial presentation
 - B. Ongoing or recurrent pain with impact on function or QOL needs treatment
 - C. Use terms synonymous with pain
 - 1. Burning
 - 2. Discomfort
 - 3. Aching
 - 4. Soreness
 - 5. Heaviness
 - 6. Tightness
 - D. For those with cognitive or language impairments, or nonverbal pain behavior, recent changes in function and vocalization suggest pain as a potential underlying cause
- E. Seek reports from caregivers
- F. Symptoms of pain in impaired persons:
 - 1. Changes in gait
 - 2. Withdrawn or agitated behavior
 - 3. Moaning
 - 4. Groaning
 - 5. Crying
- G. Manage underlying disease
- H. In presence of substance abuse, refer to expert in pain and addiction management
- I. Patients with life-altering intractable pain: Refer to multidisciplinary management team
- J. Case: Mary
 - 1. Frail 89 year-old community-dwelling female
 - a. Adm to PCU for ‘chest pressure’ and hypotension
 - b. Hx of mult old comp fx of spine
 - c. Hx of delirium secondary to HF and polypharmacy
 - d. On Fentanyl patch 125 mg q 3 days
- VI. Hierarchy of Pain Assessment Techniques (Pasero & McCaffery, 1999; 2005)
 - A. Self-report
 - B. Search for potential causes of pain
 - C. Observe patient behaviors
 - D. Surrogate reporting
 - E. Attempt an analgesic trial
 - F. Use behavioral pain assessment tools
 - G. Minimize emphasis on physiologic indicators
 - 1. Changes in HR, BP, RR are not sensitive for discriminating pain from other sources of distress
 - H. Reassess and document
- VII. Pharmacologic Principles of Treatment of Persistent Pain
 - A. Benefit vs burden
 - B. Selection of appropriate agent, dose, route, and interval
 - C. Aggressive titration
 - D. Prevention of pain; relief of breakthrough pain
 - E. Use of appropriate co-analgesic (adjuvant) medications
 - F. Prevention and management of side effects
- VIII. Examples of Adjuvants or Co-Analgesia
 - A. Anticonvulsants
 - B. Antidepressants
 - 1. cyclobenzaprine, the “failed” antidepressant
 - C. Steroids
 - D. Counter-irritants
 - E. Baclofen
 - F. Capsaicin
 - G. Lidocaine
 - H. TCAs
 - I. Others
- IX. Non-Pharmacologic Treatment of Persistent Pain
 - A. Self-administered therapies

- B. Other therapies
 1. Relaxation techniques
 2. Guided imagery
 3. Music therapy
 4. Pet therapy
 5. Art therapy
 6. Biofeedback
 7. Hypnosis
- C. Rehab consult (OT, PT) for devices to minimize pain and to facilitate movement & activity
 1. Splints
 2. TENS
 3. ROM
 4. ADL re-training
- X. Anesthesia Pain Management Consult
 - A. Neuroaxial analgesia
 - B. Epidural steroid injections
 - C. Neuromodulation
 - D. Case: Helen
 1. Frail 84 year-old community-dwelling married female
 2. Adm for exacerbation of HF Stage III NYSHA
 3. PMH: severe generalized osteoarthritis, avascular necrosis R shoulder
 - a. While in hospital, becomes uncharacteristically combative during care
 - b. Tearful, non-communicative, except with family
 - c. Tells pastoral care representative "Nurses are rough with me"

- XI. Future Orientation
 - A. Ensure patient autonomy to extent possible
 - B. Compassionate communication
 - C. Equitable access to pain management

BIBLIOGRAPHY/WEBLIOGRAPHY

- American Geriatrics Society Panel Guideline on Persistent Pain in Older Adults. Management of persistent pain in older adults. *JAGS*. 2002;50:S205-S224.
- American Medical Directors Association. Chronic pain management in the long-term care setting. 2003; Columbia, MD: Author.
- American Pain Society. Pain: The fifth vital sign. 2007. <http://www.ampainsoc.org/advocacy/treatment.htm> Accessed December 21, 2007.
- Chatap G, DeSousa A, Giraud K, Vincent JP. Pain in the elderly: prospective study of hyperbaric CO2 cryotherapy (neuro-cryostimulation). *Joint Bone Spine*. In press 2007.
- Herr K, Coyne PJ, Key T, Manworren R, McCaffrey M. Pain assessment in the nonverbal patient: Position statement with clinical practice recommendations. *Pain Manag Nurs*. 2006;7: 44-52.
- Jones KR, Fink R, Hutt E, Vojir C, Pepper G. Measuring pain intensity in nursing home residents. *J Pain Symp Manage*. 2005;30:519-527.
- Melzack R. The tragedy of needless pain. *Scien Amer*.1990;262: 27-33.
- Schuler M, Njoo ., Hestermann M, Oster P, Hauer K. Acute and chronic pain in geriatrics: Clinical characteristics of pain and the influence of cognition. *Pain Med*. 2004; 5:253-262.

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Improving Patient Safety through Listening to Patients and their Families

A Dialogue with the Presidents of AACN, American College of Chest Physicians, Society of Critical Care Medicine, and American Thoracic Society

Dave Hanson, AACN

Alvin Thomas, Jr. ACCP

Philip S. Barie, SCCM

David Ingbar, ATS

SESSION SUMMARY

Communication gaps continue to be a huge problem in healthcare and contribute to a significant number of adverse patient events. A short video program will be presented that discusses the critical importance of listening to patients as a key element in ensuring safety. Following the presentation the presidents of the four critical care societies will respond and react to the challenge put forth in the video presentation from their role as an individual healthcare professional. Opportunities for the four societies to work together on a national level to drive changes in hospital systems and improve patient safety will also be discussed.

LEARNING OUTCOMES

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

1. Describe the four A's of the performance gap created by poor communication in hospitals.
2. Describe two immediate actions they will personally take in their healthcare organization to raise awareness and improve problems caused by not listening to patients and families.
3. Understand how the four critical care societies can work together to drive change in hospital systems to improve patient safety.

In the Nick of Time: Rapid Response to the Rescue

Mary Kay Bader
Connie Stalcup
Beverly Neal

Level: Intermediate

CONTENT DESCRIPTION

The Institute for Healthcare Improvement considers Rapid Response Teams (RRT) an essential element to reducing mortality in hospitals. 60-80% of patients sustaining cardiac/respiratory arrest demonstrate signs and symptoms of clinical deterioration in the eight hours prior to the event. RRT deployed in hospitals across the country seek to reduce the incidence of arrests on Medical-Surgical units. Training nurses to recognize the early signs of deterioration or the “gut feeling that something is not quite right” and signaling the RRT is vital to reducing out of ICU arrests. Calling the RRT to quickly assess the patient in extremis, institute immediate interventions, and transfer patients to a higher level of care, if needed, are fundamental strategies to reverse the death spiral. Although RRT have been reported to reduce morbidity and mortality, instituting them challenge hospitals due to costs and resources. In this presentation, one hospital’s efforts to institute a RRT will be presented as well as the outcomes associated with the implementation of a dedicated RRT nurse.

LEARNING OUTCOMES

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

1. Identify key elements of constructing a RRT protocol
2. Deliver one strategy to fund a RRT program
3. Develop a RRT standardized protocol including a proactive rounding program to identify at risk patients, maximize resources, and responses to other populations such as sepsis and stroke
4. Apply interventions used in actual case scenarios to clinical practice.

SUMMARY OF KEY POINTS

- I. Introduction
 - A. IHI’s directive “Saving 100,000 lives”
 1. Out of ICU arrests have high mortality
 2. 60-80% demonstrate signs of decline in preceding 6-8 hours
 - B. Past 10 years higher acuity patients occupying Med Surg beds
 1. Nursing Shortage
 2. Staffing patterns
- II. Literature Review on RRTs
 - A. Increase in “preventable” deaths in hospitals across country
 1. Failures in planning: Includes assessments, treatments, goals
 2. Failure to communicate: Patient-to-staff, staff-to-staff, staff-to-physician, etc.
 3. Failure to recognize a problem

- B. These three problems often lead to failure to rescue!
- C. Clinical instability prior to arrest
 1. 70% (45/64) arrests with evidence of respiratory/neurologic deterioration with 8 hours (Schein, Chest 1990; 98: 1388-92)
 2. 66% (99/150) abnormal signs and symptoms within 6 hours of arrest and MD notified 25% of cases (25/99) (Franklin, Crit Care Med;1994;22: 224-247)
 - a. Warning signs within 6 hours of event: MAP <70 or >130 mmHg, Heart rate <45 or >125 per minute, Respiratory rate <10 or >30 per min, Chest pain, and Altered mental status
- D. Implementation of medical emergency teams (MET) aka Rapid Response Teams (Bellomo. Med J Aust. 2003;179(6):283-287)
 1. Key factors: Rapid assessment/stabilization, optimal communication with physicians and staff, education opportunities with floor staff, and assistance with acute patient and possible transfer to higher level of care
 2. 3 Key features must be present: RRT must
 - a. Be available for immediate response
 - b. Be present on site
 - c. Possess the critical care skills necessary to assess and respond
 3. RRT made up of one or more of the following
 - a. Highly skilled critical care nurse
 - b. Respiratory Therapist
 - c. Physician
 4. Results of implementation of a RRT
 - a. Bellomo et al (Med J Aust 2003)
 - b. Buist et al (British Medical Journal 2002)
 - c. Goldhill, Anesthesia 1999
- III. Planning of RRT
 - A. Baseline: 46% of all arrests occurring outside the ED/Critical Care areas from 2003-2006 with 62.7% overall mortality
 - B. Analysis of high risk patients
 1. ED critical patients: Stroke, Sepsis, and STEMI
 2. Vulnerable patients in non-critical care areas
 - C. Administrative team directive: Institute a RRT
 1. Multidisciplinary team met, reviewed literature, and developed a standardized Procedure with a 24/7 RRT nurse as key to providing immediate care to high risk patients
 - a. RRT Job Description: Characteristics
 - b. RRT Standardized Procedure
 - D. Funding: Convincing administration need for a dedicated 24/7 RRT nurse

1. Round 1: Presentation at QLC
2. Round 2: Rapid cycle testing - RRT 4 day clinical trial
 - a. Staffed by CNSs and 2 senior nurses
 - b. Results and study findings
3. CNO and Critical Care Director to the rescue
 - a. Plan: Seek cooperation from nurse managers of every unit to give up
 - b. Methods: Do your homework and present case to managers
4. Finalizing 4.2 FTEs to staff the initiative

IV. Implementing RRT

- A. Hiring the Ideal RRT nurse
 1. Qualities
- B. Education
 1. Educating/credentialing of RRT nurse
 2. Rollout education of hospital staff
- C. Role
 1. Proactive rounds on non-critical care units
 2. Calls:
 - a. Emergency Department
 - b. Non-critical care units
 3. Education
 4. Support
- D. Data collection
 1. Prospective data
 - a. Rounds
 - b. Calls
 2. Entering Data

V. Analysis of RRT

- A. Phase I: September 06-December 06
 1. Quantitative data:
 - a. Call data (average 200-250 calls per month)
 - b. Transfers to higher level of care
 2. Qualitative data: issues that bubble to the surface
 3. Round data
 4. Resuscitation data: Pre RRT and Post RRT
 5. Closing the loop: Taking action
- B. Phase II: September 06- September 07
 1. Quantitative data:
 - a. Call data (averaging 375-425 calls per month)
 - b. Transfers to higher level of care
 2. Qualitative data: issues that bubble to the surface
 3. Round data
 4. Resuscitation data: Pre RRT and Post RRT
 - a. Number of codes in non critical care areas
 - b. Mortality of codes in non critical care areas
 5. Closing the loop: Taking action

VI. Case Studies in RRT – The Real Deal

VII. Summary

- A. Out of Critical Care Arrests:
 1. The incidence of floor codes has been reduced from 36/year to 16/year representing a greater than 50% reduction in arrests.
 2. The mortality has decreased from 62% to 25% (statistically significant decrease $p < .05$).
 3. Transfers from the floor to CICU revealed a decline in transfer by 25-40% from year prior statistically significant ($p < 0.05$, 36.5% RRR, 3.1% ARR).
- B. The deployment of the RRT in the past year has led to the identification of important clinical and system/process issues which have been addressed or are currently being explored by the hospital.

BIBLIOGRAPHY/WEBLIOGRAPHY

- Ashcraft A. 2004. "Differentiating between pre-arrest and failure to rescue". *MedSurg Nsg* 13(4): 211-215.
- Bellomo R, Goldsmith D, Uchino S, et al. 2004. "Prospective controlled trial of effect of medical emergency team on postoperative morbidity and mortality rates". *Crit Care Med* 32 (4): 916-921.
- Bristow P, Hillman K, Chey T et al. 2000. "Rates of in-hospital arrests, deaths, and intensive care admissions: the effect of a medical emergency team." *MJA* 173: 236-240.
- Buist M, Moore G, Bernard S et al. 2002. "Effects of medical emergency team on reduction of incidence of and mortality from unexpected cardiac arrests in hospitals: preliminary study". *BMJ* 324: 1-6.
- Foraida M, DeVita M, Braithwaite S, et al. 2003. "Improving the utilization of medical crisis teams (condition C) at an urban tertiary care hospital". *Journal of Critical Care* 18(2): 87-94.
- Franklin C and Mathew J.1994. "Developing strategies to prevent in hospital cardiac arrest: Analyzing responses of physicians and nurses in the hours before the event". *Crit Care Med* 22(2): 244-247.
- Institute for Healthcare Improvement. 2005. "Getting Started Kit: Rapid Response Teams – How to Guide". 1-26.
- Rosenberg A and Watts C. 2000. "Patients readmitted to ICUs: A systematic review of risk factors and outcomes". *Chest* 118: 492-502.

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Inpatient Management of Diabetes Mellitus: Beyond the Insulin Infusion

Denise Buonocore

Level: Advanced

CONTENT DESCRIPTION

This session will highlight medical management of the patient with hyperglycemia and DM when in the hospital beyond insulin infusions. Focus will be on medications both orals and injectables and when they should be started, continued or stopped, basal, bolus, correction insulin, and insulin transition protocols.

LEARNING OUTCOMES

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

1. Identify and illustrate effective utilization of DM medications in the hospital setting including which DM medications could be continued or stopped
2. Apply principles of basal, bolus, correction insulin dosing for optimal BG control in the inpatient arena.
3. Describe different methods of transitioning patients off insulin infusions.

SUMMARY OF KEY POINTS

- I. Overview of the impact of Hyperglycemia on inpatient morbidity and Mortality
- II. Treatment of inpatient Hyperglycemia
 - A. Patients with insulin deficiency
 - B. Patients with Type 2 DM
- III. Overview of Medications to Control DM
 - A. Oral agents
 1. Secretagogues
 - a. sulfonylureas
 - b. Meglitinides
 2. Biguanides
 - a. Metformin
 3. a-Glucosidase Inhibitors
 - a. acarbose
 - b. miglitol
 4. Thiazolidinediones (TZDs)
 - a. Rosiglitazone
 - b. Pioglitazone
 5. DPP- 4 inhibitors
 - a. sitagliptin phosphate (Januvia)
 - B. Injectables
 1. Insulins
 - a. Rapid acting
 - b. Long acting

2. Glucagon-like Peptide 1 Mimetic

a. Exenatide (Byetta)

3. Amylin Analog

a. Pramlintide(Symlin)

IV. Inpatient Glycemic Goals (ADA and AACE)

A. ICU target 110 mg/dL

B. Ward target:

1. Fasting: 110mg/dL(range 90-130)

2. Post prandial 180 or less

V. Hospital Treatment

A. Insulin Infusion

B. Basal, Bolus and Correction Insulin method

C. Insulin Infusion Transition

VI. Case Studies

BIBLIOGRAPHY/WEBLIOGRAPHY

ACE/ADA Task force on Inpatient Diabetes Mellitus. American College of Endocrinology and American Diabetes Association Consensus Statement on inpatient diabetes mellitus and Glycemic control. *Endocr Pract* 2006; 12(4) July/Aug 458-469.

Abern M, Boland E, Rothenberg D, Colombo JA, Baldwin J, Baldwin D. Intensive insulin therapy after coronary artery bypass surgery. *Diabetes*.2004; 53(suppl 2):492

Bode BW, Braithwaite SS, Steed RD, et al. Intravenous insulin therapy indications, methods and transition to subcutaneous insulin therapy. *Endocr Pract* 2004; 10(suppl. 2): 71-80

Clement S, Braithwaite SS, Magee MF, et al. American Diabetes Association Diabetes in Hospitals Writing Committee: management of diabetes and hyperglycemia in hospitals. *Diabetes Care*.2004; 27:553 -591.

Inzucchi S. Management of hyperglycemia in the hospital setting. *N Engl J Med* 2006; 355:18 1903-1911.

Inzucchi S. Glycemic management of diabetes in the perioperative setting. *Int Anesthesiol Clin*. 2002; 40(2) 77-93

Nathan, D. M., Buse JB, Davidson MD et al. Management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy. *Diabetes Care*2006; 29(8) 1963-1972.

Umpierrez G, Isaacs S, Bazargan N, You X, Thaler L, and Kitabchi A. Hyperglycemia: an independent marker of in-hospital mortality in patients with undiagnosed diabetes. *J Clin Endocrinol Metab* 2002; 87(3) 978-82

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Interdisciplinary Performance Improvement: Taking the Lead

Kathryn T. Von Rueden

Level: Intermediate

CONTENT DESCRIPTION

The purpose of this mastery session is to provide participants with tools and knowledge to more effectively facilitate, lead, and participate in PI teams. This session includes an overview of several performance improvement (PI) models; discusses in detail the essential principles and components of the performance improvement process; and reviews a number of strategies to facilitate or lead successful teams. Critical aspects which will be discussed in this session include: identifying appropriate benchmarks by which to gauge clinical performance and focus PI initiatives in the critical care arena; assembling an interdisciplinary team; methods to analyze and present data effectively; development of action plans; strategies to implement transformations required for change; and evaluating the impact of the changes.

LEARNING OUTCOMES

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

1. Describe different models which can be used to guide the performance improvement process.
2. Apply knowledge of the key process steps to a performance improvement initiative.
3. Utilize a variety of charts and graphs in the analysis and effective presentation of performance data.

SUMMARY OF KEY POINTS

- I. Overview of Performance Improvement
- II. Performance Improvement Models
 - A. PDCA (Rapid cycle change model)
 1. Focus: Using available data
 2. Plan: Develop action plans
 3. Do: Implement action plans
 4. Check: Measure the impact
 5. Act: Hold the gain or revise the plan
 - B. FADE (Rapid cycle change model)
 1. Focus: Using available data
 2. Analyze: Influential factors
 3. Develop: Solutions and plans
 4. Execute plan and Evaluate impact of plans
 - C. Juran: 12 Steps
 - D. RCA: Root Cause Analysis (primarily used for Level 1 or Level 2 adverse events; result in or require intervention to prevent death or serious disability)
 - E. FMEA: Failure Mode Effectiveness Analysis (primarily used to focus on processes and proactively prevent errors in execution)
 - F. 6 Sigma: DMAIC (Define, Measure, Analyze, Improve, Control)

1. Six Sigma < 3.4 defects / 1 million opportunities, or 99.9997 % perfect

III. Performance Improvement Process Steps and Strategies Applied to F-PDCA Model

A. FOCUS: Identify the Opportunity: Target a SPECIFIC issue or opportunity

1. Establishing quality indicators as triggers for PI initiatives
2. Characteristics of good quality indicators
 - a. Pertinent, Relevant, Valid, Reliable, Objective, Measurable
3. Trigger points
 - a. Thresholds, benchmarks, targets, goals
4. Process vs Outcome measures
5. Sources for Quality Indicators and Improvement Opportunities (Comprehensive list of national measures in Pyle K, Wavra T: Quality Indicators for Critical Care. *Advanced Crit Care* 2007 18(3)229-243. (note: NPSG are 2007))
 - a. JCAHO/NQF Hospital Quality Measures Data
 - b. Variation from professional organizations, HC industry, national, state standards or benchmarks, e.g. CDC, NDNQI, NTDB, NRMII
 - c. Evidence based practice protocols, guidelines; www.aacn.org; www.ihl.org/IHI/Topics/Improvement; www.mihealthandsafety.org
 - d. Efficiency Data
 - e. Unit/Dpt Specific Report Cards
6. Prioritizing PI initiatives
 - a. Develop a scoring grid, ranking patient safety issues with highest priority
7. Data Analysis & Interpretation: Required to focus PI initiative and to evaluate impact
 - a. Examples of data displays to facilitate analysis/interpretation
 - (1) Pie Chart- Show relative proportion
 - (2) Bar Chart- compares categories or quantities
 - (3) Pareto Chart- bar chart to display causes of a problem ranked by frequency
 - (4) Histogram- bar chart that displays variation at a point in time
 - (5) Scattergram- determine correlation between variables
 - (6) Run Chart- data over time compared with a median or threshold value
 - (7) Control Chart- data overtime, uses standard deviation for upper or lower limits

B. PLAN

1. Establish the Interdisciplinary Team

2. Meeting “AVs”
3. Define the Team Charge and Establish Project Goals
 - a. Goals must be: Simple, Objective, Measurable
 - b. Balance of “efficiency” with “quality measures
4. Analyze the current process, practice, or performance
 - a. How can we improve, and “do better”?
 - b. What are the causes of variation or less than optimal performance?
5. Examples of tools to analyze process:
 - a. Gap analysis, e.g. current practice vs best practice/evidence
 - b. Flow chart- analyze and understand the process
 - c. Fishbone- causal interactions
6. Explore Potential Solutions
 - a. Brainstorming technique
 - b. Methods to determine priority and feasibility of various solutions/actions
 - c. Consider: Cost, Time, Support, Challenges, Metrics
7. Develop “Solutions” and action plans
 - a. Use of E-B resources e.g. from literature, professional organizations
 - b. Identify driving and restraining forces
 - c. Strategies to overcome obstacles to change
 - d. Share action plans, processes, protocols, etc. widely
 - e. Consider pilot studies
 - f. Soliciting feedback from stakeholders
8. Define Implementation Plan Need to answer: Who –What – When – Where
 - a. Include re-measures or milestones
- C. DO: Implement the “solutions” and action plan
 1. Kick off date and Market the project
 2. Monitor implementation plan and maintain communication lines
 3. Solicit feedback and Re-convene the team for periodic updates
- D. CHECK: Evaluate impact / Re-measure metrics:
 1. Were goals/outcomes achieved? Are data sufficient to evaluate impact?

2. Reporting frequency
3. Share Results Widely!
- E. ACT: Adopt or Adjust
 1. Continue to monitor/track improvement
 2. Continue to share data and performance
 3. Ask: What are the next steps to CONTINUOUSLY improve?
- IV. Take Homes
 - A. PI is a continuous process!
 - B. Organization and pre-meeting planning is critical to success
 - C. Can’t solve a “problem” before we know what it is
 - D. Implementation plan is essential
 - E. “Talk it up” in every phase, especially post-implementation
 - F. It takes a village....
 - G. Don’t be shy about publishing/sharing even the smallest gains and achievements

BIBLIOGRAPHY/WEBLIOGRAPHY

- Buonocore D. Leadership in Action: Creating a change in practice. AACN Clin Issues 2006;15: 170-181.
- Curry SJ: Organizational Interventions to encourage guideline implementation. Chest 2000 118:40S-46S.
- Curtis RJ, Cook DJ, Wall RJ, et al: Intensive care unit quality improvement: A “how to” guide for the interdisciplinary team. Crit Care Med 2006; 34:211-218.
- Harrigan S, Hurst D, Lee C, et al: Developing and implementing quality initiatives in the ICU: Strategies and outcomes. Crit Care Clinics No America. 2006; 18: 469-479.
- JCAHO: Tools for performance measurement in healthcare: A quick reference guide. JCAHO, Oakbrook Terrace, IL 2002.
- Kleinpell R (guest ed) Quality Indicators in Acute and Critical Care. Crit Care Nurs Clinics 2006;18(4). Entire issue.
- Perkins S, Connerney I, Hastings C: Outcomes management: From concepts to application. AACN Clin Issues 2000; 11: 339-350.
- Pronovost P, Holzmueller C, et al: How will we know patients are safer: An organization-wide approach to measuring and improving safety. Crit Care Med 2006;34:1988-1995.

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Intensive Power Units: Healthy Work Environments that Promote Certification

Milisa Manojlovich
Greta Lynn Krapohl

Sponsored by AACN Certification Corporation

Level: Beginner

CONTENT DESCRIPTION

The purpose of this presentation is to demonstrate how healthy work environments contain characteristics that contribute to empowered nurses, who in turn are able to gain specialty certification. Key topics for this session include: a discussion of healthy work environment characteristics that promote nurse empowerment; strategies to build powerful work environments and strategies to support nursing staff in certification efforts. A case study will be used to highlight strategies that build healthy work environments that are also powerful, and demonstrate how the transformation to powerful work environments can promote certification. This session will be of special interest to managers, directors, clinical nurse specialist and staff leaders seeking to create healthy, powerful work environments, and promoting specialty certification. The session has two prerequisite skills: a passionate commitment to building powerful work environments and the conviction that specialty certification is worth its investment.

LEARNING OUTCOMES

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

1. Describe four work environment characteristics known to promote nursing empowerment.
2. Discuss strategies that will transform an intensive care unit into an intensive power unit.
3. Design specific, tailored strategies to foster certification.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Power-creating elements of a healthy work environment
 - A. Information
 - B. Support
 - C. Resources
 - D. Opportunity
- III. Strategies that nurse leaders can use to create powerful work environments
 - A. Strong, visible, supportive nursing leadership
 - B. Adequate staffing and resources
 - C. Professional model of nursing care
 - D. Collegial nurse/physician relationships
 - E. Nursing involvement in hospital affairs
- IV. Linking Empowerment to Certification
 - A. Meaning

- B. Impact
- C. Autonomy
- D. Competence
- V. Case study presentation
 - A. A case study will be presented which will allow participants to
 1. identify key work environment characteristics that promote power
 2. devise strategies to add missing power elements to a work environment
 3. demonstrate how power elements can be used to promote certification
 - B. Discussion
- VI. Conclusions

BIBLIOGRAPHY/WEBLIOGRAPHY

- Aiken LH, Clarke SP, Sloane DM, Sochalski J, Silber JH. Hospital nurse staffing and patient mortality, nurse burnout, and job dissatisfaction. *JAMA*. 2002;288:1987-1993.
- Irvine DM, Sidani S, McGillis Hall L. Linking outcomes to nurses' roles in health care. *Nurs Econ*. 1998;16:58-64. Available from: <http://www.cinahl.com/cgi-bin/refsvc?jid=374&accno=1998042672>.
- Kirkman BL, Rosen B. Beyond self-management: Antecedents and consequences of team empowerment. *Academy of Management Journal*. 1999;42:58-74.
- Laschinger HKS, Almost J, Tuer-Hodes D. Workplace empowerment and magnet hospital characteristics. *JONA*. 2003;33:410-422.
- Laschinger HKS, Shamian J, Thomson D. Impact of magnet hospital characteristics on nurses' perceptions of trust, burnout, quality of care, and work satisfaction. *Nursing Economic\$*. 2001;19:209-219.
- Laschinger HKS, Purdy N, Cho J, Almost J. Antecedents and consequences of nurse managers' perceptions of organizational support. *Nursing Economic\$*. 2006;24:20-29.
- Manojlovich M. Predictors of professional nursing practice behaviors in hospital settings. *Nursing Research*. 2005a;54:41-47.
- Spreitzer GM. Psychological empowerment in the workplace: Dimension, measurement, and validation. *The Academy of Management Journal*. 1995;38:1442-1465.

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Into Thin Air: High-Altitude Illness

Susan Dukes
Cindy Wright
Sarah Creason

Level: Beginner

CONTENT DESCRIPTION

High-Altitude Illness can strike as low as 5,000 ft. Even though early signs of high-altitude illness are mild and usually self-limiting, the more severe forms can be fatal. Millions of people each year are exposed to the potential adverse consequences of high altitude. This presentation will present high-altitude illness in its various forms to include high altitude cerebral edema and high altitude pulmonary edema. It will also address who is most susceptible looking at personal characteristics, geographic locations, and various assessment tools. Signs and symptoms and pathophysiology will be centered around hypoxia and principles of edema. Evidence based practice strategies will be examined in regards to management and prevention of high-altitude illness.

LEARNING OUTCOMES

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

1. Recognize what high-altitude illness is in its various forms.
2. Identify who is at risk for developing high-altitude illness as well as its signs and symptoms.
3. Discuss the treatment and prevention strategies for high-altitude illness.

SUMMARY OF KEY POINTS

- I. High-Altitude Illness
 - A. What is it?
 - B. What causes it?
 - C. What are the risk factors?
 - D. How can it be prevented?
- II. Acute Mountain Sickness
 - A. Pathophysiology
 - B. Diagnosis
 - C. Treatment

III. High-Altitude Cerebral Edema

- A. Pathophysiology
- B. Diagnosis
- C. Treatment

IV. High-Altitude Pulmonary Edema

- A. Pathophysiology
- B. Diagnosis
- C. Treatment

V. Implications for Oxygen Therapy at High Altitudes

BIBLIOGRAPHY/WEBLIOGRAPHY

- Barry, P.W. & Pollard, A.J. (2003). Altitude illness. *BMJ*. 326;915-919.
- Dennie, M.L. & Bayley, E.W. (2002). Into thinner air: preparing for changes in altitude may save your life. *AJN*. 102 Supplement;8-12.
- Gabry, A.L., Ledous, X., Mozziconacci, M. & Martin, C. (2003). High-altitude pulmonary edema at moderate altitude (<2,400 m; 7,870 feet): a series of 52 patients. *Chest*. 123;49-53.
- Gallagher, S.A. & Hackett, P.H. (2004). High-altitude illness. *Emerg Med Clin N Am*. 22;329-355.
- Grisson, C.K., Weaver, L.K., Clemmer, T.P. & Morris, A.H. (2006). Theoretical advantages of oxygen treatment for combat casualties during medical evacuation at high altitude. *The Journal of Trauma Injury, Infection and Critical Care*. 61(2);461-467.
- Honigman, B., Theis, M.K., Koziol-McLain, J., Roach, R., Yip, R., Houston, C. & Morre, L.G. (1993). Acute mountain sickness in a general tourist population at moderate altitudes. *Annals of Internal Medicine*. 118(8);587-592.
- Rupert, J.L. & Koehle, M.S. (2006). Evidence for a genetic basis for altitude-related illness. *High Altitude Medicine & Biology*. 7(2);150-167.
- West, J.B. (2004). The physiologic basis of high-altitude diseases. *Annals of Internal Medicine*. 141;789-800.

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Intraabdominal Hypertension: The Silent Killer

Timothy R. Wolfe

Level: Intermediate

CONTENT DESCRIPTION

The underlying pathophysiology of intraabdominal hypertension is an inflammatory induced capillary leak, which causes bowel and mesenteric edema, leading to tissue expansion within a fixed compartment (the abdomen). The result is increased intraabdominal pressure, a silent process that is difficult to detect unless specifically measured, yet may lead to multiple organ dysfunction and failure if not treated. This syndrome occurs in a substantial percentage of critically ill patients, ranging from severe sepsis to major trauma.

The presentation begins with several case studies followed by a detailed discussion of the impact IAH has on each of the bodies major organ systems. A concise medical literature review then highlights the leading recent articles on the topic. The end of the course will provide an evidence-based algorithm for monitoring and treating intra-abdominal hypertension in an effort to help critical care practitioners improve these patients outcomes. Finally, new ideas for bedside research opportunities will be presented and the discussion will be opened to any and all questions. The lecture is appropriate for all critical care provider levels, though some basic understanding of physiology is necessary to fully appreciate the lecture.

LEARNING OUTCOMES

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

1. Recognize that intra-abdominal hypertension occurs in all ICU populations, especially those with systemic inflammatory response syndrome.
2. Understand the pathophysiology effects of intra-abdominal hypertension (IAH) on all of the body's major organs.
3. Recognize that early detection and intervention for intra-abdominal hypertension before the abdominal compartment syndrome develops results in reduced ICU length of stay, improved outcomes and lower mortality.

SUMMARY OF KEY POINTS

Intraabdominal hypertension is very common in critically ill patients and is just as prevalent in medical patients as in surgical and trauma patients. It is a silent process – it cannot be detected without an established method of measurement and monitoring, yet it causes substantial organ ischemia and correlates with patient

mortality. Waiting for the clinical manifestations of intra-abdominal hypertension to develop (i.e. the abdominal compartment syndrome) leads to delayed diagnosis, loss of opportunity for early intervention, increased organ failure, increased mortality, and transition from an urgent medical process to an emergent surgical process. Critical care providers need to recognize this transition and begin monitoring all high risk patients so they can establish a trend in the intra-abdominal pressure, allowing them to intervene early and appropriately while the syndrome is still amenable to medical therapy. Waiting for abdominal compartment syndrome to manifest itself before an intra-abdominal pressure is checked to confirm the clinical diagnosis should no longer be acceptable critical care practice. Instead, the goal should be to prevent abdominal compartment syndrome by early monitoring, early medical interventions and early surgical decompression if needed.

BIBLIOGRAPHY/WEBLIOGRAPHY

- Cheatham, MI, Malbrain, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med* 2007 (Epub ahead of print).
- Ejike JC, Humbert S, Bahjri K, Mathur M. Outcomes of children with abdominal compartment syndrome. *Acta Clinica Belgica* 2007; 62-Supplement 1:141-148.
- Kimball EJ. Intra-abdominal hypertension and the abdominal compartment syndrome: The "ARDS" of the gut. *International Journal of Critical Care* 2006:31-39.
- Oda S, Hirasawa H, Shiga H, et al. Management of Intra-abdominal Hypertension in Patients With Severe Acute Pancreatitis With Continuous Hemodiafiltration Using a Polymethyl Methacrylate Membrane Hemofilter. *Ther Apher Dial* 2005; 9:355-61.
- Sun, Zx, Huang, Hr, Zhou. Indwelling catheter and conservative measures in the treatment of abdominal compartment syndrome in fulminant acute pancreatitis. *World J Gastroenterol* 2006; 12:5068-70.
- Wolfe TR, Gallagher J. Intra-abdominal hypertension: Pitfalls, prevalence and treatment options. *AACN News* 2006.
- Educational web site: www.abdominal-compartment-syndrome.org
- World society of abdominal compartment syndrome: <http://www.wsacs.org/>

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Intraosseous (IO) Devices for Adults: A New Life Line

Michael W. Day

Level: Intermediate

CONTENT DESCRIPTION

Emergency vascular access is a continued issue for most critical care nurses, especially with the new American Heart Association (AHA) guidelines for Advanced Cardiac Life Support (ACLS). Intraosseous (IO) devices in the adult population are relatively new, but understanding the various options available and the safe and correct use of IOs will assist the nurse in providing an emergency life line to adult patients in an emergency situation. The session will begin with a review of the new ACLS guidelines as they pertain to vascular access and delivery of medications. The session will then continue with brief history of IO devices, incorporating the anatomy of bony vascular systems. The session will then describe the various IO devices (FAST 1, EZ-IO, Bone Injection Gun [BIG]) currently approved by the United States Federal Drug Administration. Each device will be described in detail, including its indications, contraindications, insertion methods and sites, cautions and removal. By understanding the newly approved technology of IO devices for adults, the participant will understand their use and their utility in providing safe care to critically patient, who have no intravenous access. A case study will provide an opportunity for the participants to utilize their recently acquired knowledge.

LEARNING OUTCOMES

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

1. Discuss the general use of intraosseous (IO) devices in the adult population
2. Describe the insertion, removal, advantages and disadvantages of IO devices

SUMMARY OF KEY POINTS

- I. Introduction
- II. Intraosseous Devices
 - A. Identified as a possibility in
 - B. Early experiments included
 - C. Supplanted by simple and less invasive intravenous (IV) catheters
 - D. Re-established as a viable pediatric IV adjunct
 1. Pediatric Life Support (PALS)
 2. Advanced Cardiac Life Support (ACLS) 2005 Guidelines
 - a. "Safe and effective"
 - b. Useful for medications and fluids, including blood
 - c. Algorithms cite IV and IO access at the same time
 - d. De-emphasized use of endotracheal tube for drug delivery due to concerns regarding inconsistent absorption.

E. Unique features

1. Can be quickly inserted when no other access is available
 - a. Usually within 60 – 90 seconds
 - b. 80 – 95% success rate of placement
2. Depending upon the device, may be placed in several locations:
 - a. Sternum
 - b. Humerus
 - c. Tibia
3. Medullary canals of target bones
 - a. Very vascular
 - b. Non-collapsible
 - c. Rapidly enters the central circulation
 - d. Flow rates of up to 125 ml/min, under pressure
 - e. Very stable, once placed
 - f. Can be left in place for up to 24 hours or until more conventional IV access is obtained.
 - g. Placements do NOT interfere with other emergency procedures
 - (1) CPR
 - (2) Intubation
 - (3) Cricothyroidotomy
4. They look like something out of a medieval torture chamber!!!!
 - a. Not something nurses have typically done in the past
 - b. You will appreciate these devices if you have EVER had the experience of not being able to get IV access in an emergency

F. Institution requirements to be met BEFORE clinical use started

1. Institution approval – Most commonly through Pharmacy & Therapeutics Committee
2. Appropriate policies and procedures
 - a. When is the device appropriate
 - b. How will the device be inserted
 - c. Who will insert the device
 - d. Quality Assessment (QA)
3. Education and training
 - a. Initial
 - b. On-going

III. F.A.S.T. 1

A. Development

1. "First Access for Shock and Trauma" = F.A.S.T.1™
2. Developed by Pyng Medical Corporation, Vancouver, British Columbia, Canada

3. How it works
 - a. Cylindrical delivery device that fits in the palm of the hand
 - b. Delivery device has multiple “prongs” around recessed metal tipped, infusion tube
 - c. Delivery device placed over manubrium and pressure is exerted
 - d. When appropriate tension is achieved, a metal tipped, infusion tube is delivered into the bone
 - e. As the delivery device is removed, the infusion tube remains embedded
 - f. “Why the manubrium?”
 - (1) Universal depth in most adults
 - (2) Rapidly empties directly into central circulation
 - (3) Out of the way of other life saving procedures (CPR, intubation)

B. Insertion

1. Identify the sternal notch
2. Attach the target/strain relief patch so that the notch on the patch matches the patient’s sternal notch
3. Remove the delivery device’s protective cover and place device tip directly over the target hole on the patch
4. Insure that the device is PERPENDICULAR to the manubrium, not the patient’s body
5. Push down on the device until the release of the metal tipped, infusion tube is heard and felt
 - a. Considerable force may be necessary to trigger the deployment of the device
 - b. A two-handed grip may be necessary for those with lesser upper body strength
6. Remove the delivery device, exposing the embedded infusion tube
7. Attach the exposed infusion tube to the male connector of the patch tubing
8. Confirm placement by attaching a syringe to the female end of the patch tubing and aspirating bone marrow
9. Remove the syringe and attach purged IV tubing to the female end of the patch tubing and begin administering fluid
10. Apply the protective dome to the Velcro of the patch
11. Attach the enclosed UNOPENED remover to where it will be easily seen
 - a. On the patient
 - b. On the patient’s chart
12. If the IV fluid does NOT flow under pressure, or leakage of fluid occurs, remove the F.A.S.T.1™
 - a. A second attempt may be made using the same anatomical landmarks, but with a NEW F.A.S.T.1™
 - b. If the second F.A.S.T.1™ attempt is also unsuccessful, remove the device and attempt alternative IV access

C. Removal

1. Remove the protective dome and disconnect the male connector from the infusion tube
2. Using aseptic technique, open the remover package and remove the cover from its threaded tip
3. Hold the infusion tube perpendicular to the manubrium (not the patient’s body)
4. Insert the remover, turn the remover clockwise until the threads engage the stainless steel tip of the infusion tube
5. Continue turning the remover clockwise until it stops turning, indicating that it is locked on to the threads
 - a. If the remover does not engage the threads, withdraw the remover
 - b. Make a second attempt at threading the remover
 - c. If the remover CANNOT be threaded, the F.A.S.T.1™ will have to be removed by a minor surgical procedure
6. Pull the remover straight up, perpendicular to the manubrium, extracting the Infusion Tube
7. Remove the target/strain relief patch and apply a pressure, with a dry, sterile dressing

D. Advantages

1. Small package & device
2. Easily accessed insertion site that is the same for each patient
3. Quickly inserted

E. Disadvantages

1. May be less effective, due to altered circulation, in patients with:
 - a. Fractured sternum
 - b. Previous sternotomy
2. Use of device in patients with extremely small body size that is clearly “outside normal body size” may harm the patient and the risks and benefits must be weighed
3. NOT recommended when:
 - a. Tissue damage directly over the manubrium would prevent adherence of the target/strain relief patch
 - b. Condition that may affect the density of the manubrium
 - (1) Severe osteoporosis
 - (2) Multiple myeloma

IV. Bone Injection Gun (B.I.G.™)

A. Development

1. Developed by Dr. Marc Waisman, Waismed LTD in Israel in the early 1990s
2. In use in 18 countries worldwide
3. How it works
 - a. Resembles a short “magic marker” with a colored cap and “shoulders”
 - b. Colors designate use
 - (1) Blue = Adult
 - (2) Red = Pediatrics
 - (3) Green = Veterinarian use only

- c. A loaded tension spring injects both a both a trocar and needle
 - d. The trocar is removed and the needle remains in place
 - e. Approved by the FDA for insertion in:
 - (1) Proximal tibia
 - (2) Humeral head (as an alternative site)
- B. Insertion**
1. Identify the appropriate insertion site
 - a. Proximal tibia
 - (1) Identify the tibial tuberosity
 - (2) Move 1 – 2 cm medially
 - (3) Move 1 cm proximally, toward the head
 - b. Humeral head
 - (1) Used as a alternative site, if neither tibia is available for use
 - (2) Lay the patient’s arm across their abdomen with the hand at the umbilicus
 - (3) Identify the distal end of the clavicle
 - (4) Move two finger widths toward the elbow to find the insertion site
 - (5) In some patients (very obese or muscular), the insertion site is one finger width TOWARD the body’s midline
 2. Cleanse the insertion site
 3. Place the barrel of the appropriate B.I.G.TM against the insertion site so that it is perpendicular to the tibia and NOT TILTED UP TOWARDS THE KNEE
 4. Holding the B.I.G.TM in position, use the other hand to squeeze the two sides of the red safety latch together and remove it from the device
 5. DO NOT DISCARD THE RED SAFETY LATCH, AS IT HAS TWO IMPORTANT USES!!!
 - a. It may be needed to assist in removing the trocar
 - b. It will be used to secure the needle after it is inserted
 - c. It will be needed in removing the needle
 6. While maintaining position on the insertion site, grasp the “shoulders” of B.I.G.TM with the 1st and 2nd fingers of your other hand
 7. Inject the trocar and needle by pressing your palm firmly into the rear part of the B.I.G.TM housing
 8. Slightly twist the housing to remove it from the trocar and needle
 9. Stabilize the needle and twist and withdraw the trocar from the needle
 10. If the trocar does not easily release from the needle, grasp the hub of the trocar with the safety latch and twist and withdraw the trocar from the needle
 11. Attach a syringe to the needle hub to prevent air infiltration
 12. Confirm placement:
 - a. Aspirate a small amount of bone marrow (thick serosanguinous fluid)
 - b. Inject a small amount of IV fluid, watching for extravasation
 13. Attach a primed infusion set to the needle and begin administration
 - a. In the conscious patient, an infusion of lidocaine is recommended before administering IV fluids
 - b. Optimal IV fluid flow is best achieved by using a pressure device on the IV bag, up to 300 mm/Hg
 - c. Pressure limits on IV pumps limit their effectiveness with IO devices
 14. Slide the red safety latch around the inserted needle and tape into place.
 15. Because of the needle’s size, it may be used for bone marrow aspiration or blood sampling
- C. Removal**
1. Remove the tape and the red safety latch from around the needle
 2. Grasp the hub of the needle and withdraw while rotating it
 3. If needed, the red safety latch may be used to help grip the hub of the needle
 4. Apply a dressing to the insertion site
 5. Dispose of the needle in the nearest sharps container
- D. Advantages**
1. Small package
 2. Lightweight
 3. Quickly inserted
- E. Disadvantages**
1. Different sizes for different populations
 2. Once placed, device may be overlooked during resuscitation efforts
 3. Requires good two hand coordination on the part of the operator
 4. NOT recommended in patients with:
 - a. Osgood-Schlatter’s disease
 - b. Fractures above or below the insertion site
 - c. Any known condition that compromises the circulation of the extremity in question
- V. EZ-IO®**
- A. Development**
1. Developed by Vidacare Corp, in conjunction the University of Texas Health Science Center – San Antonio
 2. How it works
 - a. Inserted with use of a power driver
 - (1) Hand held
 - (2) Lithium powered
 - (3) Reusable
 - b. Needle set, with stylet and needle
 - c. Two different needle sets, based on patient’s weight
 - (1) AD for patients weighing 40 kg or more
 - (2) PD for patients weighing 3 to 39 kg
 - d. Needle set inserted into power driver
 - e. Power driver inserts needle into bone

- f. Stylet removed and needle remains in place
 - g. Approved by the FDA for insertion in:
 - (1) Proximal tibia
 - (2) Distal tibia
 - (3) Humeral head (as an alternative site)
- B. Insertion**
1. Identify the appropriate insertion sites
 - a. Proximal tibia
 - (1) Identify the tibial tuberosity
 - (2) Move 1 – 2 cm medially
 - (3) Move 1 cm proximally, toward the head
 - b. Distal tibia
 - (1) Identify the medial malleolus
 - (2) Move two finger widths up the leg
 - (3) Positioned midline on the medial aspect of the leg
 - c. Humeral head
 - (1) Used as an alternative site, if neither tibia is available for use
 - (2) Lay the patient's arm across their abdomen with the hand at the umbilicus
 - (3) Preferred method
 - (a) Palpate the mid-shaft of the humerus
 - (b) Palpate up the arm toward the humeral head until the greater tubercle is located
 - (c) Move one finger width lateral
 - (4) Alternative method
 - (a) Identify the distal end of the clavicle
 - (b) Move two finger widths toward the elbow to find insertion site
 2. Cleanse the insertion site
 3. Assemble the following equipment:
 - a. Appropriate size needle set
 - b. Power driver
 - c. 10 ml syringe, filled with normal saline and attached to a flushed IV extension set
 4. Insert the needle set into the power driver and remove the safety cap
 5. Stabilize the limb from behind and position the power driver so that the needle is at a 90° angle to the limb
 6. Determine if the 5 mm line on the needle is visible
 - a. If it is NOT visible, there is excessive tissue at the site and it should NOT be used for insertion
 - b. If it is visible, the site is useable for insertion
 7. Apply steady, firm pressure and activate the power driver, stopping when:
 - a. The hub contacts the skin
 - b. A sudden decrease of resistance is noted
 8. Hold the needle hub and gently pull the power driver straight up
 9. Continue to hold the needle hub and rotate the stylet counter clockwise to remove it from the needle
 10. Confirm correct placement by:
 - a. Observing that the needle is firmly seated at a 90° angle to the limb
 - b. Observing a small drop of blood at the tip of the stylet as it is withdrawn
 - c. Aspirating a small amount of bone marrow
 - d. Observing a free flow of fluids without extravasation
 11. Attach the pre-flushed IV extension set and flush with 10 mls of normal saline
 12. In a conscious patient, consider instilling lidocaine prior to starting fluid infusion
 13. Attach a primed infusion set to the needle and begin administration
 - a. In the conscious patient, an infusion of lidocaine is recommended before administering IV fluids
 - b. Optimal IV fluid flow is best achieved by using a pressure device on the IV bag, up to 300 mm/Hg
 - c. Pressure limits on IV pumps limit their effectiveness with IO devices
 14. The needle may be stabilized with a dressing
- C. Removal**
1. Remove the IV tubing from the needle
 2. Attach a 5 or 10 ml syringe by rotating it clockwise onto the needle hub
 3. Stabilize the limb
 4. Begin gently pulling the syringe, while at the same time rotating it clockwise
 5. Do NOT rock the needle back and forth
 6. If the needle hub separates from the needle, use a large needle forceps to grasp and remove the needle as described above
 7. Dress the site with a sterile bandage
- D. Advantages**
1. Single device for all patients > 3 kg
 2. Multiple sites for insertion
- E. Disadvantages**
1. Difficulty assessing needle depth (< or > than 5 mm) in low light situation
 2. Once placed, device may be overlooked during resuscitation efforts
 3. Requires good two hand coordination on the part of the operator
 4. NOT recommended in patients with:
 - a. Excessive tissue or inability to locate landmarks
 - b. Fractures of the targeted bone
 - c. Previous orthopedic procedures near insertion site
 - d. Infection at insertion site
- VI. Case Study**
- VII. Summary**

BIBLIOGRAPHY/WEBLIOGRAPHY

- American Heart Association. (2005). 2005 Emergency Cardiovascular Care, Author: Dallas, TX.
- Porth, C.M. (2005). Pathophysiology: Concepts of Altered Health States (7th ed.), Lippincott, Williams & Wilkins: Philadelphia, PA.
- F.A.S.T. 1 www.pyng.com, accessed 04 November 2007
- B.I.G. www.waismed.com, accessed 11 November 2007
- EZ-IO Training Manual www.vidacare.com, accessed 14 November 2007

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Introduction to Crucial Conversations and Links to a HWE

Emily Moss

Level: Beginner

CONTENT DESCRIPTION

Problem-plagued organizations often share the same root cause: perplexed employees who aren't quite sure how to bring up touchy, controversial, and complex issues, and resolve them. When three key forces show up—strong emotions, differing opinions, or high stakes—business results and quality of patient care suffer.

Crucial Conversations teaches individuals and teams how to willingly and effectively surface and discuss ideas in a way that leads to virtually everyone buying into the decisions—creating broad alignment, maximizing synergy, and ensuring commitment to the best ideas. You will learn to share information safely, get ideas and feelings out in the open, and maintain high levels of respect—all without causing resistance or resentment.

LEARNING OUTCOMES

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

1. Use their skills to step up to controversial and heated issues
2. Professionally discuss issues in a way that makes it safe for everyone to speak
3. Create an environment where people share their best ideas, make wise choices, and then act on their choices with conviction

Is Chlorhexidine (CHG) the Key in Infection Prevention?

Mary Lou Sole
Sponsored by Sage Products, Inc.

Level: Intermediate

CONTENT DESCRIPTION

Infection prevention is an essential strategy to improve patient safety, especially in the critical care patient population. Critically ill patients are at higher risk for ventilator associated pneumonia, and infections in the bloodstream, urinary tract, and surgical site. The incidence of Infection with drug-resistant organisms is also rising. Chlorhexidine Gluconate (CHG) is being increasingly studied and used in successful infection prevention programs. This session reviews the evidence related to use of CHG to prevent these common infections.

LEARNING OUTCOMES

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

1. Discuss the etiology of common infections in the critically ill patient, including the role of biofilms.
2. Describe the research related to the effectiveness of CHG in infection prevention.
3. Identify best practice strategies for preventing common infections.

SUMMARY OF KEY POINTS

- I. Infection Prevention: Important for all
- II. Etiology of infections in critically ill patients
 - A. Ventilator-associated pneumonia (VAP)
 - B. Catheter-related bloodstream infections (CR-BSI)
 - C. Surgical site infections (SSI)
 - D. Urinary tract infections (UTI)
- III. Common organisms for each infection
- IV. Increasing development of infections with multidrug resistant organisms
- V. Role of biofilms in infection
 - A. Thin, usually resistant layer of microorganisms that form and coat on various surfaces
 - B. Devices/surfaces implicated in wide variety of infections
 1. Endotracheal tube
 2. Teeth and gums
 3. Central lines
 4. Skin
 5. Indwelling catheters
- VI. Is CHG the answer to preventing infection?
 - A. How CHG works
 - B. Role in common infections
 - C. Effectiveness on biofilms
 - D. Effectiveness on multidrug resistant organisms
 - E. Summary of latest evidence related to CHG in infection prevention

VIII. Delivery of CHG to prevent infection (includes method and %CHG concentration)

- A. Oral care best practices
- B. Central line insertion
- C. Central line dressings
- D. Bathing to reduce urinary tract infection, surgical site infection
- E. Carryover effects
 1. Reduction in CR-BSI after bathing
 2. Reduction in drug-resistant organisms, such as VRE after bathing
 3. Reduction in surgical site infection after nasal and oral application
- F. Potential other uses of CHG
 1. Hand hygiene of healthcare workers
 2. Disinfection of equipment

IX. Options to CHG?

- A. Oral care—peroxide
 - B. Devices—silver-coating
- X. Issues related to CHG
- A. Side effects
 1. Staining of oral surfaces: teeth, restoration, tongue (oral)
 2. Alterations in taste (oral)
 3. Skin irritation (topical)
 4. Hypersensitivity with gels and impregnated devices
 - B. Resistance
 - C. Concentration of products
 - D. Delivery methods
 - E. Pediatrics

XI. Best practice strategies

- A. Protocols and checklists—include CHG in “bundles” of practices
- B. Equipment availability
 1. Readily available
 2. Kits
 3. Cost-benefit analysis
- C. Ongoing monitoring, reporting, and feedback
- D. Champions

BIBLIOGRAPHY/WEBLIOGRAPHY

- Bleasdale SC, et al., Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. *Arch Intern Med*, 2007. 167(19):2073-9.

Chaiyakunapruk N, et al., Chlorhexidine compared with povidone-iodine solution for vascular catheter-site care: a meta-analysis. *Ann Intern Med*, 2002. 136(11):792-801.

Costerton JW, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science*, 1999. 284(5418):1318-22.

Gastmeier P, Geffers C. Prevention of catheter-related bloodstream infections: analysis of studies published between 2002 and 2005. *J Hosp Infect*, 2006. 64(4):326-35.

Gastmeier P, Zuschneid I, Geffers C. Antimicrobially impregnated catheters: An overview of randomized controlled trials. *J Vasc Access*, 2003. 4(3):102-10.

Ho KM, Litton E. Use of chlorhexidine-impregnated dressing to prevent vascular and epidural catheter colonization and infection: a meta-analysis. *J Antimicrob Chemother*, 2006. 58(2):281-7.

Kola A, Gastmeier P. Efficacy of oral chlorhexidine in preventing lower respiratory tract infections. Meta-analysis of randomized controlled trials. *J Hosp Infect*, 2007. 66(3):207-217.

Pineda LA, Saliba RG, El Solh AA. Effect of oral decontamination with chlorhexidine on the incidence of nosocomial pneumonia: a meta-analysis. *Crit Care*, 2006. 10(1):R35.

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It's about Flow: Surgery in Adults with Congenital Heart Disease

Cheryl Rader
Nancy Richards

Level: Intermediate

CONTENT DESCRIPTION

Due to the advances in the diagnosis and treatment of congenital heart disease (CHD) over the past 30 years, the number of patients reaching adulthood is increasing steadily. Currently more adults than children are living with CHD and this population is estimated to be growing at 5% per year. The purpose of this session is to review congenital heart anomalies, describe past and present surgical correction and discuss the challenges encountered when these patients present as adults for subsequent cardiac surgery. Case studies will be utilized to highlight the presentation, surgical procedure, challenges and outcomes of this special population. Content will focus on the unique post-operative considerations and include an overview of life-style implications.

LEARNING OUTCOMES

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

1. Describe common congenital heart anomalies along with past and present surgical treatment options.
2. Discuss unique challenges that face the surgical team when caring for the adult with congestive heart disease.
3. Describe lifestyle implications for adults with congenital heart disease.

SUMMARY OF KEY POINTS

- I. Introduction
- II. Review of Fetal Cardiac Circulation
- III. Common Congenital Anomalies
 - A. Acyanotic
 1. Atrial Septal Defects
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 2. Ventricular Septal Defects
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 3. Patent Ductus Arteriosus
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges

4. Bicuspid Aortic Valve
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 - d. Case Study
5. Coarctation of the Aorta
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 - d. Case Study
- B. Cyanotic
 1. Tetralogy of Fallot
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 - d. Case Study
 2. Ebstein's Anomaly
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 3. Transposition of the Great Arteries
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
 4. Univentricle
 - a. Presentation
 - b. Surgical treatment/challenges
 - c. Post-operative care and challenges
- IV. Lifestyle Implications
 - A. Psychosocial
 1. Neurocognitive
 2. Social
 3. Psychological
 4. Quality of Life
 - a. Employment
 - b. Health Insurance
 - c. Contraception, Pregnancy and Childbirth
 - B. Transition of Care: Child to Adult

It's Not Just a Ventilator Anymore... More New Ventilator Modes

Nancy J. Ames

Level: Intermediate

CONTENT DESCRIPTION

A large percentage of critical care patients admitted to ICUs have acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) and require mechanical ventilation. Eighty percent of the patients that are ventilated can be managed with standard modes that are familiar to most critical care nurses. But twenty percent of the patients with ARDS require innovative approaches utilizing open lung strategies to survive their lung disease. This one-hour session will assist the critical care nurse in understanding these complex modes and strategies. The new modes that are being used in critical care units, such as, airway pressure release ventilation (APRV), and the combination or dual modes using targeted volumes with pressure ventilation, such as, pressure-regulated volume control (PRVC) will be introduced and advantages and disadvantages reviewed. High frequency oscillatory ventilation (HFOV) that had been used for years in the pediatric critical care population and is now being used in adults. Mechanics of this new mode and its open lung approach will be reviewed. This lecture will introduce critical care nurses, who are familiar with managing patients on ventilators, how to improve this management by providing an understanding of how these unique modes and strategies relate to the pathogenesis of ARDS. Through a case study presentation, the critical care nurse will understand how important the appropriate ventilator strategy is in preventing further lung damage. After attending this Concurrent Session the critical care nurse will be able to better understand the importance of customizing the ventilator and using the newer modes to improve patient outcomes.

LEARNING OUTCOMES

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

1. Compare and contrast different ventilator modes commonly utilized in critical care including strengths and weaknesses.
2. Identify three new modes of mechanical ventilation that are becoming popular in critical care units.
3. Outline current published research in using mechanical ventilation.

SUMMARY OF KEY POINTS

I. Case study

A 21-year-old male college student was admitted to the MICU with a diagnosis of respiratory failure. One week ago, he had developed upper respiratory symptoms which progressed despite over-the-counter remedies. His chest x-ray taken in the emergency department showed left lower lobe consolidation

and a question of some consolidation on the right. He was admitted with the presumed diagnosis of community-acquired pneumonia.

He presented to the MICU on a non-rebreathing mask. His respiratory rate was 40 and labored. Pulse oximetry displayed 90% with a good waveform. His cardiac rhythm was sinus tachycardia at 130. He had a fever of 38.2° C. Pan cultures had been obtained in the Emergency Department. He was hypotensive with a blood pressure of 80/50. His medical history was insignificant. ABGs on arrival to the MICU were: pH 7.18, PaO₂ 52, PaCO₂ 38, SaO₂ 90%, HCO₃ 21. He was intubated without difficulty and placed on a Servo 300 ventilator in the SIMV-VC mode. The settings were the following: FiO₂ 100%, VT 700, SIMV 10, PS 10 and PEEP 5. His static compliance was 46 cm of H₂O. The patient was over-breathing the ventilator at a rate of 15 and the pulse oximeter was recording 100%. Sedation and analgesia were provided. His heart rate remained greater than 140 beats/min and his B/P ranged from 110/80 after addition of a nor-epinephrine infusion at 10 mcg/minute.

The patient remained on volume ventilation until the next day with FiO₂ in the 70% range and saturations in the low 90s. The patient was placed on a rotational bed and frequent percussion and vibration were ordered.

Initial culture results of sputum were positive for methicillin resistant staphylococcus aureus (MRSA). In an attempt to decrease the FiO₂, the PEEP was increased to 10 cm H₂O. The FiO₂ was decreased to 50%; the mode was changed to PRVC (pressure-regulated volume control) with a target volume of 750 cc and a rate of 15. ABGs on these settings were adequate and the patient was maintained on these settings for a few hours. Some short periods of desaturation were treated with attempts to increase the PEEP to 12 cm H₂O and a change in the I: E ratio to 1:1. The peak pressures remained in the high 30s (cm H₂O) and a plan was made to discontinue the versed and fentanyl drips and further wean the FiO₂.

The next morning the patient experienced frequent episodes of desaturation into the low 70s (%) that required manual ventilation with a bag-valve-mask system to increase the FiO₂. Chest x-ray demonstrated increased consolidation with patchy bilateral infiltrates. The FiO₂ was increased to 100%. In order to deliver the tidal volume of 750 cc in the PRVC mode, the pressure had to be increased to 45 cm H₂O. The FiO₂ could not be weaned below

100%. The PEEP was increased to 15 cm H₂O; the mode was changed to pressure control (PC) of 45 cm of H₂O with VT ranging from the 600-705 ml. The patient was neuromuscular blocked. The patient was ventilated in the pressure control mode with increasing peak pressures in the 50s (cms H₂O) and inability to wean the FiO₂.

How does the ventilator mode change affect the nursing management of the patient? What do the different modes mean as far as delivery of the gases?

II. Pathogenesis of ARDS and Lung Protective Ventilation Strategies or How the Ventilator Can Help or Hurt

A. Prevention of Ventilator induced lung injury (VILI) or Ventilator associated lung injury (VALI) with the open lung approach

B. Heterogeneous Disease- 3 different areas in the lungs

C. Low Tidal Volumes – ARDS Network Study

1. 6ml/kg vs 12ml/kg
2. Peak airway pressure less than 35 cm of H₂O
3. Plateau pressures

D. PEEP and recruitment maneuvers

III. Modes of ventilation

A. Assist-control (AC) or assisted mandatory ventilation (AMV)

B. Synchronized intermittent mandatory ventilation (SIMV)

1. Characteristics
2. Strengths
 - a. Security of preset mandatory ventilation
 - b. Patient uses respiratory muscles -can be used as a weaning mode
 - c. Decreased alkalosis seen in A/C mode
3. Limitations
 - a. Inadequate flow or no PS set can cause increased work of breathing
 - b. If rate is set too low patient experiences fatigue and tachypnea

C. Pressure support ventilation (PSV) or pressure support (PS)

1. Characteristics: PS is a patient-triggered mode. When the patient's inspiratory pressure meets the assist-sensitivity threshold, a set level of inspiratory pressure is delivered.
2. Strengths
 - a. Decreased work of breathing
 - b. Patient determines own minute ventilation; improved patient-ventilator synchrony
 - c. PSV does not control tidal volume; patients with unreliable ventilatory drive should have back-up mandatory rate because
3. Limitations

D. Not Really Modes!!

1. Continuous positive airway pressure (CPAP)
2. Positive-End -Expiratory Pressure (PEEP)
 - a. Treatment PEEP
 - b. Auto-PEEP or Intrinsic PEEP-Total PEEP = Set PEEP + auto-PEEP

c. Complications of PEEP

E. Pressure control ventilation (PCV)

1. Characteristics: No preset tidal volume (VT) is selected. The amount of pressure used to achieve a desired tidal volume is set together with a selected rate.
2. Strengths
 - a. Airway pressures are constant and in the setting of acute lung injury are minimized while maintaining mean airway pressures.
 - b. Decelerating flow pattern is used.
3. Limitations
 - a. Minute ventilation (VE) must be monitored. VT varies with changes in compliance
 - b. Auto-PEEP and barotrauma can occur.
 - c. Higher pressures may cause cardiovascular compromise an increase use of vasopressors
 - d. More sedation and analgesia might be required.

F. PCV with inverse I: E ratio (PCIRV)

1. Characteristics
2. Strengths
 - a. Noncompliant alveoli may be recruited with the longer inspiratory time
 - b. Peak airway pressures are minimized while mean airway pressures are maintained
3. Limitations
 - a. May increase the incidence of auto-PEEP
 - b. Because of uncomfortable mode- increased need for sedation and analgesia some patients may require neuromuscular blockade

G. Dual controlled Modes

1. PRVC (pressure-regulated volume control) - Offered on the Servo 300 ventilator
 - a. Characteristics
 - b. Strengths
 - c. Limitations
2. Volume Control Plus-Puritan Bennett 840
 - a. Characteristics
 - b. Strengths
 - c. Limitations
3. Volume support (VS)
 - a. Characteristics
 - b. Strengths
 - c. Weaknesses

H. All About Airway Pressure Release Ventilation (APRV)

1. Characteristics: sets a high level of CPAP and a release level which allows carbon dioxide to be cleared. It allows spontaneous breathing at all phases of the cycle. Two pressure levels are set as well as an inspiratory time and expiratory time.
2. Strengths
 - a. Prevents lung over-distention
 - b. Lower mean and peak airway pressures which prevent barotrauma and lung injury
 - c. Spontaneous breathing during all cycles and decrease use of sedation and paralytics-more comfortable mode?

- d. Less cardiac compromise and decrease use of vasopressors
- 3. Limitations
 - a. Learning curve required by clinical staff to utilize mode successfully and safely
 - b. Minute ventilation must be monitored
 - c. Not applicable in patients with severe airflow obstruction
- I. A Few Words about High Frequency Oscillatory Ventilation (HFOV)
 - 1. Characteristics: Higher mean airway pressure (Paw) is applied to achieve and maintain recruitment by use of a piston driven system. Delivers very small tidal volumes at extreme rates e.g. 180-420 breaths/min.
 - 2. Strengths
 - a. Uses open lung approach
 - b. Unique method of ventilation and oxygenation
 - 3. Limitations
 - a. One ventilator
 - b. Staff learning curve
- IV. Recent research and clinical implications
 - A. Badin, S. Gress, T., et al. Mechanical ventilation management by pulmonologists and surgeons in patients with adult respiratory distress syndrome. *Am J Med Sci.* 2007 Sep; 334(3):155-9.
 - 1. Clinical implications:
 - a. Compares ventilator management by two groups of physicians
 - b. 97 patients with ARDS

B. Malhorta, A. Low-tidal-volume ventilation in the acute respiratory distress syndrome. *N Engl J Med.* 2007 Sep 13;357(11):1113-20. Review.

1. Clinical implications:

- a. Review article for ARDS and low tidal volume

C. Chan, KP, Stewart, TE, Mehta, S. High-frequency oscillatory ventilation for adult patients with ARDS. *Chest.* 2007 Jun; 131(6):1907-16. Review

1. Clinical implications

- a. Reviews clinical implications of HFOV
- b. Clinical review

D. Myers, TR & MacIntyre, NR. Respiratory controversies in the critical care setting; Does airway pressure release ventilation offer important new advantages in mechanical ventilator support? *Respir Care* 2007 Apr;52 (4):452-8;

1. Clinical Implications

- 2. Review article for APRV

V. Summary

VI. References

BIBLIOGRAPHY/WEBLIOGRAPHY

- Burns, S. Working with respiratory waveforms: how to use bedside graphics. *AACN Clinical Issues.* 2003;14(2):133-144.
- Cairo, J.M. & Pilbeam, S. *Mosby's Respiratory Care Equipment*, 7th ed. St Louis; 2004.
- Pillbeam, SP. *Mechanical Ventilation: Physiology and clinical applications*, 3rd ed. St Louis: Mosby, 2000.
- Pierce, Lynelle. *Management of the Mechanically Ventilated Patient*, 2nd edition. Saunders, 2007.

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It's Not Just for Neuro Nurses: The Comprehensive Neuro Exam

Elizabeth Anness
Kelly Tirone

Level: Beginner

CONTENT DESCRIPTION

The purpose of this session is to enhance the critical care nurse's neuro exam including the much overlooked and under assessed cranial nerves. A complete comprehensive neuro exam may detect abnormalities, which along with further interventions will help to localize specific disease processes. This session will benefit all critical care nurses caring for patients with neurological illness or injury. Key concepts to be emphasized in this session will include the importance of frequent thorough neuro assessments, fundamental neuro anatomy noting specific regions and their associated dysfunctions, complete neuro exam including function and location of the cranial nerves.

LEARNING OUTCOMES

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

1. Describe the importance of early signs of neurologic deterioration.
2. Apply an understanding of neuro anatomy including specific regions and their role in pinpointing sites of dysfunction.
3. Demonstrate a complete neuro exam.

SUMMARY OF KEY POINTS

- I. Importance of the neurological assessment
 - A. Neurological injury occurs in any type of patient
 1. Cardiac arrest
 2. Traumatic insult
 3. Embolic or hemorrhagic stroke
 4. Common and uncommon neurologically injured patient
 - B. Integral and necessary component of overall assessment
 1. Baseline assessment
 2. Frequent assessment needed to monitor ever-evolving neurological exam
 - C. Time is brain!
 1. Signs of clinical deterioration can happen minutes to hours prior to sentinel event
 2. Identified changes may lead to further testing & treatment
- II. Overview of neurological anatomy
 - A. Cerebrum
 1. Frontal Lobe
 2. Temporal Lobe
 3. Parietal Lobe
 4. Occipital Lobe

- B. Brain Stem
 1. Midbrain
 2. Pons
 3. Medulla
 - C. Cerebellum
- III. Overview and Techniques of a complete neurological assessment
 - A. Assessment of the Conscious Patient
 1. LOC
 - a. Orientation
 - b. Memory
 - c. Attention Span
 - d. Language
 - (1) Speech
 - (2) Language deficit
 2. Pupil Evaluation
 - a. Size
 - b. Equality
 - c. Shape
 - d. Reaction to light
 3. Cranial Nerves Assessment
 4. Motor Assessment: (Frontal)
 - a. Ability to follow commands
 - b. Strength of extremities
 - (1) Upper and lower extremities
 - (2) Right and left sides
 - c. Pronator Drift
 5. Sensory Assessment: (Parietal)
 - a. Upper and lower extremities
 - b. Proprioception
 - c. Sensory deficit
 6. Coordination: (cerebellar)
 - a. Finger to nose
 - b. Rapid alternating movements
 - c. Heel to shin
 - B. Assessment of the Unconscious Patient
 1. Motor response and eye opening
 - a. Central pain
 - (1) Source
 - (a) Trapezius squeeze
 - (b) Supraorbital pressure
 - (c) Earlobe squeeze
 - (d) No longer use sternal rub!
 - (2) Response (not a test of strength)
 - (a) Localization; considered NORMAL response
 - (b) Flexion/Extension; considered

ABNORMAL response

- b. Peripheral pain
 - (1) Source
 - (a) Nail bed pressure
 - (b) Other peripheral stimuli
 - (2) Response (not a test of strength)
 - (a) Upper extremity
 - i. Withdrawal
 - ii. Flexion
 - iii. Extension
 - (b) Lower extremity
 - i. Withdrawal from pain
 - ii. Triple flexion response
- 2. Cranial Nerves
 - a. Assessment of cranial nerves III, V, VII, IX, X
 - b. No doll's eyes or ocular cephalic testing by RN
- 3. Pupil Evaluation
 - a. Size
 - b. Equality
 - c. Shape
 - d. Reaction to light
 - (1) Nystagmus
 - (2) Hippus

- IV. Case presentation of implementation of a neuro exam on a patient presenting with a neurological injury
 - A. Traumatic brain injured patient with evolving mid-line shift
 - B. Post-operative tumor debulking with cerebral hemorrhaging

V. Discussion and questions: 10 minutes

BIBLIOGRAPHY/WEBLIOGRAPHY

Rengachary, S., Ellenbogen, R., (2005) Clinical Evaluation of the nervous system. In Principles of Neurosurgery. Second Edition. Mosby, New York.

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