Shock

Trent Heather, BSN, RN, BA, Clinical Care Supervisor in the Surgical ICU at Hennepin County Medical Center.

Shock

- A state of inadequate perfusion relative to tissue demand
- Inadequate oxygen delivery relative to tissue demand
  - Systemic tissue perfusion is a product of CO and SVR

Cardiac Output

- A normal CO is 4-8 liters per minute
  \[ CO = SV \times HR \]
- Stroke volume = amount of blood ejected by the left ventricle with each contraction
  (A normal SV is 60-100ml)
- Heart rate = beats per minute
  (A normal HR is 60-100 bpm)

4 Types of Shock:

1. Cardiogenic
2. Hypovolemic
3. Distributive:  - Neurogenic
   - Anaphylactic
   - Sepsis
4. Obstructive

Shock is a result of inadequate:

- Oxygen supply
- Oxygen delivery (DO2)
- Oxygen utilization (VO2)

Shock

- Components needed for oxygen supply
  - Adequate ventilation (exchange of air between lungs and atmosphere so that O2 can be exchanged with CO2)
  - Adequate hemoglobin to carry oxygen to the tissue cells
Shock

- Components necessary for oxygen delivery
  - Adequate pump = cardiac output
  - Adequate volume = stroke volume

**DO2=Oxygen delivery (the result of cardiac output)**

Shock

- Components necessary for oxygen utilization
  - Adequate functional vascular bed
  - VO2 = represents “Oxygen utilization”

4 Factors effect SVO2 Balance

- Cardiac Output-(oxygen delivery)
- Hemoglobin-(oxygen delivery)
- SAO2(oxygen supply)
- VO2-(oxygen utilization)

Stages

- **Initial** - At this stage, shock is reversible
- **Compensatory** - Compensatory mechanisms kick in to return cells to preshock state
- **Progressive** - Compensatory efforts begin to fail and irreversible cellular damage occurs
- **Refractory** - Progressive end organ dysfunction becomes irreversible and unresponsive to therapeutic interventions

Types of Shock

- Cardiogenic – pump
- Hypovolemic – volume
- Distributive – vascular bed
  - Neurogenic
  - Anaphylactic
  - Septic

Shock

- Heart and brain
  - Increase metabolic rates
  - Decreased stores of energy substrate
  - Require perfusion pressure >60 to perfuse organs and prevent cell death
Neuroendocrine Response

- Baroreceptors and chemoreceptors
  - Norepi produces vaso and splenic constriction
  - Reduce vagal response
  - Vasopressin
    - Constriction
    - Renal tubules water reabsorption
  - Aldosterone
    - Reabsorption of Na

Sympathetic Activation

- Alpha receptors
  - Vasoconstriction
- Beta receptors
  - Dilation of circulation to brain and heart

Cellular Response

- Hypoperfusion
  - Decrease filtration
    - Ion pump dysfunction
  - Aerobic to anaerobic metabolism
  - Increase metabolites increase osmolarity
  - Reabsorption of fluid into intravascular bed
  - Increase interstitial fluid
  - Lactic Acidosis
  - Cell death

Cardiovascular Response

- Decrease stroke volume causes increase HR
  - CO = SV x HR
- Venoconstriction
  - 2/3 of volume is in the venous bed

Pulmonary Response

- Increase RR and depth of breathing

  In decompensation
  - Increase PVR reduces tidal volume, increases dead space- decrease gas exchange
  - Increase work of breathing
  - Increase demand on resp muscles
  - Lung injury ARDS

Renal Response

- Conserve water and Na
  - By release of ADH, aldosterone

  In decompensation
  - Tubular obstruction by cellular debris
  - Decrease blood flow
  - Toxic injury
Images have been removed from the PowerPoint slides in this handout due to copyright restrictions.

### Stages of Shock

- **Initial stage (preshock)**
  - Change from aerobic to anaerobic
  - Glycogen stores used early
  - Slowly build up lactic acid
  - No signs or symptoms

- **Compensatory Stage**
  - (Warm shock, compensated shock)
  - Neural
    - SNS
  - Hormonal
    - ADH
  - Chemical
    - CO2

- **Progressive**
  - Compensatory mechanisms fail
  - Cell death
  - Organ failure

- **Refractory**
  - Irreversible damage and death

### Multi-system Failure

- Cellular anoxia results from:
- Cellular depletion of ATP
- Energy debt
- Accumulation of anaerobic end-product metabolism-(waste) further impairs cells

### Metabolic Acidosis

- Metabolites override vascular tone
  - Hydrogen ions build up
  - Lactate
  - Cell swelling and leaking
Diagnosis

- Medical history
- Physical exam
- Laboratory evaluation
  - Na, K, Chloride, serum bicarb, creat & bun, coags, liver function, cardiac enzymes, ABG, lactate
- Pulmonary artery catheterization*

Shock - Compensatory Stage moving to Progressive Stage

- Assessment
  - BP
  - HR
  - Neuro
  - Renal
  - Skin
  - Lungs
  - Hemodynamic

Treatment of any Type of Shock

- Identify cause
  - Type of shock
- Oxygen
  - Hemoglobin
  - Saturation
- Support Blood Pressure
  - Volume
  - Inotropic agents
- Psychological
  - Patient
  - Family (all shock mortality = 35–60%)

Goal for Treatment

- Restore Oxygen Transport

True Emergency!

- Cardiogenic shock is the most difficult type of shock to treat and has one of the highest mortality rates of the different shock types.
- Goal is to save the patient’s life and treat the cause

Cardiogenic Shock

- Most common cause of death in hospitalized patients with MI
- Mortality Range 70%-80% in 70’s
  50%-60% in 90’s
  48% in 2004 NRMI database
- Occurrence 5 to 7%
  - 40% LV
  - RV, VSD, Pap muscle rupture, free wall rupture, hypovolemia

Braunwald 7th edition
### Cardiogenic Shock

**Pathophysiology**
- Decrease stroke volume
- Decrease cardiac output
- Increase heart rate
- Vasoconstriction
- Decrease urine output
- Pulmonary congestion

### Cardiogenic Shock - Intrinsic

**Cause**
- Severe ischemia or infarction
- Cardiomyopathy
- Valvular disease or dysfunction
- Low cardiac output syndrome
- Severe brady or tachy rhythms
- Free wall rupture

### Signs and Symptoms

- Low blood pressure and tachycardia
- Skin cool, clammy and possibly dusky; slow capillary refill
- Hemodynamic monitoring is usually instituted
- Lung sounds with crackles; patient short of breath or dyspneic
- Restlessness, anxiety, and possibly lethargy and confusion

### Cardiogenic Shock Diagnosis

- History – past cardiac disease?
  - Shock assessment
- EKG
- ECHO
- Enzymes-Troponin, CK-MB

### EKG

- T wave inversion
  - Ischemia
- ST segment elevation
  - Injury
- Q wave
  - Infarction

*Acute coronary syndrome

### Treating Cardiogenic Shock

- Requires an aggressive strategy
- Specific goals:
  - Identify and correct underlying cause
  - Improve tissue perfusion. *Especially important in the case of MI.*
Cardiogenic Shock Treatment

(Thrombolytic Agents)
- Aspirin, Heparin,
- GP 11b/111a inhibitors

(Hemodynamic monitoring)
- Assess volume
- Assess response to treatment

(Angioplasty/Surgery)
(Inotropes, Vasodilators, Ventricular assist devices)
(Sedatives, Analgesics, rest, oxygen)

Hypovolemic Shock

• Cause
  - Hemorrhagic
  - Non hemorrhagic
    • Diarrhea/vomiting-increase fluid output
    • Heat stroke-lack of H2O
    • Burn/ascites-fluid shift
    • "Third spacing"

• Pathophysiology
  - Decrease circulating volume
  - Cellular hypoxia
  - Cellular death and acidosis

Pathophysiologic Process

• Decreased circulating volume leads to decreased preload and stroke volume, decreased cardiac output, and finally hypotension and inadequate tissue perfusion

  • Begins when 15% or appx. 750 ml of intravascular volume has been lost

Classification of Hypovolemia

• 1. Mild <20%  Mild tachycardia
    No BP changes

• 2. Moderate 20% - 40%  Same plus:
    Increase HR >120
    Orthostatic changes
    Oliguria
    Tachypnea

• 3. Severe >40%  Same plus:
    Hemodynamic instability
    Multi-system failure

Treatment

• Best to prevent its occurrence by monitoring for signs of fluid loss and correcting before shock occurs

  • Treatment includes finding the cause and correcting it by replacing specific fluid lost.
    (**Place 2 large bore IV catheters.

  • Specifics of treatment:
    * Fluid and blood
    * Colloid fluids
    * Transfusion
Hypovolemic Shock

- Assessment
  - BP: low
  - HR: increase
  - Neuro: irritable, coma
  - Renal: decrease output
  - Skin: pale cool clammy
  - Lungs clear
  - Hemodynamic assessment
    - CVP, RA, PW: low
    - CO, CI: low
    - SVR: increase

Hypovolemic Shock

- Treatment replace what’s lost!
  - Volume
    - Blood
    - Fluids

Intravenous Fluid Replacement

- Crystalloids
  - Saline, ringers
    - Distribute freely
    - 2 – 6 times more required than estimated fluid loss

- Colloid
  - Albumin, Hespan
    - More expensive

- Not proven more effective

Electrolyte Imbalances

- Hyponatremia
  - Overcorrection can lead to paralysis and coma
  - Prolonged can lead to Neuro injury
  - 115 mEq/l and symptomatic = nonaggressive therapy
  - If symptoms or severe (< 115) 1 – 2 mEq/hr to 120 to 125
  - Lasix if fluids adequate

Electrolyte Imbalance

- Hypernatremia
  - Overcorrection can lead to cerebral edema
  - Free water to correct gradually
  - Change no faster than 1 – 2 mEq/hr to max 15 – 20 mEq in 24hr

Hypovolemic Shock - Trauma

- If trauma associated with hypovolemia
  - Tissue injury and inflammatory response
  - Increase fluid going to inflammation
  - Mal-distribution of blood flow
Treatment Hypovolemia - Trauma

- ABC’s
- Stasis
- Early stabilization of FX*
- Debridement of devitalized and contaminated tissue*
- Evacuation of Hematoma*

* Decrease inflammatory response

Goal for Treatment

- Restore Oxygen Transport

Case Study #2

- 20 year old male gun shot wound with uncontrolled bleeding. What are you going to do?
  - BP = 60/30
  - HR = 120
  - RR = 26 clear lungs
  - Neuro = unconscious
  - Skin = cold
  - Now what?

Uncontrolled Hemorrhage Houston Study
598 Adults Penetrating Injuries Bp<90

- Immediate fluid flushes
  - 62% survived
  - 30% complication
    - ARDS, ARF, pneumonia
    - Wound infection
- Delayed fluid
  - 70% survived
  - 23% complication
  - Hospitalization shorter

International resuscitation research center

Case Study #2

- Timing of fluid resuscitation
  - Early 2L bolus delays hemostasis
  - 2L Bolus at hemostasis trigger rebleeding
    - Vulnerable clot 0-34min

Lunch time!

Mapstone, J of Trama 03
Hirshberg J of Trama 06
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References


References


References


References

• Cardiogenic Shock retrieved from emedicine.medscape.com/article/152191-overview
• Critical Care Medicine Tutorials-The patient is hypotensive: is this due to hypovolemia retrieved from www.continumtutors.com/cct1/shock/clinshock3.htm
• Critical Care Medicine Tutorials-The patient is hypotensive is this pump failure retrieved from www.continumtutors.com/cct1/shock/clinshock4.htm
• Critical Care Medicine Tutorials-The patient is hypotensive is there Abnormal Vasodilation retrieved from www.continumtutors.com/cct1/shock/clinshock5.htm
• Critical Care Medicine Tutorials-Invasive Cardiovascular Monitors When I use these retrieved from www.continumtutors.com/cct1/shock/clinshock7.htm
• Shock, Cardiogenic retrieved from emedicine.medscape.com/article/759992-print
• Shock retrieved from www.merckmanuals.com/professional/vaso6/b/v067/v067b.htm

References:

• TCHP Education Consortium –Hemodynamic Monitoring Primer
• Hypovolemia retrieved from en.wikipedia.org/wiki/Hypovolemia

References:

• Critical Care Medicine- Venous Oximetry: The concept of Svo2 and Scvo2 retrieved from icucare.blogspot.com/2009/08/venous-oximetry-concept-of-svo2-and.html
Hand Washing

Compliance
Frequency
Skin irritation
Jewelry
Nails

Handwashing

• Compliance:
  – RN….52%
  – MD….23%

• Michigan study bacteria on hands:
  – Nails..before 73%
  » After...68%
  » No nails 32% .. 26%

Another study

• RN’s washed %
  • Before care 62
  • After care 87
  • Move dirty to clean 60
  • After remove gloves 80
  • Before invasive procedures 57
  • After direct contact fluids 87
  • Before touching own eyes etc 3

Patient Susceptibility

• Age
• Why they are in the hospital
• Co-morbid conditions
• Contacts with carriers
• Nutrition
• Stress
Fever

- Benefit
  - >100.4 (38C) kills bugs, helps neutrophils and antibodies, potentiates antibiotic activity

- SCCM:
  - 100.4 fever
  - 101.5 treat
  - 102 most likely infection

Consequences

- dehydration
- increased metabolism
- increased cardiac output
- delirium
- increased MVO2
- patient discomfort
  
  Each 1° raises metab rate 13%

Fever

- Management of non-beneficial fever:
  - Thermoregulation is impaired at 104°/40 C
  - Evaporative cooling best
  - Meds
  - antibiotics

Cultures

- Why?
  - To accurately identify bacteria

- Where?
  - Blood
  - Urine
  - Sputum
  - Lines
  - CSF

Blood

- Bacteria showers 1 hr after temp spike
- Draw blood before antibiotics
- Peripheral sticks, usually 2 sets
- Redraw 24 hrs later
- Prep site, allow to dry
- Don’t change needle
- 8-10cc’s per bottle
- Contamination risk

Sputum

- Color, amt, odor?
- QBAL (qualitative bronchial alveolar lavage) vs suction
Urinary Tract Infections:

- Treatment
  - Remove catheter
  - Culture
  - Antibiotics
  - Sterile technique

S/S
- Fever
- Urgency
- Frequency
- Dysuria
- Supra-pubic tenderness
- Positive urine culture

40% infections
Sepsis in immunosuppressed

Pneumonia: S/S

- Sputum
- Positive culture
- Fever
- Tachypnea
- Auscultation
  - quiet over area, coarse around

Pneumonia: Risks

- Host factors
- Bacterial colonization
- Aspiration
- Contaminated equipment
- Aerosolization
- Inadequate pulmonary clearance

Ventilator Associated Pneumonia (VAP)

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Consultant, Clinical Research and Critical Care Nursing
Issued 01/2008
Reviewers: Suzi Burns, Mary Jo Grap, Judy Verger, and Lori Jackson

Prevention of Ventilator Associated Pneumonia (VAP)

Lecture Content

- Epidemiology of VAP
- Prevention strategies
  - HOB elevation
  - Ventilator equipment changes
  - Continuous removal of subglottic secretions
  - Handwashing
Epidemiology of Ventilator Associated Pneumonia (VAP)

Nosocomial Pneumonias
- Account for 15% of all hospital associated infections
- Account for 27% of all MICU acquired infections
- Primary risk factor is mechanical ventilation (risk 6 to 21 times the rate for nonventilated patients)

Susceptibility to Nosocomial Pneumonias

Primary Route of Bacterial Entry into Lower Respiratory Tract
- Micro or macro aspiration of oropharyngeal pathogens
- Leakage of secretions containing bacteria around the ET cuff

VAP Etiology
- Most are bacterial pathogens, with Gram negative bacilli common
- Pseudomonas aeruginosa
  - Proteus spp
  - Acinetobacter spp
- Staphlococcus aureus
- Early VAP associated with non-multi-antibiotic-resistant organisms
- Late VAP associated with antibiotic-resistant organism

Significance of Nosocomial Pneumonias
- Mortality ranges from 20 to 41%, depending on infecting organism, antecedent antimicrobial therapy, and underlying disease(s)
- Leading cause of mortality from nosocomial infections in hospitals
Significance of Nosocomial Pneumonias

- Increases ventilatory support requirements and ICU stay by 4.3 days
- Increases hospital LOS by 4 to 9 days
- Increases cost - > $11,000 per episode
- Estimates of VAP cost/year for nation > $1.2 billion

VAP Prevention

Continuous Removal of Subglottic Secretions

Use an ET tube with continuous suction through a dorsal lumen above the cuff to prevent drainage accumulation.

Continuous Removal of Subglottic Secretions

- Smulders et al. Chest 2002;121:858-862

CDC Guideline for Prevention of Healthcare Associated Pneumonias
2004 ATS/IDSA Guidelines for VAP 2005

Smulders et al. Chest;121:858-862

HOB Elevation

HOB at 30-45°

CDC Guideline for Prevention of Healthcare Associated Pneumonias
2004 ATS/IDSA Guidelines for VAP 2005

VAP Reduction with ET Suction Above the Cuff

- No Suction
- Suction

HOB at 30-45°
HOB Elevation

- Ibanez et al. *JPEN* 1992;16:419-422
- Davis et al. *Crit Care* 2001;5:81-87

**HOB at 30-45°**

HOB Elevation Leads to Significant Deduction in VAP

Is HOB Elevation Done?

Despite effectiveness of HOB elevation, compliance is poor.


Handwashing

**What role does handwashing play in nosocomial pneumonias?**

VAP Prevention

Wash hands or use an alcohol-based waterless antiseptic agent before and after suctioning, touching ventilator equipment, and/or coming into contact with respiratory secretions.
VAP Protection

- Use a continuous subglottic suction ET tube for intubations expected to be > 24 hours
- Keep the HOB elevated to at least 30 degrees unless medically contraindicated

No Data to Support These Strategies

- Use of small bore versus large bore gastric tubes
- Continuous versus bolus feeding
- Gastric versus small intestine tubes
- Closed versus open suctioning methods
- Kinetic beds

Oral Care

- Role of oral care, colonization of the oropharynx, and VAP unclear – dental plaque may be involved as a reservoir
- Limited research on impact of rigorous oral care to alter VAP rates
- Surveys indicate most nurses use foam swabs rather than toothbrushes in intubated patients

Need Further Assistance?

For more information or further assistance, please contact a clinical practice specialist with the AACN Practice Resource Network.

practice@aacn.org

(800) 394-5995, x217

VAP

Risk Factors

- ETT > 6 days
- Re-intubated within 72 hours
- Neurosurg, trauma, or burn patient
- Decreased LOC + secretions
- H2 blockers
- NG tube present

Pneumonia: Management

- WASH HANDS!
- Oral care q2-4h
- Suction above cuff
- Separate suction canisters
- Clean in-line suction catheter
- Position side to side
Catheter Related Infections

Increased risk with:
- emergency visit
- more lumens
- central line
- long time
- TPN/lipids
- inexperienced operator
- Cultures
- tip of line and blood culture

Bundle report 2009

- VAP prevention
  - wean ASAP, sedation vacation, head of bed up at 30 degrees, DVT & PUD prophylaxis
- CRBSI/CLABSI prevention
  - hand hygiene before line insertion, maximal sterile barriers, CHG skin antiseptics (30/30 rule), optimal site selection, daily assessment

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CMS’s “Never Events”

- Blood incompatibility
- Air embolism
- Surgical site infection CABG
- UTI
- Vascular catheter associated infection
- Fall and trauma
- Pressure ulcer III and IV

Infectious Diarrhea

Causes
- Antibiotics
- Tubefeeding

Signs and Symptoms
- Fever
- Watery diarrhea
- Cramping

Management
- DC antibiotics
- Metronidazole, then oral Vanco

C diff

- Isolation
- Contaminates every surface
  - 78% still contaminated after cleaning
- Wash with soap and water
  - Alcohol doesn’t kill
  - Bleach works
- 20% uniforms
Common Infections: Sinusitis

Causes and Risks
- Tubes in the nose
- Antibiotics
- Open head injury

Signs and Symptoms
- Fever
- Drainage-not often
- Pain/Pressure
- Smell

Necrotizing Fasciitis

#1 organism =
Group A streptococcus (GAS)
Many other organisms can cause it too!
Gas gangrene: clostridial myonecrosis

4% of bacteria are in us all the time, 1% cause problems

Necrotizing Fasciitis

Non-specific erythema
- Edema
- Extreme pain
- Pallor/gray discoloration
- Anesthesia
- Purpura
- Hemorrhagic bullae
- Gas bubbles on X-ray

Hemorrhagic blistering

Pustule

Blistering
Necrotizing Fasciitis

Surgical debridement

Antibiotics

Supportive care

Case study

• 79 yo male fell 3 days ago, abrasion to (L) arm
• PMHx: metastatic squamous cell CA of the lips with neck dissection 2 months ago
• Arm is now “ecchymotic up and down its entire extent, and the hand is now cool and mottled”

Case study

• Hypotensive
• OR:
  – Fascial and muscle debridement
  – Amputation mid upper arm
  – Culture of tissue:
    • “many gram negative cocci”
    • “many Group A beta hemolytic Streptococcus isolated”

No further debridement of wound needed in the OR
• 2 days later a R antecubital A-line infiltrated. Area white and cold
• 6 hrs later area red with streaks, hand dark and cold
• Debridement with multiple further dressing changes and trimming.
• Cultured same Group A Strep

Meningitis

Viral

Rarely fatal
Treatment is symptomatic
Symptoms last 7-10 days
Resp secretions

Bacterial

May be fatal
Treatment is supportive
Antibiotics a must!
Break in dura, URI, strep
Symptoms of Meningitis

- Fever
- Severe headache
- Nuchal rigidity
- Photophobia
- Confusion/sleepiness

Diagnose: LP, WBC, glucose

Isolation
10 days after surgery

Brain Abscess

Causes

Signs and Symptoms

- Headache
- Fever
- Focal neuro changes
- In 50% - seizures, nuchal rigidity, n/v, papilledema

Cardiac Valve Infection

Causes

- Cardiogenic fever
- Drug abuse

Signs and Symptoms

- General
- Regurgitant murmur
- S/s embolization
- Fever with shaking chills

Cardiac Valve Infection

Diagnosis and Treatment

- Echocardiogram
- Blood cultures
- Antibiotics
- Surgical debridement/excision/replacement

Surgical Wound with Dehiscence

Causes

- Dehiscence
- Evisceration

Signs and Symptoms

- Pain
- “boggy” stretching of suture line
- Fever
- Increased WBC

Treatment

- If minor, call MD immediately
- If major, call for help immediately

- Sterile NS soaked 4X4’s
- Comfort
- Surgical debridement w/wo secondary closure
- Antibiotics
Candida

- Common in mouth (thrush)
- Moist areas, low oxygen
- Systemic difficult to treat
- Multiple antibiotics increase risk
- Treat:
  - Nystatin s/s
  - Suppositories
  - Powder/cream
  - Fluconazole or amphotericin B

MRSA

- Penicillin is now completely useless against staph aureus
- Oxacillin on lab results
- Vancomycin treatment of choice
- Nosocomial strains resistant up to 50% time

VRE

- Generally effects only debilitated or immunocompromised pts
- Large cause vanco use for MRSA
- Synercid
- Linezolid

MRSA Cases Last 10 Years

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Increased x 4.7  Increased x 2.4

Hospital Acquired VRE 2008
Next up:

- Pseudomonas
- Klebsiella

Other Bug Fighters

- Amphotericin B
  - Antifungal
  - Fever, chills, rigors, and n/v
  - May do test dose first
  - Premedicate
  - Monitor VS q 15' X 1 hr, then q2h
  - May give fluid flush before and/or after

Infection case study

- 38yo female from Ontario
- Hx HA for 4 days, felt “crappy”
- ER 6/10 w/ HA, weak/numb LE’s
- Admitted 8pm
- CSF cx, yellow glucose 77, hi protiens
- r/o cord compression, r/o GB, ?viral
- Head CT negative

Case study

- 0800 BP 220/110, 120, 20’s, sats 80’s
- SICU ? Guillian-Barre
- Numb and paralized to above nipple line
- Drowsy, but oriented
- Runs V-tach, change in voice quality
- Intubated and sedated
- BP 41/28 140

Case study

- Back to CT no change
- Norepi, phenyl, dopamine....swan
- Hemodynamics normal
- Flash pulm edema...... no cough
- No brainstem reflexes , fixed pupils
- Ventic ICP 15

Case study

- Work up:
  - Infection... culture everything
  - Exposed to chicken pox 2 wks ago
  - Canada
  - HIV
  - Infectious disease consult
    - West nile vs rabies
Case study

• Results
  – No growth to date
  – No herpes simplex seen in CSF
  – Viral encephalomyelitis of unknown origin
  – + HIV

Case study

• No change in status, drips, neuro
• 6/11 2000 CT
  – Significant brain stem swelling, hydrocephalus
  MRI
  -inflamm consistent w/ encephalitis

  “Acute Disseminated Encephalomyelitis”
  +Varicella chain RX

Case study

• Repeat CT
• Intraparenchymal hemorrhage in pons and 4th ventricle
• Removed from support

Sepsis and Septic Shock

SIRS

• Systemic Inflammatory Response Syndrome
• signs and symptoms of infection without identifiable source

  2 or more:
  T>100.4 <96.8
  HR>90
  RR>20
  WBC>12,000 or <4000

Sepsis

• HR > 90
• Temp > 38º C (100.4º F)
  or < 36º C (96.8º F)
• RR > 20 or PaCO2 < 32 mm Hg
• WBC > 12K < 4K, or > 10% bands
• Infection causes inflammatory response
Severe Sepsis

sepsis + signs of organ system failure, hypoperfusion, or hypotension

Septic Shock

sepsis + hypotension + perfusion defects

Who’s At Risk?

- being in a health care setting
- having natural defenses broached
- immunocompromise: age, HIV, diabetes
- co-morbid conditions
- surgery, trauma, or necrosis of abdomen

Causative Organisms

**Gram negative organisms**
- Klebsiella, E.coli,
- enterobacter

**Gram positive organisms**
- GAS, strep

**Opportunistic organisms**
- yeast, aspergillus
- C diff

**Table 1. Consensus conference group definitions of the stages of sepsis**

I. Systemic inflammatory response syndrome (SIRS)
   Two or more of the following:
   1. Temperature >38°C or <36°C
   2. Heart rate >90
   3. Respiratory rate of >20
   4. WBC count of >12 x 10^9/L or <4 x 10^9/L or 10% immature forms (bands)

II. Sepsis
   SIRS plus a culture-documented infection

III. Severe sepsis
   Sepsis plus organ dysfunction, hypotension, or hypoperfusion (including but not limited to lactate acidosis, oliguria, or acute alteration in mental status)

IV. Septic shock
   Hypotension (despite fluid resuscitation) plus hypoperfusion abnormalities
After Exposure...

- Release of Cytokines
- Vasodilation
- Inadequate tissue perfusion
- Third-spacing
- Capillary permeability

Hyperdynamic state
Hypodynamic state

Hyperdynamic Stage

Early - Fix them now!

- Tachycardia
- Hypotension
- High cardiac output
- Low SVR

Symptoms

More Symptoms

- Endocrine
  - Insulin resistant
- Labs
  - Lactate/pH
  - WBC’s
  - Platelets/albumin
  - ABG/SVO2

Care of the Septic Patient:
Source Control

- Find source of infection
  - Can't ID bug up to 70 % of the time
- Treat source
  - Surgical
  - Medical
  - Antibiotics

- Prevent new infections

Hemodynamic Support

Fluid resuscitation
- Crystalloids
- Colloids (not trauma)
- 6-10 L, retain 25%

Vasopressor/Inotropic Support
- Dopamine
- Dobutamine
- Norepinephrine
- Phenylephrine
Supportive Care

**Pulmonary Management**
- Decrease O<sub>2</sub> demands
- ARDS/PEEP
- Prone positioning

**Nutrition**

**Family Support**

---

Hypodynamic Stage

**Late!**

- Tachycardia
- Hypotension AND Low cardiac output
- High SVR

---

Symptoms

- **Cardiovascular**
  - Tachy, low BP... low HR
  - Low CO, hi SVR/PAWP
  - Cool, clammy, mottled
    - hypothermic
- **Pulmonary**
  - Crackles, ARDS
  - Acidotic
  - PEEP, PCV

---

More Symptoms

- **Neurological**
  - Coma

- **Gastrointestinal**
  - No BS
  - Lg NG output
  - Transmigration of bacteria

---

More Symptoms

- **Renal**
  - Anuric
  - Hi BUN/Cr

- **Hepatic**
  - LFT’s up
  - DIC ?
  - bleeding

---

More Symptoms

- **Endocrine**
  - Insulin resistance...hi glucose...low....
  - MDF lowers HR
- **Laboratory results**
  - ABG...acid/lactate
  - Coag up
  - Platelets low, fibrinogen low
  - Electrolytes off

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Images have been removed from the PowerPoint slides in this handout due to copyright restrictions.
Atelectasis in ARDS

Disseminated Intravascular Coagulation

- Coagulation & Inflammation
- Fibrinolyis
- Inappropriate clotting
- Microemboli
- Loss of clotting factors
- Widespread cell death
- Bleeding

DIC

- Clotting:
  - Platlets/fibrinogen sent to stop bleeding and clot
- Hemorrhage:
  - Plasminogen activated...lyses clot
  - Develop FSP, FDP, D-dimer (potent anticoagulants)
  - Consumption of platlets/fibrinogen

DIC case study

- 39 yo F, placenta previa, placenta accretia
- 34 wks bleeding... C-section
- Amniotic fluid embolus
- Coded immediately after delivery and went into fulminate DIC

DIC case study

- LABS
  - Hgb...9.6 (1245) pre op
    - 6.3 (1846) code troponin
    - 3.7 (1950) in SICU 1.2
    - 11.4 (2123) SICU 5.5
    - 4.9 (2225) 0030 9.5
  - Other labs:glucose >200
    - Calcium low
DIC blood products
- PRBC’s: 50 units
- Platlets: 7 6pks
- FFP: 28 units
- Cryo (jumbo): 8

DIC case study
- Summary:
  - Clotted an 8” section of infrarenal vena cava
  - MODS
  - Planned transfer to U of M for clot removal, dislodged clot with neuro, renal, and cardiac affected.
  - Has since recovered enough to be alert, reorientable, and getting ready for rehab

DIC

Relationship of DIC & sepsis

Hemodynamic Support for sepsis/septic shock

**Fluid balance**
- Fluids
- Diuretics
- Dialysis/CVCC

**Vasoactive Drugs**
- Inotropes
- Vasodilators
- Vasopressors

Other measures
- Pulmonary management
- Management of DIC
- Family support
**Other Measures**

**Xigris (drotrecogin alfa [activated])**
- Inhibits TNF
- Blocks leukocyte adhesion
- Limits thrombin-induced inflammatory responses within the microvascular endothelium

**Complication - BLEEDING!**
- Infused 48-96 hours
- 12 mcg/kg/hr - 30 mcg/kg/hr
- Only used in SEVERE sepsis

**Nitric Oxide**
- Vasodilates by inhibiting angiotensin II and sympathetic vasoconstriction
- Inhibits platelet/WBC adhesion

**NO Inhibition -**
\[ N\text{-monomethyl-L-arginine} = L\text{-NMMA} \]

**Vasopressin**
- 0.04 u/min
- Restores responsiveness to vasopressors
- NO mediates inhibition

**Insulin**
- < 150 recommended by SCCM
- High blood sugar creates sticky leukocytes

**Adrenal replacement therapy**
- Modulates inflammatory cytokines
### Other measures

- Nutritional assistance
  - Glutamine: T-cell improve, bactericidal, essential amino acid
  - Selenium: Less renal problems
  - Vit C: radical scavenger
  - Vit E: ATP, less peroxidation

### Sepsis case study

- 44 yo MVA unrestrained, ejected
- PMHx: cocaine, ETOH, smoker
- Splenic fracture
- Multiple rib fx with flail chest/hemopneumo
- Intubated
- Labs unremarkable

### Case study

- **Day 3**
  - Attempts at weaning fail
  - VS stable except T103
  - Labs stable pO2 73
  - QBAL culture gram + cocci
  - Plan: early tracheostomy, re-culture

### Case study

- **Day 4**
  - Lungs deteriorating
  - FiO2 up to 90%
  - 99/43 120 14 102T
  - +2 pitting edema
  - QBAL >100,000 group A strep
  - Treat for ARDS

### Case study

- **Day 5**
  - Continue with poor ventilatory state, flolan
  - Intubated, sedated, paralyzed
  - 100/60 110 14 103.2 T
  - Swan: SVO2 75% (89%) CO 14 SVR 463
  - CT negative for PE
  - Cultures GAS, staph
  - Troponins rising, afib

### Case study

- **Day 6**
  - Echo
  - PEEP 15
  - PCV
  - Troponins continue to rise
  - Check cortisol levels
  - Proned
Case study

- Day 7
- FiO2 70% AC 550  R23  PEEP 12
- Sats low 90’s, SVO2 60’s
- T 102.7
- diuretics

Case study

- Slowly improved to SIMV with periodic trach dome trials
- Transferred to a rehab vent facility after 21 days

Toxic Shock Syndrome

What bugs?

Signs/Symptoms

- Mild prodromal symptoms
- Precipitous acute illness: high fever, n/v, abdominal pain, severe muscle pain, headache
- profuse diarrhea, macular erythroderma

Treatment

- Early diagnosis
- Early antibiotics - broad spectrum
- Supportive care

Toxic shock case study

- 28 yo F
- 2 previous cases of toxic shock (14, 18)
- c/o fever, myalgia, * groin pain
- Day 3, neuro change... intub... CT neg w/ free fluid in abd... ex lap... compartment syndrome
- Day 6, abd open, hypotensive on dopamine and levophed, low SVR and hi CO... sepsis

TS case study

- Dx: “distributive shock with multi-organ dysfunction, possibly toxic shock”
- Summary
- Day 9
  - Fixed, dilated pupils  cerebral edema
  - No source of infection ID’d
Multiple Organ Dysfunction Syndrome

- Cardiovascular
- Pulmonary
- Renal
- CNS
- Hepatic
- Splanic

MSOF

- Causes
  - Primary
    - Aspiration leading to ARDS
    - PE
    - Trauma to ABD
  - Secondary
    - Hypoperfusion
    - Microemboli

APACHE II criteria

Cardiovascular failure
- HR ≤ 54/min
- MAP ≤ 49 mmHg
- V-tach, V-fib or both
- Serum pH ≤ 7.24
- PaCO2 ≤ 40 mmHg

Respiratory failure
- RR <5/min or > 49/min
- PaCO2 > 50 mmHg
- P(A-a)O2 > 350 mmHg
- Vent/CPAP dependent day 4

Renal failure
- UO <479 mL/24h or <159 mL/8h
- Serum BUN > 100 mg/dL
- Serum Creatinine >3.5 mg/dL

Hepatic failure
- Serum Bilirubin >6 mg/dL
- PT >4 seconds over control (without systemic anticoagulation)
Hemodynamic, Shock, and Infection in Critical Care

APACHE II criteria

- Hematologic Failure
  - WBC < 1000/μL
  - Platelets < 20,000/μL
  - Hematocrit < 20%

APACHE II criteria

- CNS failure
  - GCS ≤ 6 (without sedation)

Multiple Organ Dysfunction Syndrome

- Fluid resuscitation
- PRBC administration
- Supportive care for each organ system

Neurogenic & Anaphylactic Shock

Sam Johnson was involved in a car accident in which he was driving and hit from behind. When EMS arrived, he was confused but able to indicate that he could not move his arms or feet. VS were stable and he was immobilized and transferred to the ED. Spine films revealed a C6-C7 fracture of the spinal cord. Physical assessment revealed loss of movement and sensation from 1 inch above his nipple line and down. Sam was started on a methylprednisolone infusion and transferred to the SICU.

Once in the SICU, Sam’s VS were unstable. His HR dropped to 50 and his BP dropped to 84/44. Neurogenic shock was anticipated.
What is Neurogenic Shock?

Loss of autonomic nervous system regulation below level of injury causes massive vasodilation and bradycardia.

Causes of Neurogenic Shock

- Spinal Cord Injury
  - T6 or higher
  - Anesthesia
  - Drug overdose
  - Pain
    - All interrupt SNS and vasomotor center

Signs of Neurogenic Shock

- Bradycardia
- Profound hypotension
- Warm and dry skin below LOI
- May have...
  - Mentation changes: Paralysis
  - Nausea/vomiting: Apnea/tachypnea

Interventions for Neurogenic Shock

- ABC’s and oxygen
- Check fluid status
- Remove underlying cause
- Vasopressors x 72 hours
- Fluids early to prevent parasympathetic NS from firing

Anaphylactic Shock
1. Blood Transfusion Reaction
Sarah Reed, 68/yo, is admitted to the MICU with a GI bleed. She has received 3 units of blood on your shift and you just started her fourth unit of blood 20 minutes ago. She complains to you that she “feels funny” and her face is flushed and her temperature has gone from 98.8 to 101.6 F.

Four Types of Blood Reactions

- Acute Hemolytic Transfusion Reaction
  - Rx to ABO/Rh
- Febrile Non-Hemolytic Transfusion Reaction
  - Rx to Antibodies or elements
- Mild Allergic Reaction
  - Hives/itch
- Anaphylactic Reaction
  - Severe Rx to proteins

Signs of a Transfusion Reaction

- Non-Hemolytic (antibodies)
  - Fever
  - Chills
    - >1.5 baseline
    - Flushed

- Mild Allergic
  - Hives
  - Urticaria

- Most common

Hemolytic Transfusion Reaction

- Rx to blood type
  - Fainting, dizziness, anxiety
  - Chest pain
  - Hypotension
  - Bronchospasm
  - Blood urine
  - Flank/back pain
  - Nausea/vomiting

Emergency Measures for a Blood Transfusion Reaction

- Stop the blood!!!
  - Assess ABC’s
- Remove blood from the line or change
- Medications
- Send labs

COMPLICATIONS
(poor oxygen delivery)

- Discomfort
- Anemia
- Acute kidney failure
- Shock
- Lung dysfunction
- Cardiac ischemia
- DIC
Massive transfusion

• 1 blood volume replaced in 24 hrs
• Mortality 50% (higher in elderly)
• 45% pts with >10 units in 24 hrs develop ARDS (many researchers feel actually TRALI then ARDS later)
• Infection rate 50% if >7 units

Massive transfusion complications

• MSOF, ARDS
• 47% develop coagulopathy
• 25u components exposes to 80 different donors
• Febrile non-hemolytic transfusion Rx
  – Occurs 20%  
  – Risk:  
    • 1:5 platelets  
    • 1:100 RBC’s
• Delayed hemolytic reaction 1:2500
  – 2-14 days post TX
  – Fever, jaundice
  – From clearance of antibody coated RBC’s

Antibodies and TRALI

• Found in donor serum
  – Most common cause because able to react with entire circulating blood pool of WBC’s
• Can be in recipient
  – Less frequent because limited # WBC’s in donor product

Radiology

• Bilateral pulmonary infiltrates
  – Appear at time of reaction and resolve in 96 hrs in 80% pts
  – ABG’s are altered for same time frame
  – Infiltrates persist for 7 days in other 20%
  – White out from WBC aggregation and sequestration in lung

Massive Transfusion

• Complication is diffuse microvascular bleeding or “oozing” coagulopathy
  – Labs cannot predict
  – Deplete coag factors to 37% after 10 units and still have normal coagulation
  – Platelets drop inversely to blood given (50-20u)
2. Anaphylaxis and Anaphylactic Shock

Agnes White is a 56y/o beekeeper who was tending her bees when she accidentally knocked over one hive. It is estimated that she was stung over 100 times. She is admitted to your unit from the ER, where she received 5 liters of fluid. She is edematous, with a HR of 124, and BP of 70/44.

Anaphylaxis: When the Immune System Goes Nuts

Histamine and other substances released in mass

Massive vasodilation
Increase in capillary permeability

(IGE stim-mastcell-histamine-platelet activating factor)

Causes of Anaphylaxis

Latex

- Home
  - Diapers
  - Mouse pads
  - Erasers
  - Rug backing
  - Zip lock bags
  - lottery tickets
  - socks
- Hospital
  - Ace wraps
  - Electric cords
  - Shoe covers
  - Stethoscope tubing
  - Injection ports
  - masks

Symptoms of Anaphylactic Shock

- Hypotension
- Tachycardia
- Decreased SVR
- Edema
- Wheezing/SOB
- Nausea/vomiting

Hemodynamics of anaphylactic shock

- BP- low
- HR- high
- CO- low
- RA/CVP- low
- PAOP(wedge)- low
- SVR- low
Treatment for Anaphylaxis

- ABC’s
- Epinephrine SQ 1:1000 Q10-15”
- Steroids (hydrocortisone 5mg/Kg)
- Benadryl

Tx for Anaphylactic Shock

- All of anaphylaxis interventions
- Epinephrine IV 5-10 mcg
- Fluid volume resuscitation (lose 40% into interstitium)
- Epi or dopamine drips
- Amrinone or milrinone (bronchodilates)

Putting It All Together

Ms. E. is a 54 yo female who was admitted for chest pain and MI. She had chest pain for three days before going to the MD. Physical exam shows:
- RR 26 and labored
- HR 136, BP 72/48
- Skin cool and pale
- Troponin 4.1

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)

Putting It All Together

Mr. A, a long-time nursing home resident, comes to the hospital via ambulance. Physical exam shows:
- RR 28, crackles 1/3 up bilaterally
- HR 122, BP 70/46
- Skin warm, sweaty
- UO 150 with cath; foul smelling, cloudy

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)

Putting It All Together

Mr. B. is a 57 yo male returning from the PACU after spinal surgery for chronic thoracic back pain. Physical exam shows:
- RR 10
- HR 45, BP 80/44
- Skin warm and dry from nipple line down; cool and clammy from the nipple line up

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)

Putting It All Together

Ms. C. is a 16 yo female who has had uncontrolled diarrhea and vomiting for three days. Physical exam shows:
- RR 22
- HR 136, BP 84/36
- Skin cool and pale
- UO 35 through cath; clear

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)
Putting It All Together

A 46yo female comes to the ER complaining of SOB.

Physical exam shows:
• RR 24, labored
• Inspiratory and expiratory wheezes progressing to audible stridor
• HR 130, BP 180/94 initially then drops to 80/44, HR 145
• Skin warm and red

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)

Putting It All Together

Mr. D. is a 77 yo patient with a COPD exacerbation and ventilator dependence. Physical exam shows:
• RR 20 on AC 12
• HR 106; BP 120/78
• Skin warm and slightly diaphoretic
• Temp 102.6°F

What will the CVP/RA be?
- the wedge?
- the CO?
- SVR? (vasodilated or vasoconstricted)