Hope Dykes

N441 Concept Review Exam 2

Emphasized information

1. **Total water composition**
    1. Intracellular (ICF)
        1. Approximately 2/3 of fluid in body

        Fluid in the cells
1. Extracellular (ECF)
    1. Approximately 1/3 of fluid in body
    1. interstitial fluid

        Fluid in spaces between cells
    2. intravascular

        Plasma

1. Third spacing
    1. Loss of ECF into a space that does not contribute to equilibrium between the ICF and ECF
    2. Early evidence: decreased urine output
    3. s/s: increased HR, decreased BP, decreased CVP, edema, increased body weight, imbalances in I/O
    4. Occur in patients whith hypocalcemia, decreased iron intake, severe liver diseases, alcoholism, hypothyroidism, malabsorption, immobility, burns, cancer

2. **Regulation of Fluid**

    a. Heart/ blood vessels

    Hydrostatic and osmotic pressure allows fluid movement across membranes

    Move fluid throughout body for equal distribution

        Lack or incorrect movement causes edema (extremity and pulmonary)

    b. Lungs

Eliminate water vapor (insensible loss) at rate of approx 300mL/day

Loss increases with increased respiration rate or in dry climate

c. Kidneys

Regulate through urine output

Usual daily urine volume for adult is 1-2L

Output = approx 1mL/kg/hr

d. Pituitary

Releases ADH causing kidneys to retain water

3. **Hypovolemia/Hypervolemia**

a. s/s

Hypo

Imbalance	Contributing Factors	S/Sx & Laboratory Findings

Fluid Volume Deficit (hypovolemia)	Loss of water and electrolytes, as in vomiting, diarrhea, fistulas, fever, excess sweating, burns, blood loss, gastrointestinal suction, and third-space fluid shifts; and decreased intake, as in anorexia, nausea, and inability to gain access to fluid. Diabetes insipidus and uncontrolled diabetes both contribute to a depletion of extracellular fluid volume.	o S/Sx: Acute weight loss, ↓ skin turgor, oliguria, concentrated urine, capillary filling time prolonged, low CVP, ↓ BP, flattened neck veins, dizziness, weakness, thirst and confusion, ↑ pulse, muscle cramps, sunken eyes, nausea, increased temperature; cool, clammy, pale skin o Labs indicate: ↑ hemoglobin and hematocrit, ↑ serum and urine osmolality and specific gravity, ↓ urine sodium, ↑ BUN and creatinine, ↑ urine specific gravity and osmolality

Hyper

Imbalance	Contributing Factors	S/Sx & Laboratory Findings

Fluid Volume Excess (hypervolemia)	Compromised regulatory mechanisms, such as renal failure, heart failure, and cirrhosis; overzealous administration of sodium-containing fluids; and fluid shifts (i.e., treatment of burns). Prolonged corticosteroid therapy, severe stress, and hyperaldosteronism augment fluid volume excess.	o Acute weight gain, peripheral edema and ascites, distended jugular veins, crackles, elevated CVP, shortness of breath, ↑ BP, bounding pulse and cough, ↑ respiratory rate, ↑ urine output o Labs indicate: ↓ hemoglobin and hematocrit, ↓ serum and urine osmolality, ↓ urine sodium and specific gravity

b. Nursing Management

Hypo

Oral fluids preferred if possible

Monitor I/O Q8 *(output <0.5/1 mL/kg/hr)*, daily weight *(loss of 0.5kg)*, vitals *(weak/rapid pulse*, *orthostatic hypotension*, *decreased body temp)*, CVP, LOC, breath sounds, skin color, volume overload, skin turgor

Hyper

Diuretics

Thiazide (block Na reabsorption in distal tubule)

hydrochlorothiazide

Loop (greater loss of Na and H2O by blocking reabsorption in loop of Henle)

Furosemide, bumetanide, torsemide

Dialysis

Hemo- or peritoneal

Monitor potassium and acid-base balance

Measure I/O, daily weight, assess breath sounds, monitor dependent edema, low-sodium diet

c. Isotonic solutions→

0.9 Normal Saline (0.9NS)

Lactated Ringers (LR)

Hypo--> use hypertonic solutions

3% NaCl

5% NaCl

D5NS

D10W/D20W/D50W

Hyper --> use hypotonic solutions

0.45%/0.33%/0.225% NaCl

D2.5W

4. **Hyponatremia/Hypernatremia**

Electrolyte	Functions	Sources & Losses	Regulation

• Sodium (Na+) o Chief electrolyte of ECF o Normal serum sodium: 135-145 mEq/L	o Regulates extracellular fluid volume; Na+ loss or gain accompanied by a loss or gain of water o Affects serum osmolality o Role in muscle contraction and transmission of nerve impulses o Regulation of acid–base balance as sodium bicarbonate	o Normally enters the body through the GI tract from dietary sources, such as salt added to processed foods, sodium preservatives added to processed foods o Lost from gastrointestinal tract, kidneys, and skin	o Transported out of the cell by the sodium-potassium pump o Regulated by renin-angiotensin-aldosterone system o Elimination and reabsorption regulated by the kidneys o Sodium concentrations affected by salt and water intake

1. Contributing Factors

1. Hypo
    1. Draining wounds, v/d, dilutional, renal failure, SIADH
2. Hyper
    1. Inadequate water intake, excess water loss, sodium gain (rare)

2. s/s
    1. Hypo
        1. Poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes, confusion
    2. Hyper
        1. Thirst, elevated temperature, dry/swollen tongue, sticky mucosa, neurologic symptoms, restlessness, weakness
3. Nursing Management
    1. Hypo
        1. Assessment/prevention, dietary sodium/fluid intake, monitor at-risk patients, know effects of medications (diuretics, lithium)
    2. Hyper
        1. Fluid replacement (water deficit)
        2. D5W (sodium excess, diuretics, restrict dietary sodium)
        3. Assessment/prevention, assess for OTC sources of sodium, offer/encourage fluids to meet needs, sufficient water with tube feedings

5. **Hypokalemia/Hyperkalemia**

Electrolyte	Functions	Sources & Losses	Regulation
• Potassium (K+) o Major cation of ICF o Normal serum potas	o Controls intracellular osmolality o Regulator of cellular enzyme activity o Role in the transmission of	o Adequate quantities via a well-balanced diet o Leading food sources: fruits and vegetables, dried peas and beans, whole	o Regulated by aldosterone o Eliminated by the kidneys (no effective method of conserving potassiu

sium: 3.5-5.0	electrical impulses in nerve, heart, skeletal, intestinal, and lung tissue; protein and carbohydrate metabolism; and cellular building o Regulation of acid–base balance by cellular exchange with H+	grains, milk, meats o Lost via kidneys, stool, sweat, emesis o Gastrointestinal (GI) secretions contain potassium in large quantities, so can be lost through vomitus	m) o Additional regulation via transcellular shift between the ICF and ECF compartments

a. Contributing Factors

Hypo

GI losses, medications, alterations of acid-base balance, hyperaldosteronism, poor dietary intake

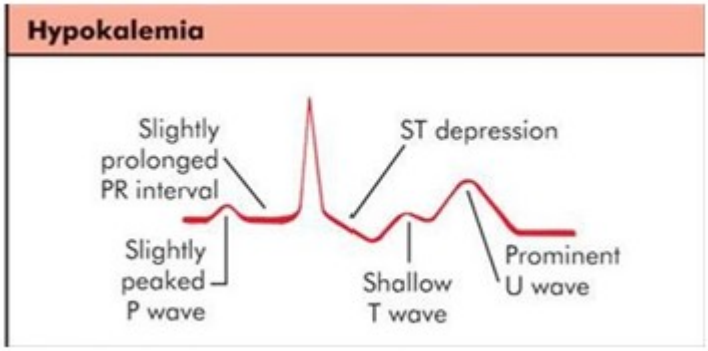
Hyper

Treatment related, impaired renal function, hyperaldosteronism, tissue trauma, acidosis

b. s/s

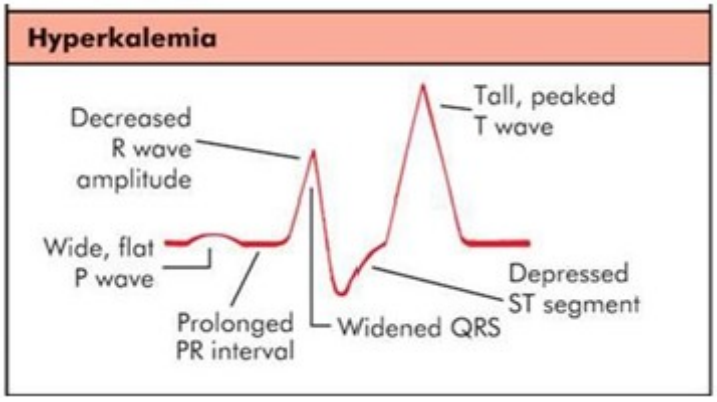
Hypo

Fatigue, anorexia, n/v, dysrhythmias, muscle weakness/cramps, paresthesia, glucose intolerance, decreased muscle strength, DTR



Hyper

Cardiac changes and dysrhythmias, muscle weakness (potential respiratory impairment), paresthesia, anxiety, GI manifestations



c. Nursing Management

Hypo

Medical

Increased dietary potassium, potassium replacement, IV for severe deficit

Assess, monitor EKG/ABG

Hyper

Monitor EKG, limit dietary K, cation-exchange resin (Kayexalate), IV sodium bicarb, IV calcium gluconate, regular insulin and hypertonic dextrose IV, beta-2 agonists, dialysis

6. **Hypocalcemia/Hypercalcemia**

Electrolyte	Functions	Sources & Losses	Regulation
• Calcium o Most abundant electrolyte in the body o 99% of body calcium is stored in bone o 1% inside cells o Normal total serum calcium level: 8.6–10.2 mg/dL o Normal ionized serum calcium level: 4.5–5.1 mg/dL	o Role in blood coagulation and in transmission of nerve impulses o Helps regulate muscle contraction and relaxation o Activates enzymes that stimulate essential chemical reactions in the body o Major component of bones and teeth	o Absorbed from foods in the presence of normal gastric acidity and vitamin D o Lost via feces and urine o Sources include milk, milk products, and cheese; dried beans; fortified orange juice; green, leafy vegetables; small fish with bones; and dried	o Primarily excreted by GI tract; lesser extent by kidneys o Regulated by parathyroid hormone and calcitonin o High serum phosphate results in decreased serum calcium level; low serum phosphate leads to increased serum calcium

		peas and beans	

a. Contributing Factors

Hypo

Hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications

Hyper

Malignancy, hyperparathyroidism, bone loss r/t immobility

b. s/s

Hypo

Tetany, circumoral numbness, paresthesia, hyperactive DTR, Trousseau (arm), Chovstek (face), seizures, dyspnea, laryngospasm, abnormal clotting, anxiety

Hyper

Muscle weakness, incoordination, anorexia, constipation, n/v, abdominal/bone pain, polyuria, thirst, EKG changes, dysrhythmias

c. Nursing Management

Hypo

Medical

IV calcium gluconate, calcium and vitamin D supplements, diet

Nursing

Assess, assist w/ weight-bearing exercises to decrease bone calcium loss, education r/t diet and meds, IV calcium administration

Hyper

Medical

Treat underlying cause, fluids, furosemide, phosphates, calcitonin, bisphosphonates

Nursing

Assess, encourage ambulation, fluids of 3-4L/day (preferably containing sodium), fiber, ensure safety

7. **Hypomagnesemia/Hypermagnesemia**

Electrolyte	Functions	Sources & Losses	Regulation
• Magnesium (Mg2+) o 2nd most abundant ICF cation after potassium o Normal serum concentration: 1.3–2.3 mEq/L	o Metabolism of carbohydrates and proteins o Activator for many intracellular enzyme systems o Role in neuromuscular function o Acts on cardiovascular system, producing vasodilation	o Enters the body via GI tract o Found in green, leafy vegetables; nuts; sea-food; whole grains; dried peas and beans; cocoa o Lost via urine with use of loop diuretics	o Eliminated by kidneys o Regulated by parathyroid hormone

a. Contributing Factors

Hypo

Alcoholism, GI losses, enteral/parenteral feeding deficient in mag, meds, rapid administration of citrated blood, DKA, sepsis, burns, hypothermia

Hyper

Renal failure, DKA, excessive administration of mag

b. s/s

Hypo

Neuromuscular instability, muscle weakness, tremors, athetoid movements, EKG changes and dysrhythmias, alterations in mood/LOC, dysphagia

Hyper

Flushing, lowered BP, n/v, hypoactive DTR, drowsiness, muscle weakness, depressed respirations, EKG changes and dysrhythmias

c. Nursing Management

Hypo

Medical

Diet, oral mag, mag sulfate IV

Nursing

Assess, ensure safety, education for diet/meds/alcohol use, IV administration of mag sulfate

Hyper

Medical

IV calcium gluconate, loop diuretics (furosemide), IV NS or LR, hemodialysis

Nursing

Assess, no meds containing mag, education about OTC meds with mag

8. **Hypophosphatemia/Hyperphosphatemia**

Electrolyte	Functions	Sources & Losses	Regulation
• Phosphate (PO_4^{-}) o Major ICF anion; a buffer anion in both ICF and ECF o Normal serum phosphate level: 2.5–4.5 mg/dL	o Role in acid–base balance as a hydrogen buffer o Promotes energy storage; carbohydrate, protein, and fat metabolism o Bone and teeth formation o Regulation of hormone and coenzyme activity o Role in muscle and red blood cell function	o Enters body via gastrointestinal tract o Sources include all animal products (meat, poultry, eggs, milk, bread, ready-to-eat cereal) o Absorption is diminished by concurrent ingestion of calcium, magnesium, and aluminum	o Eliminated by kidneys o Regulation by parathyroid hormone and by activated vitamin D o Phosphate and calcium are inversely proportional; an increase in one results in a decrease in the other

a. Contributing Factors

Hypo

Alcoholism, refeeding after starvation, pain, heat stroke, respiratory alkalosis, hyperventilation, DKA hepatic encephalopathy, major burns, hyperparathyroidism, low mag/ potassium, diarrhea, vitamin D deficiency, use of diuretics and antacids

Hyper

Renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemo

b. s/s

Hypo

Confusion, muscle weakness, tissue hypoxia, muscle and bone pain, increased susceptibility to infection

Hyper

Soft-tissue calcifications, most symptoms d/t associated hypocalcemia

c. Nursing Management

Hypo

Medical

Oral or IV phosphorus replacement

Nursing

Assess, encourage foods high in phosphorus, gradual introduction of calories for malnourished patients receiving parenteral nutrition

Hyper

Medical

Treat underlying disorder, vit D preparations, calcium-binding antacids, phosphate-binding gels/antacids, loop diuretics, IV 0.9, dialysis

Nursing

Assess, avoid high-phosphorus foods, education (diet, phosphate-containing substances, signs of hypocalcemia)

9. Labaratory testing→ Calcium, Potassium

a. Creatinine Normal = 0.6-1.2

b. Hematocrit Normal = 36-50

c. Urine sodium = 40-220

10. **Acid Base Balance (Normal/abnormal)**

a. pH

7.35 - (*7.4)* - 7.45

b. PaCO2

35 - (*40)* - 45

c. HCO3

22 - (*24)* - 26

d. PaO2

80 - 100

e. O2 Sat

>94%

11. **Acidosis/Compensation**

a. Metabolic

s/s: headache, confusion, drowsiness, increased respiratory rate/depth, decreased bp, decreased cardiac output, dysrhythmias, shock

HCO3 <22 in addition to pH <7.35

Partial compensation: CO2 <35 (alkalotic levels), pH/bicarb still abnormal

Total compensation: CO2 <35, pH/bicarb return to near/normal levels

b. Respiratory (hypoventilation)

s/s: sudden increase in pulse/respiratory rate/bp, mental changes, feeling of fullness in head

CO2 >45 in addition to pH <7.35

Partial compensation: HCO3 >26, pH/CO2 still abnormal

Total compensation: HCO3 >26, pH/CO2 return to near/normal levels

12. **Alkalosis/Compensation**

a. Metabolic

s/s: signs of decreased calcium/potassium, respiratory depression, tachycardia,

HCO3 >26 in addition to pH >7.45

Partial compensation: CO2 <35, pH/HCO3 still abnormal

Total compensation: CO2 <35, pH/HCO3 return to near/normal

b. Respiratory (hyperventilation)

s/s: lightheadedness, inability to concentrate, numbness/tingling, loss of consciousness

CO2 <35 in addition to pH >7.45

Partial compensation: HCO3 <22, pH/CO2 still abnormal

Total compensation: HCO3 <22, pH/CO2 return to near/normal

13. **Shock**

a. Cardiogenic -- failure of heart to pump effectively d/t cardiac factor

Types and Causes	Examples
• Systolic dysfunction o Inability of the heart to pump blood forward	Myocardial infarction, cardiomyopathy, blunt cardiac injury, severe systemic or pulmonary hypertension, myocardial depression from metabolic problems
• Diastolic dysfunction o Inability of the heart to fill	Cardiac tamponade, ventricular hypertrophy, cardiomyopathy
Dysrhythmias	Bradydysrhythmias, tachydysrhythmias

Structural factors	Valvular stenosis or regurgitation, ventricular septal rupture, tension pneumothorax

1. s/s

    Tachycardia, hypotension, narrowed pulse pressure, dysrhythmias

    Decreased cardiac output, cardiac index

    Tachypnea, crackles

    Peripheral hypo-perfusion

        Cyanosis, pallor, diaphoresis, weak peripheral pulses, cool/clammy skin, delayed capillary refill

    Decreased urine output, Na/Water retention

    Anxiety, confusion, agitation

2. Nursing Management

    Medication administration

        Dobutamine

        Nitroglycerin

        Dopamine

        Other vasoactive medications

        Antiarrhythmic medications

    Preventing cardiogenic shock

    Monitoring hemodynamic status

    IV fluids

    Maintaining intra-aortic balloon counter-pulsation

    Ensuring safety, comfort

b. Hypovolemic -- decreased intravascular volume d/t fluid loss

1. s/s

Increased heart rate, cardiac output, respiration rate

Decreased stroke volume, CVP, PAWP d/t low circulating blood volume

Anxious, decreased urine output

2. Nursing Management

Initiate 2 large IV lines

Fluids/meds, blood if needed

IO if needed

Crystalloid solutions

3mL solution : 1 mL known blood loss

Colloid may also be used

If cause is hemorrhage, use blood

Prevent by replacing fluids for at risk patients before intravascular volume is depleted

Restore intravascular volume

Assist with treatment of cause

Monitor hemodynamic pressure, VS, ABGs, serum lactate levels, H&H, bladder pressure monitoring, & I&O

Monitor temperature à rapid fluid resuscitation can cause hypothermia à warm IV fluids

Nurse must monitor cardiac and respiratory status closely and report changes in BP, pulse pressure, CVP, heart rate and rhythm, and lung sounds

c. Distributive -- widespread vasodilation and increased capillary permeability

1. Neurogenic -- loss of sympathetic tone causing relative hypovolemia, occurs within 30 minutes of a spinal cord injury

Risk factors

Spinal cord injury

Spinal anesthesia

Depressant action of medications

a. s/s

Bradycardia, hypotension, hypothermia

b. Nursing Management

Atropine, vasopressors, monitor

No corticosteroids

Cautiously increase fluids since hypotension NOT r/t fluid loss

2. Anaphylactic -- d/t severe allergic reaction producing overwhelming systemic vasodilation, relative hypovolemia

Risk factors

Hx of medication sensitivity

Transfusion reaction

Hx of reaction to insect bites/stings

Food allergies

Latex sensitivity

a. s/s

Present w/I 2-30 minutes of exposure

Rapid onset hypotension, neurologic compromise, respiratory distress, cardiac arrest, GI distress, skin/mucosal tissue irritation

b. Nursing Management

Prevention

Maintain patent airway

Endotracheal intubation may be needed

IM Epi

Diphenhydramine, ranitidine

Nebulized bronchodilators

Fluid resuscitation

3. Septic -- d/t overwhelming infection causing relative hypovolemia- most common type of distributive shock

Risk factors

Immunosuppression

Extremes of age (<1 & >65)

Malnourishment

Chronic illness

Invasive procedures

Emergency &/or multiple surgeries

a. s/s

RR >22

Alterated mentation (GCS <15)

SBP <100

Persisting hypotension requiring vasopressors to maintain MAP >/= 65

Blood lactate >2 despite adequate volume resuscitation

b. Nursing Management

Help determine cause

Ensure early removal of invasive devices

Prevent VAP

Debride wounds

Infection prevention

IV vasopressors (norepinephrine)

Glucocorticoids (refractory to adequate fluid resuscitation/vasopressors)

1. Obstructive -- d/t physical obstruction impeding the filling/outflow of blood resulting in reduced CO

a. s/s

Anxiety, restlessness, altered mental state
Hypotension (d/t decreased cardiac output/stroke volume)
Rapid/weak/thready pulse
Cool/clammy/mottled skin (d/t vasoconstriction/hypo-perfusion)
Oliguria (d/t decreased renal perfusion)

b. Nursing Management

Early recognition

Mechanical decompression (pericardial tamponade, pneumo)

Thrombolytic therapy for PE

Superior vena cava syndrome (radiation, debulking, removal of mass/cause)

14**. Compensatory Mechanism in Shock** -- non-progressive, measures to increased CO to restore tissue perfusion and oxygenation

a. S/S

Vasoconstriction causing increased heartrate, increased heart contractility

Cool, clammy skin, hypoactive bowel sounds, decreased urine output

Inadequate tissue perfusion

Increased respiratory rate (d/t need to compensate for metabolic acidosis)

Confusion

Metabolic acidosis (lactic acid build up)

b. Nursing Management

Early intervention

Identifying cause of shock, administering IV fluids/oxygen, obtaining necessary lab tests to r/o and treat metabolic imbalances or infection

Assess for shock--> recognize subtle clinical signs of compensatory stage before BP drops

15. **Progressive Stage of Shock** -- compensatory mechanisms begin to fail

Body System	Clinical Manifestations
Resp.	o Respirations are rapid and shallow, crackles are heard over the lung fields o Decreased pulmonary blood flow causes arterial oxygen levels to decrease and CO2 levels to increase o Hypoxemia and biochemical mediators cause an intense inflammatory response and pulmonary vasoconstriction, perpetuating pulmonary capillary hypoperfusion and hypoxemia o Hypoperfused alveoli stop producing surfactant and subsequently collapse o Pulmonary capillaries begin to leak, causing pulmonary edema, diffusion abnormalities (shunting), and additional alveolar collapse à this condition is called acute lung injury (ALI) o Interstitial inflammation and fibrosis are common consequences, leading to acute respiratory distress syndrome (ARDS)

CV	o A lack of adequate blood supply leads to dysrhythmias and ischemia, HR is rapid, sometimes exceeding 150 bpm o May complain of chest pain and even suffer a myocardial infarction (MI) o Levels of cardiac enzymes and biomarkers (e.g. troponin) increase o Myocardial depression and ventricular dilation may further impair the heart's ability to pump enough blood to the tissues to meet oxygen requirements
Neuro	o Blood flow to brain becomes impaired (cerebral hypo-perfusion) & mental status deteriorates o Initially may exhibit subtle changes in behavior (agitation and confusion), later may become lethargic & begin to lose consciousness
Body System	**Clinical Manifestations**
Renal	o MAP falls <65 mmHg = GFR drops o Acute kidney injury (AKI) à increase BUN & Cr, fluid & electrolyte shifts, acid-base imbalances, & loss of renal-hormonal regulation of BP o Urinary output decreases to <0.5 mL/kg per hour (or <30 mL per hour)
Hepatic	o Decreased blood flow to liver impairs ability of liver cells to perform metabolic and phagocytic functions; consequently, patient less able to metabolize medications and metabolic waste products, such as ammonia and lactic acid. o Pt more susceptible to infection as liver fails to filter bacteria from blood o Liver enzymes (AST, ALT, LDH), and bilirubin levels are elevated, and pt develops jaundice

GI	o GI ischemia can cause stress ulcers in stomach = risk for GI bleeding o In small intestine, mucosa can become necrotic and slough off, causing bloody diarrhea o GI ischemia leads to bacterial toxin translocation, in which bacterial toxins enter the bloodstream through the lymphatic system o Net result is interference with healthy cellular functioning and ability to metabolize nutrients
Hematologic	o Inflammatory cytokines activate the clotting cascade, causing deposition of microthrombi in multiple areas & consumption of clotting factors o Disseminated intravascular coagulation (DIC) o Bruises (ecchymoses) and bleeding (petechiae) may appear o Coagulation times (e.g., PT, aPTT) are prolonged

Medical Management

Support respiratory system, optimize intravascular volume, support pumping of heart, improve competence of vascular system

Blood glucose <180

Early enteral nutritional support

Nursing Management

Early interventions

Observe for subtle changes in assessment

Hemodynamic monitoring

EKG, ABG, electrolytes, physical/mental status changes

Rapid/freq administration of meds/fluids

Ventilation, dialysis, IABP

- Prevention of complications
  - Monitor blood levels of meds, aseptic technique during invasive procedures
  - Reduce incidence of VAP
    - Frequent oral care, aseptic suctioning, turning, elevating HOB >30
- Promoting rest and comfort
  - Cluster care
  - Protect temp from extremes
- Supporting family members
  - Keep comfortable and informed
  - Encourage rest
  - Offer visit from chaplain

16. **Irreversible Stage** -- refractory stage, total body failure

Finding	Compensatory	Progressive	Irreversible
Blood Pressure	Normal	Systolic <90 mm Hg; MAP <65 mm Hg Requires fluids resuscitation to support blood pressure	Requires mechanical or pharmacologic support
Heart Rate	>100	>150	Erratic or asystole
Respiratory Status	>20 breaths/min PaCO2 <32 mm Hg	Rapid, shallow respirations; crackles PaO2 <80 mm Hg PaCO2 >45 mm Hg	Requires intubation and mechanical ventilation and oxygenation
Skin	Cold, clammy	Mottled, petechiae	Jaundice
Urinary Output	Decreased	<0.5mL/kg/h	Anuric, requires dialysis
Mentation	Confusion &/or agitation	Lethargy	Unconscious
Acid-Base Balance	Respiratory Alkalosis	Metabolic acidosis	Profound acidosis

BP low, renal/liver failure, complete organ failure

Medical Management

*same as progressive

Judgement can only be determined irreversible retrospectively d/t pt failure to respond to treatment

Abx may be attempted to reduce severity

Nursing Management

Continue prescribed treatment, monitor patient, prevent complications, protect from injury, provide comfort

*same as progressive

17. **Tissue Perfusion**

Monitor for:

Change in LOC, VS, urinary output, skin color/temp

Lab values

Base deficit, lactic acid levels

Serum sodium and blood glucose elevation (d/t release of aldosterone and catecholamines)

Monitor hemodynamic status

SBP <90 or drop of 40 from baseline

MAP <65

Pulse pressure

Systolic - diastolic = pulse pressure

Narrowing pulse pressure (<30-40) indicates shock earlier than drop in BP

18**. Vasoactive Agents Used in Shock**

Medication	Desired Action in Shock	Disadvantages
• Inotropic Agents o Dobutamine o Dopamine o Epinephrine o Milrinone (Primacor)	Improve contractility, increase stroke volume, increase cardiac output	Increase oxygen demand of the heart
• Vasodilators o Nitroglycerin o Nitroprusside	Reduce preload and afterload, reduce oxygen demand of heart	Cause hypotension
• Vasopressor Agents o Norepinephrine (Levophed) o Dopamine o Phenylephrine o Vasopressin	Increase blood pressure by vasoconstriction	Increase afterload, thereby increasing cardiac workload; compromise perfusion to skin, kidneys, lungs, gastrointestinal tract

Fluid replacement

Crystalloids	Advantages	Disadvantages
• Crystalloids o 0.9% sodium chloride (normal saline solution) • o Lactated Ringer's	Widely available, inexpensive Lactate ion that helps buffer metabolic acidosis	Requires large volume of infusion; can cause hypernatremia, pulmonary edema, abdominal compartment syndrome Requires large volume of infusion; can cause metabolic acidosis, pulmonary edema, abdominal compartment syndrome
• Colloids	Rapidly expands plasma volume of crystalloid (or	Expensive; requires human donors; limited supply; can cause heart failure

o Albumin (5%, 25%)	colloid equivalent).	

19**. SIRS**

a. s/s

Temp >101 or <96.8

HR >90 bpm

RR >20, CO2 >32

WBC >12000, <4000, >10% bands

Hypotension (decreased perfusion)

b. Nursing management→

Address high fever

Administer antibiotics

Administer Fluids

Assess for EKG changes

Monitor:

Oxygenation, vitals, EKG, urine output, LCO, skin color/temp/moisture/cap refill/turgor

Explain procedures

IV access

Titrate IV drips to maintain hemodynamic parameters

Monitor central venous pressure, pulmonary artery pressures, cardiac output, pulse pressure

Educate patient, reassure family

20. MODS

a. s/s

Body System	Manifestations
Respiratory	o Severe dyspnea, tachypnea o PaO2/FiO2 ration <200 o Bilateral fluffy infiltrates on CXR o V/Q mismatch o Refractory hypoxemia
Cardiovascular	o Myocardial depression o Massive vasodilation o Decrease SVR, BP, MAP o Increase HR o Biventricular failure
Central Nervous System	o Acute change in neurologic status à confusion, disorientation, delirium o Fever o Seizures o Failure to wean, prolonged rehabilitation
Endocrine System	o Hyperglycemia
Renal System	• Pre-renal: o BUN/Cr rate >20:1 • Intrarenal: o BUN/Cr <10:1
Gastrointestinal System	o Hypoperfusion à decrease peristalsis, paralytic ileus o GI bleeding
Hepatic System	o Bilirubin >2, increased LFTs o Hepatic encephalopathy
Hematologic System	o Coagulopathy (increased PT & PTT, decreased platelet count) o Increased d-dimer

b. Nursing Management

Medical

- Early detection of initial signs of infection
- Controlling initiating event
- Promote adequate organ perfusion
- Provide nutritional support
- Maximize patient comfort

Address end-of-life decisions

Provide information/support to family

Survival

- Rehabilitation goals/expectation
    - Massive loss of skeletal muscle makes rehab a slow, long process