CERTIFICATION REVIEW:
Endocrine and Behavioral

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Content Description

This session will discuss two areas of the test plans for CCRN and PCCN; Endocrine and Behavioral. In the Endocrine portion of the presentation, we will discuss diabetic ketoacidosis (DKA) and Hyperosmolar Hyperglycemic State (HHS), with a focus on identifying similarities and differences in causes, presentation, diagnostics and treatment. Hypoglycemia will be presented with identification of patients at risk, presentation, and treatment. Diabetes Insipidus (DI) and Syndrome of Inappropriate AntiDiuretic Hormone (SIADH) will be discussed, including causes, presentation, diagnosis, and treatment. In the Behavioral portion of the presentation, delirium, dementia, and depression will be discussed, and differentiation made. Anxiety and substance abuse will also be presented. Sample question similar to those on the exams will be presented for both topics.

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

1. Differentiate between Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State
2. Give one example of treatment for hypoglycemia
3. Differentiate between depression, dementia, and delirium

REFERENCES

NOTE: Please refer to outline for references pertaining to this session.
**Endocrine for CCRN and PCCN**

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<thead>
<tr>
<th>CCRN and PCCN</th>
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<tr>
<td>• Acute hypoglycemia</td>
<td>• Hyperosmolar hyperglycemic state</td>
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<td>• Diabetic ketoacidosis</td>
<td>• Diabetes insipidus (DI)</td>
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<td>• Syndrome of inappropriete secretion of antidiuretic hormaon (SIADH)</td>
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*Exam Tip: Approximately 5% of the CCRN and 4% PCCN exams will focus on the endocrine system, approximately 7.5 and 5 questions respectively.*

**I. Diabetic Ketoacidosis**

A. Most serious metabolic disturbance of Type I diabetes. It is a state of insulin deficiency combined with an increase in the level of insulin-antagonistic hormones. The result is altered metabolism of carbohydrate, fat and protein and hyperglycemia.

B. Diagnostic criteria
   1. Blood glucose >250 mg/dl
   2. Arterial pH < 7.3
   3. Serum bicarb < 15 mEq/L
   4. Ketonemia or ketonuria

C. Etiology
   1. Initial presentation of Type I diabetes
   2. Missed or reduced insulin doses
   3. Change in type of insulin or dosage
   4. Growth spurts (adolescents) or sudden weight loss or gain
   5. Stress
   6. Illness
   7. Trauma
   8. Malfunctioning insulin pump
   9. Alcohol or drug use
   10. Medication side effects: glucocorticoids increase gluconeogenesis; thiazide diuretics, diazoxide and phenytoin decrease insulin resistance

D. Pathophysiology
   1. Can develop within 24 hours
   2. Lack of insulin causes cellular glucose uptake, release of free fatty acids and increased gluconeogenesis by the liver.
   3. Glucose unable to enter cells, builds up in blood and hyperglycemia occurs.
   4. Hyperglycemia promotes osmotic diuresis with dehydration (avg fluid loss is 6 liters), hyper osmolality and electrolyte depletion
   5. Ketoacidosis - inability of cells to utilize glucose for fuel results in the breakdown of fat and muscle tissue (lypolyis); fat converted into glucose (gluconeogenesis) and free fatty acids metabolized into ketones by the liver which releases hydrogen ions thereby causing metabolic ketoacidosis.
   6. Acid-base balance:
      Ketones dissociate and yield hydrogen ions
      pH may drop as low as 7.00
Lungs try to compensate by blowing off excessive CO₂
  Rapid and deep – Kussmaul’s
Acidosis causes potassium to come out of cells

E. Clinical manifestations
  1. Blurred vision
  2. Diminished level of consciousness (behavior, affect, confusion, coma)
  3. GI discomfort - nausea, abdominal cramping, vomiting
  4. Dehydration - thirst, hypotension, tachycardia, flushed, dry skin, poor skin turgor, dry mucus membranes
  5. Tachypnea, Kussmaul respirations (if pH is less than 7.2)
  6. Fatigue, weakness, muscle cramps
  7. Polyuria, polydipsia
  8. Acetone odor on breath

F. Laboratory findings
  1. Elevated serum glucose > 250 mg/dl
  2. Metabolic acidosis: pH less than 7.3
  3. Positive results for serum and urine ketones
  4. Azotemia
  5. Hypocalcemia in 30% of patients
  6. Hyperosmolality
  7. Sodium, chloride and bicarbonate decreased
  8. Serum osmolality > 300 mmol/L
  9. Elevated BUN and creatinine in severely dehydrated

G. Management
  1. Airway, breathing and circulation
  2. Provide supplemental oxygen
  3. IV access and fluid replacement is a big priority
     - Infuse 1L bolus of NSS in 1-2 hours
     - Administer IV fluids to correct dehydration based on corrected Na level
       - 0.9 NaCl is recommended if corrected Na level is low
       - 0.45 NaCl is recommended if corrected Na level normal or high
       - 5% dextrose added to prevent hypoglycemia when the glucose level is less than 250 mg/dl
  4. Administer regular insulin in IV bolus and then a continuous infusion. The goal is to decrease serum glucose by 50-75 mg/dl/hr
     - Bolus 0.15 units/kg regular insulin
     - Infusion at 0.1 unit/kg/hr
     - Monitor glucose q 1-2 hr
     - Change IV infusion to D₅ 0.45%NSS when serum glucose < 250 mg/dl to prevent hypoglycemia and cerebral edema
     - Discontinue insulin drip and begin SC insulin when serum glucose normalizes
  5. Acidosis is usually corrected when dehydration and hyperglycemia are corrected. Sodium bicarbonate IV may be given if pH is less than 7.00
  6. Electrolytes
Initial hyperkalemia will change to hypokalemia with correction of dehydration and hyperglycemia
Need to monitor closely
Will need to add potassium to IV
    May add in form of KPO4 if hypophosphatemia is present
7. Patient and family education after patient is stabilized
    Knowledge level
    Compliance
    Diabetic education for newly diagnosed diabetics
    Sick day care

II. Hyperosmolar Hyperglycemic State (HHS)
    Other names: HHNC, HHNK, HONK, HNS,
    A. Potentially lethal complication of Type II diabetes
        1. Dehydration worse than DKA
        2. Serum glucose higher
    B. Diagnostic criteria
        1. Blood glucose > 600 mg/dl
        2. Arterial pH > 7.3
        3. Bicarb > 15 mEq/L
        4. Serum osmolality >320 mmol/L
    C. Etiology
        1. Patients usually older with cardiac disease
        2. Most common cause is infection
            Pneumonia
            UTI
            An open wound
        3. Common medications they are on: steroids, thiazide diuretics, beta-blockers
        4. Institutionalized elderly also at greater risk
    D. Pathophysiology
        1. Occurs over days to weeks
        2. Insulin deficiency
        3. Tissue resistance
        4. Glucagon release from liver increases serum glucose even more
        5. Serum hypersomolality increases
        6. Fluid from cells and interstitial spaces drawn into vascular space in an attempt to return plasma osmolality to normal
        7. Patients often have altered sensorium; do not feel thirst which worsens dehydration (can be as much as 10 liters)
        8. Catecholamines cause release of hormones that promote glucose production
        9. Hemoconcentration increases blood viscosity which increases risk for clotting
        10. Acidosis not usually present
    E. Clinical manifestations
        1. Tachycardia, hypotension
        2. Confusion, seizure, coma, aphasia; it is often the change in mental status that brings the patient to the hospital
3. Polyuria

F. Laboratory findings
1. Serum glucose >600 mg/dl; Often > 1000 mg/dl
2. Hematocrit, creatinine increased
3. Serum osmolality > 330 mmol/L
4. BUN higher than in DKA; may have elevated creatinine
   Kidney impairment due to renal hypoperfusion
5. Potassium and sodium may be decreased
6. pH usually normal

G. Management
1. Fluid therapy
   As with DKA, treatment of dehydration highest priority
   Bolus of 1 liter NSS
   Solution change depends on sodium level
   Switch to 0.45% NSS when sodium is > 140 mEq/L
   If patient is elderly or has CHF hx, will need to monitor closely for fluid overload
2. Insulin therapy as with DKA to correct hyperglycemia
   May require higher doses due to insulin resistance
   If serum glucose does not fall at rate of 50-75 mg/dl/hr, double infusion rate
3. Monitor electrolytes
   May normalize with decrease in serum glucose
   May require replacement
4. Patient and family teaching
   Assess knowledge
   Assess compliance
   Explain diabetes disease process
   Discuss causes of HHS
   S/S to report to physician

**DKA versus HHS**

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<thead>
<tr>
<th></th>
<th>DKA</th>
<th>HHS</th>
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<tbody>
<tr>
<td>Diabetes</td>
<td>Type I</td>
<td>Type II</td>
</tr>
<tr>
<td>Glucose</td>
<td>Average 675 mg/dl</td>
<td>Usually &gt; 1000 mg/dl</td>
</tr>
<tr>
<td>Dehydration</td>
<td>6 liters</td>
<td>10 liters</td>
</tr>
<tr>
<td>Ketones</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>pH</td>
<td>&lt;7.35</td>
<td>Usually normal</td>
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III. Hypoglycemia
A. Decrease in serum glucose level to 50 mg/dl or below. Glucose production lags behind glucose use
B. Etiology and Risk Factors
   1. Those receiving insulin therapy
      Insulin greater than the body requires
Injection sites rotated from hypertrophied area to one with unimpaired absorption
Use of regular insulin can be associated with rapid fall in glucose levels
2. Those receiving oral hypoglycemia therapy
3. Insufficient calorie consumption
4. Strenuous physical activity
5. Excessive alcohol intake which inhibits gluconeogenesis
6. Other significant health issues
   Those taking b-adrenergic blocking agents which can impair recovery from hypoglycemia by inhibiting glycogenolysis
7. Decreased requirements for exogenous insulin from
   Recovery from physiologic stress
   Weight loss
   Immediate post-partum period
   Decrease in steroid use
C. Signs and Symptoms
   1. Headache, fatigue, irritability
   2. Pallor
   3. Hunger
   4. Nervousness
   5. Tachycardia
   6. Diaphoresis
   7. If not treated
      Difficulty speaking and thinking
      Visual disturbances
      Gait disturbances
      Altered LOC and eventual coma
D. Management
   1. Administer oral or IV glucose
      Remeasure serum glucose level 20-30 minutes after tx
      Administer more glucose if needed
      Maintain serum glucose level at 80-110 mg/dl
   2. Mild hypoglycemia
      Blood glucose 60-80 mg/dl
      Patient is completely alert
      Symptoms may include:
      Pallor
      Diaphoresis
      Tachycardia
      Palpation
      Shakiness
   3. Moderate hypoglycemia
      Blood glucose less than 60 mg/dl
      Patient is conscious and able to swallow
      Symptoms may include:
      Difficulty concentrating
      Confusion
Slurred speech
4 Severe hypoglycemia
   . Blood glucose less than 60 mg/dl
   . Patient uncooperative or unconscious
   . Patient requires IV and 50% dextrose
      If unable to obtain IV access – 1 mg SQ glucagon
E. Potential complications
   1. Seizures
   2. Dysrhythmias

IV. Diabetes Insipidus
A. Types
   1. Neurogenic – deficiency of antidiuretic hormone (ADH)
      Intracranial surgery
      Tumors
      Infections
      Severe head trauma
   2. Nephrogenic – insensitivity to ADH
      Renal disease
      Sickle cell disease
      Cystic fibrosis
      Medications: alcohol, dilantin, lithium
B. Presentation
   1. Sudden onset of polyuria
   2. 5-40 liters in 24 hours
   3. May see thirst in conscious patient
C. Diagnosis
   1. Low urine osmolality < 300 mmol/L
   2. High serum osmolality > 295 mmol/L
   3. Decreased urine specific gravity < 1.005
      Unlike Type I and II diabetes, DI has no glucose in urine
   4. Serum sodium > 145 mEq/L
D. DI Treatment
   1. Volume replacement
      Hypotonic solution
   2. Neurogenic DI: replace ADH
      IV, IM, SC, intranasal
      Desmospressin (DDAVP) drug of choice
   3. Nephrogenic: Thiazide diuretics
      Discontinue drugs responsible

V. Syndrome of Inappropriate Secretion of antidiuretic Hormone (SIADH)
A. Description
1. Opposite of DI
2. Antidiuretic hormone (ADH or vasopressin) is secreted into the blood stream in excess of the amount needed to maintain blood volume and serum osmolality
3. Excessive water absorbed in renal tubules
4. Sodium is diluted

B. Causes
1. Most common cause is malignant bronchogenic oat cell carcinoma
   Malignant cell synthesizes and releases ADH
   Also can be from pancreatic, prostatic, and duodenal cancers
   Hodgkin disease and leukemia have also been implicated
2. CNS injury or disease
3. Medications
   Hypoglycemics
   Potassium-depleting thiazide diuretics
   Tricycle antidepressants
   Chemotherapeutic agents
   Narcotics

C. Assessment findings
1. Urine output < 0.5 ml/kg/hr; concentrated
2. Lethargy, N/V
3. Labs
   Serum osmolality <250 mOsm/kg
   Urine osmolality increased; equal to or greater than serum osmolality
   Serum sodium < 120 mEq/L; urine sodium > 20 mEq/L

D. Collaborative management
1. Treatment of primary disease
2. Fluid restriction 500 ml < average daily UO
3. Sodium replacement
   Hypertonic sodium solution 3% - 5%
   Increase serum sodium no more than 12 mEq/day or a to 2 mEq/L/hr to prevent neurologic complications
4. May administer furosemide
5. Medications that inhibit tubule response to ADH
   Demeclocycline (Declomycin), Lithium
6. Monitor hydration
   Close, frequent measurement of I&O
   Blood and urine sodium levels
   Urine specific gravity
   Urine and blood osmolality
   Weights every 12 hours
7. Monitor neuro status every 1-2 hours
8. Low-volume hypertonic enemas for constipation
   Avoid tap water – can increase water intoxication
9. Patient/family education

Certification Questions
1. Which intervention is appropriate for a patient with DKA?
A. Decrease the insulin IV infusion rate when the blood glucose level reaches 180 mg/dl
B. Switch from 0.45% sodium chloride solution to a glucose-containing fluid when the blood glucose level reaches 250 mg/dl
C. Administer insulin preparations subcutaneously when the blood glucose level reaches 200 mg/dl
D. Provide intermediate-acting insulin when the blood glucose level returns to 180 mg/dl

2. A patient with Type II diabetes was found unresponsive, sitting at his desk at work. An admitting diagnosis of Hyperosmolar, Hyperglycemic State (HHS) was made in the emergency department. The nurse assigned to this patient anticipates his highest priority needs by preparing:
   A. Large amounts of intravenous normal saline for infusion
   B. An insulin drip made with glargine insulin for infusion
   C. Oxygen at 40% via mask
   D. Padded side rails and a bite block

3. The patient’s wife asks how her husband could have developed diabetic ketoacidosis, since he has been so careful to manage his diabetes properly these past 10 years. Which of the following represents the best explanation the nurse could provide to this question?
   A. “Your husband must have been cheating on his diet, or skipping insulin doses.”
   B. “In times of stress, the body produces more cortisol than normal, and blood sugar increase.”
   C. “Diabetic ketoacidosis is the result of a genetic abnormality affecting every other generation.”
   D. “Your husband recently started a daily exercise routine without consulting his primary care providers.”

4. A patient exhibits dizziness, incoherent speech, diaphoresis and combative behavior symptomatic of:
   A. DKA
   B. HHS
   C. Moderate hypoglycemia
   D. Severe hypoglycemia

5. A patient with a blood glucose of less than 60 mg/dl who is conscious should receive:
   A. 15 g carbohydrate by mouth
   B. 30 g carbohydrate by mouth
   C. One ampule D50 IV
   D. One mg glucagon SQ

6. A patient had a craniotomy 2 days ago for removal of a tumor. He is awake and talking to the nurse and demonstrates no neurologic deficit. Blood pressure is 110/80 mmHg, pulse is 92 bpm, and respiratory rate is 22 breaths/min. Urine outputs have been approximately 60 ml/hr over the last 2 days, but he has had a recent change.
He has had 300-400 ml/hr of urine output over the last several hours. The urine has a specific gravity of 1.002. The nurse checks his serum glucose and finds that it is 100 mg/dl. The IV solution most appropriate for fluid replacement would be:

A. 5% dextrose in water (D5W)
B. Normal saline
C. Lactated Ringer’s solution
D. 10% dextrose in water (D10W)

7. The cause of hypernatremia in this patient is:
A. Sodium retention
B. Water loss
C. Water gain
D. Aldosterone excess

8. A patient who has been battling oat cell lung carcinoma is admitted for chemotherapy. The nurse observes that the patient is lethargic. His urine output has been 5 – 15 ml/hr for the last four hours, and is concentrated. The nurse requests an order from the physician for confirmatory lab tests for SIADH. Which of the following lab tests would indicate SIADH?
A. Serum and urine sodium elevated, serum and urine osmolality decreased
B. Serum and urine sodium decreased, serum and urine osmolality elevated
C. Serum sodium decreased, urine sodium elevated, serum osmolality decreased and urine osmolality elevated
D. Serum sodium elevated, urine sodium decreased, serum osmolality elevated and urine osmolality decreased

9. Patients with SIADH may be treated with:
A. 3% sodium IV infusion
B. 0.9% sodium IV infusion
C. D5 ½ NS IV infusion
D. D5W IV infusion

Behavioral for CCRN and PCCN

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<td>- Abuse/neglect</td>
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<tr>
<td>- Mood disorders and depression</td>
<td>- Antisocial behaviors, aggression, violence</td>
</tr>
<tr>
<td>- Substance abuse</td>
<td>- Developmental delays</td>
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Exam Tip: Approximately 4% of both the CCRN and PCCN exam will focus on the behavioral aspects of care, approximately 6 and 5 questions respectively.

I. Delirium and dementia
A. Dementia
Description
Decline in intellectual functioning that is severe enough to interfere with the ability to perform routine activities
Loss of cognitive abilities leads to impairments in memory, reasoning, planning, and personality
Most people with dementia are elderly
Not an inevitable part of aging

Causes
Alzheimer's disease (AD) most common cause
Vascular or multi-infarct dementia.

B. Delirium
Description
Behavioral disorder characterized by disorientation, confusion, perceptual disturbances, restlessness, distractibility, and sleep-wake cycle disturbances.
Patients with dementia are at higher risk of developing delirium

Causes
Most common in postsurgical and elderly patients
ICU stay also puts patient at risk
Metabolic disturbances, poly pharmacy, immobility, infections, dehydration, electrolyte imbalances, sensory impairments may all be factors in development of delirium

Collaborative interventions
Prevention is key

Environmental modifications
Reorientation techniques or memory cues such as a calendar, clocks, and family photos may be helpful
Minimize stimulation
Minimize television noise
The environment should be stable, quiet, and well-lighted
Support from a familiar nurse and family should be encouraged.
Family members and staff should explain proceedings at every opportunity, reinforce orientation, and reassure the patient.
Have patient repeat information
Sensory deficits should be corrected, if necessary, with eyeglasses and hearing aids.
Physical restraints should be avoided
Delirious patients may pull out intravenous lines, climb out of bed, and may not be compliant
Perceptual problems lead to agitation, fear, combative behavior, and wandering
Do not confront if behavior is bizarre
Severely delirious patients benefit from constant observation
May be cost effective for these patients and help avoid the use of physical restraints. These patients should never be left alone or unattended.

Medications
Sedative-hypnotics, anxiolytics can precipitate delirium
Low-dose neuroleptic such as haloperidol (Haldol)
Short-acting benzodiazepines, such as lorazepam (Ativan)
Most likely will resolve with time, resumption of normal sleep patterns, and medications
Patients usually remember
May be embarrassed

II. Mood disorders and depression
A. Common mood disorders are bipolar disorder and depression
Bipolar disorder most likely not on exam
B. Depression
Description
Mental state characterized by a pessimistic sense of inadequacy and a despondent lack of activity
Causes
Medical illness
Social isolation
Pessimism
Psychologic history
Collaborative interventions
Education regarding depression in medical illness
Common after diagnosis
Usually temporary
Encourage patient to discuss feelings
Correct/clarify negative distortions regarding situation
Reassure with realistic information
Consistency in care providers promotes trust
Medications, psychiatric consult
if severe
C. Anxiety
Not listed as such in blueprint, but may be consider a mood disorder
Due to hospitalization, diagnosis, procedures, unfamiliar routines
May be exacerbated by pre-existing anxiety disorder
Presentation
Excessive, ongoing worry and tension
Unrealistic view of problems
Restlessness, irritability
Difficulty concentrating
Trouble falling or staying asleep
Collaborative interventions
Breathing techniques
III. Substance abuse

A. Description
Overindulgence in and dependence of a drug or other chemical leading to effects that are detrimental to the individual's physical and mental health, or the welfare of others
Patients with history of substance abuse at risk for increased LOS, higher infections rates, ARDS, acute delirium and restraint use
Estimated that 75% of patients with substance abuse also have a psychiatric disorder

B. Presentation
Related to substance being abused
May be confounded by psychiatric disorder
Agitated, restless, anxious, depressed, combative
Alcohol withdrawal syndrome (AWS)
   Shaking, paranoid behavior, seizures, death
May be difficult to identify

C. Collaborative interventions
History and physical
Ongoing assessments
   AWS may not be symptomatic until 72-96 hours after last drink
Psychiatric consult
May require higher than usual doses of opioids and anxiolytics
Alcohol abuse requires folic acid and thiamine

Certification Question
1. The nurse notes that a 42-year-old patient admitted with pancreatitis two days ago is becoming restless and agitated. The nurse knows that the patient’s pancreatitis was caused by a two-day drinking “binge.” The nurse anticipates that the physician will order:
   A. Haloperidal (Haldol)
   B. Close observation
   C. Restraints
   D. Lorazepam (Ativan)

2. A patient scheduled to undergo cardiac catheterization tells the nurse, “I don’t think I’ll be able to sleep tonight.” The patient refuses the ordered sleeping medication. Which of the following should the nurse suggest to enhance sleep?
   A. Counting sheep
   B. Imagery
   C. Watching television
Mrs. Banks, 86, is admitted for pneumonia. She has a PMH of Type 2 diabetes and hypertension. During the day, she is alert, oriented, and cooperative. In the late evening and early morning, she is confused and pulling at her oxygen and IV lines. The nurse’s initial response would be to:

A. Administer haloperidol (Haldol) per PRN order
B. Place the patient in soft wrist restraints
C. Optimize sleep-wake cycles by controlling lighting and noise
D. Assign the Unlicensed Assistive Personnel (UAP) to constant observation of the patient

Bibliography


