



**Dr. Rossi's**

# **PMHNP**

**EXAM UPDATES: OCTOBER 2025**

## PMHNP Certification Exam Tips October 2025

Clarity Education Systems

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## Ensuring Quality and Patient Safety in Psychiatric Environments

Ensuring quality and patient safety in psychiatric environments requires a multifaceted approach that integrates evidence-based practice, regulatory compliance, and interdisciplinary leadership. *PMHNPs, as advanced practice nurses, play a critical role in navigating healthcare systems, advocating for policy improvements, and implementing innovative care models to enhance mental health outcomes.* Effective communication and collaboration are essential for building partnerships and driving systemic change, particularly for diverse patient populations with complex psychiatric needs. This review details the strategies, regulatory frameworks, and leadership responsibilities PMHNPs must employ to safeguard patient safety and improve care quality.

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### Major Points

#### 1. Integrating Research and Scholarly Activities:

- **Role of Research:** PMHNPs utilize evidence-based research to inform clinical practice, improving treatment efficacy and safety (e.g., implementing trauma-informed care models).
- **Scholarly Activities:** Conducting or disseminating research (e.g., publishing in journals, presenting at conferences) translates findings into practice improvements.
- **Impact on Outcomes:** Research-driven interventions (e.g., CBT for PTSD) reduce adverse events.
- **PMHNP Responsibilities:**
  - Stay updated on literature (e.g., *Journal of Psychiatric and Mental Health Nursing*).
  - Implement evidence-based protocols (e.g., standardized suicide risk assessment with C-SSRS).
  - Share findings with interdisciplinary teams to standardize best practices.

#### 2. Navigating Regulatory Frameworks and Policy Environments:

- **Key Regulations:**
  - **The Joint Commission:** Sets standards for patient safety in psychiatric facilities (e.g., medication reconciliation, restraint reduction).
  - **CMS Conditions of Participation:** Ensure quality care in Medicare/Medicaid-funded settings.
  - **Mental Health Parity Act:** Mandates equal coverage for mental health services, impacting access.
- **Policy Advocacy:** PMHNPs advocate for policies enhancing access (e.g., telepsychiatry funding) and reducing disparities (e.g., for underserved populations).

- **Innovative Care Models:**
  - Collaborative care models integrate psychiatric and primary care, improving outcomes (~30% better depression remission).
  - Trauma-informed care addresses diverse needs, reducing retraumatization risks.
- **PMHNP Relevance:** Understand regulations to ensure compliance; advocate for equitable care access.

### 3. Communication and Collaboration Skills:

- **Effective Communication:**
  - Articulate patient needs to stakeholders (e.g., policymakers, insurers) to secure resources.
  - Use active listening and empathy (e.g., Client-Centered Therapy principles) to engage patients.
- **Collaboration:**
  - Build interdisciplinary teams (e.g., with social workers, psychologists, primary care) to address social determinants of health (SDOH).
  - Partner with community organizations to enhance support (e.g., housing programs for SUD patients).
- **Impact:** Interdisciplinary initiatives reduce hospital readmissions (~25% lower).
- **PMHNP Relevance:** Lead team-based care, facilitate stakeholder dialogue, and advocate for patient-centered policies.

### 4. Ensuring Patient Safety:

- **Strategies:**
  - Implement standardized protocols (e.g., suicide risk screening, medication error prevention).
  - Monitor adverse events (e.g., EPS from antipsychotics, falls in elderly).
  - Use quality improvement tools (e.g., Plan-Do-Study-Act cycles) to address safety gaps.
- **Specific Populations:**
  - **Children/Adolescents:** Screen for SUD, bullying (e.g., CRAFFT); ensure safe medication use.
  - **Adults:** Address dual diagnosis with integrated treatment (e.g., CBT + MAT).

- **Elderly:** Prevent falls, manage polypharmacy in dementia care.
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## Safety Issues

### 1. Adverse Events:

- Medication errors (e.g., antipsychotics causing EPS) or inadequate suicide screening increase risks.
- **Mitigation:** Use electronic health records (EHR) for medication reconciliation; implement C-SSRS.

### 2. Health Disparities:

- Underserved populations face barriers to quality care (e.g., ~40% lower access for minorities).
- **Mitigation:** Advocate for telepsychiatry, culturally competent care.

### 3. Regulatory Non-Compliance:

- Failure to meet Joint Commission standards risks patient harm (e.g., improper restraint use).
- **Mitigation:** Train staff on regulations; conduct audits.

### 4. Burnout and Communication Breakdown:

- Poor interdisciplinary collaboration increases errors (~20% higher in fragmented teams).
  - **Mitigation:** Foster team communication; provide PMHNP self-care resources.
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## High-Yield Information

### • Key Features:

- Quality/Safety: Integrate research, comply with regulations, lead interdisciplinary teams.
- PMHNP Leadership: Advocate for policy, implement innovative care models.

### • Applications:

- Diagnostic: Use standardized tools (e.g., C-SSRS, CRAFFT) for safety assessment.
- Therapeutic: Implement evidence-based protocols; collaborate with stakeholders.
- Preventive: Reduce adverse events through audits, training (~15% lower errors).

- **Exam Pearls:**

- Questions test regulatory knowledge (e.g., Joint Commission), advocacy, and safety protocols.
  - PMHNPs lead quality improvement, policy advocacy.
  - Collaborative care models are evidence-based for mental health.
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### **Role of the PMHNP**

- **Assessment:** Use standardized tools (e.g., C-SSRS) to identify safety risks; assess SDOH.
  - **Intervention:** Implement evidence-based care models; advocate for policy changes.
  - **Education:** Train staff on safety protocols; educate patients on treatment access.
  - **Advocacy:** Lead initiatives for equitable care, telepsychiatry access.
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**Question:** A PMHNP leads a quality improvement initiative in a psychiatric facility after noting increased medication errors. Patients include diverse populations with limited care access. Which of the following is the most appropriate initial action to ensure quality and patient safety?

- A. Prescribe lower-dose antipsychotics to reduce side effects.
- B. Conduct a patient satisfaction survey and reassess in 3 months.
- C. Implement EHR-based medication reconciliation and advocate for telepsychiatry access.
- D. Refer all patients for individual CBT to improve adherence.

**Answer: C. Implement EHR-based medication reconciliation and advocate for telepsychiatry access:** Medication errors threaten patient safety, and EHR-based reconciliation reduces errors by ~15%. Advocating for telepsychiatry addresses access disparities for diverse populations, aligning with PMHNP leadership in quality improvement and policy advocacy. This approach ensures both safety and equity, a core PMHNP exam skill.

**Why It's High-Yield:** Tests quality improvement and advocacy, key PMHNP competencies.

- **A. Prescribe lower-dose antipsychotics to reduce side effects:** Lower doses may reduce side effects but don't address systemic medication errors or access issues. Systemic solutions are priority. **Exam Tip:** Quality improvement requires systemic interventions, not individual prescribing changes.
- **B. Conduct a patient satisfaction survey and reassess in 3 months:** Surveys assess experience but delay action on urgent safety issues like medication errors. EHR and

advocacy are more immediate. **Exam Tip:** Safety issues require proactive interventions, not delayed assessments.

- **D. Refer all patients for individual CBT to improve adherence:** CBT improves adherence but doesn't address medication errors or access disparities. Systemic safety measures are needed first. **Exam Tip:** Quality initiatives prioritize systemic fixes over therapy referrals.

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## The Amygdala in Psychiatric Disorders

The amygdala, a key structure within the limbic system, is critical for processing emotions, particularly fear and pleasure, and modulating emotional memories in collaboration with the hippocampus. Dysregulation of amygdala activity is implicated in psychiatric disorders such as anxiety disorders, post-traumatic stress disorder (PTSD), and depression, where heightened fear responses, intrusive memories, or altered emotional processing predominate. For PMHNPs, understanding the amygdala's role is essential for assessing and managing these conditions, integrating neurobiological insights with evidence-based interventions to improve patient outcomes.

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### Major Points

#### 1. Amygdala: Structure and Function:

- **Anatomy:** The amygdala is an almond-shaped cluster of nuclei in the medial temporal lobe, part of the limbic system, with connections to the hippocampus, prefrontal cortex, and hypothalamus.
- **Functions:**
  - **Emotional Processing:** Regulates fear, anxiety, and pleasure responses; critical for threat detection and emotional salience.
  - **Memory Modulation:** Works with the hippocampus to encode and retrieve emotionally charged memories (e.g., fear-based memories in trauma).
  - **Stress Response:** Activates the hypothalamic-pituitary-adrenal (HPA) axis, increasing cortisol during stress.
- **Neurotransmitter Involvement:** Modulated by GABA (inhibitory), glutamate (excitatory), and serotonin (mood regulation).

#### 2. Role in Psychiatric Disorders:

- **Anxiety Disorders:**

- Amygdala hyperactivity leads to exaggerated fear responses, hypervigilance, and avoidance (e.g., in GAD, social anxiety).
- Example: Increased amygdala activation to social cues in social anxiety disorder.
- **Post-Traumatic Stress Disorder (PTSD):**
  - Hyperactive amygdala contributes to intrusive memories, flashbacks, and hyperarousal; impaired prefrontal inhibition fails to regulate fear responses.
  - Example: Amygdala overactivation during trauma cues.
- **Depression (Major Depressive Disorder, MDD):**
  - Altered amygdala activity disrupts emotional processing, leading to persistent sadness or anhedonia; increased response to negative stimuli.
  - Example: ~20% increased amygdala volume in chronic MDD.
- **Other Disorders:** Amygdala dysregulation implicated in schizophrenia (emotional lability), bipolar disorder (manic impulsivity), and borderline personality disorder (emotional reactivity).
- **Prevalence:** Amygdala dysfunction present in ~70% of PTSD cases, ~50% of anxiety cases, and ~40% of MDD cases.

### 3. Assessment and Clinical Implications:

- **Assessment:**
  - **Clinical Interview:** Evaluate emotional symptoms (e.g., fear, sadness, hyperarousal) and trauma history.
  - **Standardized Tools:**
    - **PCL-5:** Assesses PTSD symptoms (intrusive memories, hyperarousal).
    - **GAD-7/PHQ-9:** Screens for anxiety/depression symptoms linked to amygdala dysfunction.
    - **C-SSRS:** Assesses suicide risk, heightened in amygdala-related disorders (~15% ideation in PTSD/MDD).
  - **Neuroimaging:** Functional MRI (fMRI) shows amygdala hyperactivity; not routine but supports research-based diagnosis.
- **PMHNP Relevance:**
  - Identify amygdala-driven symptoms (e.g., fear responses in anxiety) to tailor interventions.

- Coordinate with neurology for atypical presentations (e.g., rule out seizures mimicking emotional lability).

#### 4. Management Strategies:

- **Pharmacotherapy:**

- **SSRIs/SNRIs:** Sertraline, duloxetine normalize amygdala activity in anxiety/MDD; reduce hyperarousal in PTSD (e.g., sertraline 50–200 mg/day, FDA-approved for PTSD).
- **Benzodiazepines:** Avoid in PTSD due to risk of worsening memory processing; use sparingly in anxiety.
- **Antipsychotics:** Low-dose risperidone for schizophrenia-related emotional dysregulation.

- **Psychotherapy:**

- **CBT:** Reduces amygdala hyperactivity by enhancing prefrontal control (~50% symptom reduction).
- **EMDR:** Targets amygdala-driven intrusive memories in PTSD (~60% remission).
- **Mindfulness-Based Therapies:** Decrease amygdala reactivity in anxiety/depression.

- **Supportive Interventions:** Address social determinants of health (e.g., safe housing) to reduce stress-related amygdala activation.

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### Safety Issues

#### 1. Suicide Risk:

- Amygdala-driven emotional dysregulation increases suicidality in PTSD, MDD (~15–20% ideation risk).
- **Mitigation:** Screen with C-SSRS; implement safety plans, hospitalize if high risk.

#### 2. Medication Side Effects:

- SSRIs risk agitation in <25 years (FDA black box); antipsychotics cause EPS in schizophrenia.
- **Mitigation:** Start low, monitor with GAD-7/PHQ-9, use AIMS for EPS.

#### 3. Misdiagnosis:

- Amygdala-related symptoms (e.g., fearfulness) may be mistaken for primary anxiety vs. PTSD or neurological issues (e.g., temporal lobe epilepsy).

- **Mitigation:** Use PCL-5, neuroimaging if indicated, and seizure history.

#### 4. Overwhelm in Therapy:

- EMDR or CBT targeting amygdala-driven memories may temporarily increase distress.
  - **Mitigation:** Ensure patient stability with coping skills before trauma-focused therapy.
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### High-Yield Information

- **Key Features:**

- Amygdala: Emotional processing (fear, pleasure), memory modulation, HPA axis activation.
- Dysregulation linked to anxiety, PTSD, depression, schizophrenia.

- **Applications:**

- Diagnostic: Use PCL-5, GAD-7, PHQ-9 to assess amygdala-related symptoms.
- Therapeutic: SSRIs, CBT, EMDR to normalize amygdala activity.
- Preventive: Early intervention reduces chronicity (~40% lower relapse in PTSD).

- **Exam Pearls:**

- Amygdala hyperactivity drives fear/hyperarousal in anxiety/PTSD; altered in MDD.
  - Questions test neurobiological basis and treatment selection.
  - Avoid benzodiazepines in PTSD due to amygdala interference.
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### Role of the PMHNP

- **Assessment:** Screen for amygdala-related symptoms with PCL-5, GAD-7, C-SSRS.
  - **Intervention:** Prescribe SSRIs/SNRIs, refer for CBT/EMDR, coordinate with neurology for atypical cases.
  - **Education:** Explain amygdala's role in emotions to patients/families.
  - **Advocacy:** Promote access to trauma-focused therapies and integrated care.
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**Question:** A 35-year-old female with PTSD presents with frequent flashbacks, hypervigilance, and avoidance of trauma cues 1 year after a car accident. She denies suicidality. Neuroimaging suggests amygdala hyperactivity. According to DSM-5-TR and neurobiological principles, which of the following is the most appropriate intervention to address her symptoms?

- A. Prescribe lorazepam 0.5 mg PRN for hypervigilance.
- B. Continue supportive therapy and reassess in 4 weeks.
- C. Initiate sertraline 25 mg daily and refer for EMDR therapy.
- D. Start risperidone 0.5 mg daily for emotional dysregulation.

**Correct Answer: C. Initiate sertraline 25 mg daily and refer for EMDR therapy:** The patient's PTSD symptoms (flashbacks, hypervigilance, avoidance) and amygdala hyperactivity indicate a need for targeted treatment. Sertraline, an FDA-approved SSRI for PTSD, reduces amygdala-driven hyperarousal (50–200 mg/day), and EMDR addresses intrusive memories by reprocessing trauma, achieving ~60% remission. This integrated approach aligns with PMHNP priorities for evidence-based PTSD management.

**Why It's High-Yield:** Tests amygdala-targeted treatments (SSRIs, EMDR) for PTSD, a core PMHNP exam skill.

- **A. Prescribe lorazepam 0.5 mg PRN for hypervigilance:** Benzodiazepines risk dependence and impair trauma memory processing in PTSD, worsening amygdala dysfunction, per APA guidelines. Sertraline and EMDR are preferred. **Exam Tip:** Avoid benzodiazepines in PTSD due to amygdala interference.
- **B. Continue supportive therapy and reassess in 4 weeks:** Supportive therapy is less effective for PTSD than trauma-focused interventions like EMDR. Delaying targeted treatment risks symptom persistence. **Exam Tip:** PTSD requires specific therapies (EMDR, CBT) over general support.
- **D. Start risperidone 0.5 mg daily for emotional dysregulation:** Risperidone is not indicated for PTSD without psychosis; it risks EPS and doesn't address amygdala-driven trauma symptoms. Sertraline/EMDR are first-line. **Exam Tip:** Antipsychotics are inappropriate for non-psychotic PTSD.

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### Subcortical Dysfunction in Neurological and Psychiatric Disorders

Subcortical dysfunction, involving structures like the substantia nigra and striatum, is primarily linked to motor control but also significantly impacts mood regulation and cognitive processes due to disruptions in dopaminergic and other neurotransmitter pathways. Disorders such as Parkinson's disease and Huntington's disease exhibit hallmark motor symptoms (e.g., tremors, chorea) alongside psychiatric manifestations, including depression, anxiety, and cognitive

decline. For PMHNPs, understanding subcortical dysfunction is critical for assessing and managing patients with these overlapping neurological and psychiatric symptoms, coordinating interdisciplinary care, and ensuring patient safety.

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## Major Points

### 1. Subcortical Structures and Functions:

- **Key Structures:**
  - **Substantia Nigra:** Produces dopamine via the nigrostriatal pathway, regulating motor control and reward.
  - **Striatum (Caudate, Putamen):** Modulates motor planning, executive function, and emotional processing within corticostriatal circuits.
  - **Other structures:** Globus pallidus, subthalamic nucleus (part of basal ganglia).
- **Functions:**
  - **Motor Control:** Coordinates voluntary movements, gait, and posture.
  - **Mood Regulation:** Influences emotional responses via dopamine and serotonin pathways.
  - **Cognitive Processes:** Supports executive function, attention, and memory through corticostriatal loops.
- **Neurotransmitters:**
  - **Dopamine:** Critical for reward, motivation, and movement; dysregulation central to Parkinson's and Huntington's.
  - **GABA/Glutamate:** Balances excitatory/inhibitory signals in subcortical circuits.

### 2. Role in Neurological and Psychiatric Disorders:

- **Parkinson's Disease:**
  - **Pathophysiology:** Dopamine loss in substantia nigra (nigrostriatal pathway) causes bradykinesia, rigidity, tremors.
  - **Psychiatric Symptoms:** Depression (~40% prevalence), anxiety (~30%), cognitive decline (e.g., executive dysfunction in ~50% by 5 years).
  - **Mechanism:** Dopamine depletion disrupts corticostriatal mood regulation; Lewy body pathology affects subcortical circuits.
- **Huntington's Disease:**

- **Pathophysiology:** Striatal degeneration (due to HTT gene mutation) causes chorea, dystonia.
  - **Psychiatric Symptoms:** Depression (~50%), anxiety (~20%), irritability, cognitive decline (e.g., memory loss in ~60% by mid-stage).
  - **Mechanism:** Loss of GABAergic neurons in striatum disrupts emotional and cognitive processing.
- **Other Disorders:**
    - **Schizophrenia:** Dopamine dysregulation in corticostriatal pathways contributes to psychosis, motor symptoms (e.g., catatonia).
    - **Antipsychotic Side Effects:** D2 receptor blockade causes extrapyramidal symptoms (EPS) like parkinsonism (~40% risk).
  - **Prevalence:** Psychiatric symptoms in ~70% of Parkinson's and ~80% of Huntington's cases.

### 3. Assessment and Clinical Implications:

- **Assessment:**
  - **Clinical Interview:** Evaluate motor symptoms (tremors, rigidity), mood (depression, anxiety), and cognition (memory, executive function).
  - **Standardized Tools:**
    - **Unified Parkinson's Disease Rating Scale (UPDRS):** Assesses motor and non-motor symptoms in Parkinson's.
    - **Huntington's Disease Rating Scale (UHDRS):** Evaluates motor, cognitive, and psychiatric symptoms.
    - **PHQ-9/GAD-7:** Screens for depression/anxiety.
    - **MoCA:** Assesses cognitive decline in subcortical disorders.
    - **C-SSRS:** Evaluates suicide risk (~15% ideation in Parkinson's/Huntington's).
  - **Neuroimaging:** MRI shows striatal atrophy in Huntington's; PET/FDG scans detect dopamine loss in Parkinson's.
- **PMHNP Relevance:**
  - Differentiate subcortical-driven psychiatric symptoms from primary disorders (e.g., MDD vs. Parkinson's depression).
  - Coordinate with neurology for diagnostic confirmation and treatment planning.

### 4. Management Strategies:

- **Pharmacotherapy:**
    - **Parkinson's:** Levodopa/carbidopa (100–1000 mg/day) for motor symptoms; SSRIs (e.g., sertraline 50–200 mg/day) for depression/anxiety.
    - **Huntington's:** Tetrabenazine (12.5–50 mg/day) for chorea; SSRIs for depression; low-dose antipsychotics (e.g., quetiapine) for agitation.
    - **Schizophrenia:** Atypical antipsychotics (e.g., aripiprazole) to minimize EPS; monitor with AIMS.
  - **Psychotherapy:**
    - **CBT:** Addresses depression/anxiety in Parkinson's/Huntington's (~40% symptom reduction).
    - **Supportive Therapy:** Enhances coping for progressive disorders.
  - **Supportive Interventions:** Physical therapy for motor deficits; family counseling for caregiver burden.
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## Safety Issues

### 1. Suicide Risk:

- Depression in Parkinson's/Huntington's increases suicidality (~15–20% ideation risk).
- **Mitigation:** Screen with C-SSRS; implement safety plans or hospitalization.

### 2. Medication Side Effects:

- Levodopa risks dyskinesia; antipsychotics cause EPS (~40% risk in schizophrenia); SSRIs risk agitation in <25 years.
- **Mitigation:** Use low-dose atypicals; monitor with AIMS, ECG for QT prolongation.

### 3. Falls and Motor Risks:

- Parkinson's rigidity or Huntington's chorea increases fall risk (~30% annually in Parkinson's).
- **Mitigation:** Physical therapy, home safety assessments.

### 4. Misdiagnosis:

- Subcortical psychiatric symptoms (e.g., depression) may be mistaken for primary disorders, delaying neurological care.
- **Mitigation:** Use UPDRS/UHDRS, neuroimaging to confirm subcortical etiology.

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## High-Yield Information

- **Key Features:**
  - Subcortical dysfunction: Affects motor (tremors, chorea), mood (depression, anxiety), cognition (executive decline).
  - Disorders: Parkinson's, Huntington's, schizophrenia; dopamine dysregulation central.
- **Applications:**
  - Diagnostic: Use UPDRS, UHDRS, MoCA for subcortical symptoms; MRI/PET for confirmation.
  - Therapeutic: Levodopa for Parkinson's; SSRIs, atypicals for psychiatric symptoms.
  - Preventive: Monitor EPS, falls to reduce complications.
- **Exam Pearls:**
  - Subcortical dysfunction causes motor and psychiatric symptoms; distinguish from cortical (e.g., Alzheimer's).
  - Questions test EPS management, differential diagnosis.
  - Atypical antipsychotics reduce EPS risk vs. typicals.

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## Role of the PMHNP

- **Assessment:** Use UPDRS, UHDRS, PHQ-9, and MoCA to evaluate subcortical symptoms.
- **Intervention:** Prescribe SSRIs, atypicals; coordinate with neurology for motor therapies.
- **Education:** Explain neurological basis of psychiatric symptoms to patients/families.
- **Advocacy:** Promote integrated psychiatric-neurological care for complex cases.



**Question:** A 60-year-old male with Parkinson's disease presents with tremors, depression, and executive dysfunction. He reports sadness and anhedonia, denying suicidality. UPDRS confirms motor deficits, and MoCA shows cognitive decline. According to DSM-5-TR and

neurobiological principles, which of the following is the most appropriate intervention for his psychiatric symptoms?

- A. Initiate sertraline 25 mg daily and refer for CBT.
- B. Start haloperidol 1 mg daily for mood stabilization.
- C. Increase levodopa/carbidopa to address depression.
- D. Prescribe lorazepam 0.5 mg PRN for emotional lability.

**Answer: A. Initiate sertraline 25 mg daily and refer for CBT:** The patient's depression and executive dysfunction in Parkinson's disease reflect subcortical dysfunction, particularly dopamine depletion in corticostriatal pathways. Sertraline, an SSRI, is effective for Parkinson's-related depression (~40% symptom reduction), and CBT addresses mood and coping, aligning with PMHNP priorities for integrated psychiatric care.

**Why It's High-Yield:** Tests treatment of subcortical psychiatric symptoms, a core PMHNP exam skill.

- **B. Start haloperidol 1 mg daily for mood stabilization:** Haloperidol, a typical antipsychotic, worsens Parkinson's motor symptoms due to D2 blockade, increasing EPS risk (~40%). It's inappropriate for depression. **Exam Tip:** Avoid typical antipsychotics in Parkinson's.
- **C. Increase levodopa/carbidopa to address depression:** Levodopa treats motor symptoms in Parkinson's but has limited efficacy for depression, which requires targeted SSRI/CBT intervention. **Exam Tip:** Levodopa is for motor, not psychiatric, symptoms.
- **D. Prescribe lorazepam 0.5 mg PRN for emotional lability:** Benzodiazepines risk dependence and falls in elderly Parkinson's patients (~30% fall risk). Sertraline/CBT is safer and more effective. **Exam Tip:** Avoid benzodiazepines in neurodegenerative disorders.

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## Early Childhood Development of Sensory Pathways and Language Skills

The early years of life (infancy to early childhood) are critical for developing sensory pathways (vision, hearing) and language skills, driven by neural plasticity in the auditory and visual cortices. Adequate sensory and linguistic input during these critical periods is essential for normal development, and disruptions can lead to neurodevelopmental disorders (e.g., autism spectrum disorder, language disorders). For PMHNPs, understanding these developmental processes is vital for early identification of delays, coordinating interventions, and supporting families to optimize outcomes in pediatric psychiatric care.

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## Major Points

1. **Critical Periods in Sensory and Language Development:**

- **Definition:** Critical periods are time-sensitive windows during early development when neural circuits are highly plastic, requiring specific stimuli to mature properly.
- **Vision:**
  - **Critical Period:** Birth to ~3–5 years; peaks in first 6–12 months.
  - **Visual Cortex:** Located in the occipital lobe, processes visual stimuli (e.g., shapes, colors) via input from the retina through the lateral geniculate nucleus.
  - **Mechanism:** Visual input shapes synaptic connections; deprivation (e.g., congenital cataracts) leads to amblyopia or visual processing deficits.
  - **Clinical Relevance:** Early visual deficits increase risk for sensory processing issues in autism (~30% prevalence).
- **Hearing:**
  - **Critical Period:** Birth to ~3–5 years; most sensitive in first 2 years.
  - **Auditory Cortex:** Located in the temporal lobe, processes auditory stimuli (e.g., phonemes) via input from the cochlea through the medial geniculate nucleus.
  - **Mechanism:** Auditory input refines neural pathways for sound discrimination; hearing loss (e.g., from otitis media) impairs speech development.
  - **Clinical Relevance:** Untreated hearing loss linked to language delays in ~20% of cases.
- **Language:**
  - **Critical Period:** Infancy to ~7 years; peaks in first 3 years.
  - **Mechanism:** Linguistic input activates the auditory cortex and language areas (e.g., Broca's, Wernicke's areas), facilitating phonetic learning and syntax acquisition.
  - **Clinical Relevance:** Limited language exposure (e.g., neglect) increases risk for language disorders (~15% prevalence in at-risk populations).

## 2. Neurodevelopmental Implications:

- **Disorders Linked to Sensory/Language Deficits:**
  - **Autism Spectrum Disorder (ASD):** Sensory processing issues (e.g., hyper/hypo-sensitivity to visual/auditory stimuli) in ~80%; language delays in ~50%.
  - **Specific Language Impairment (SLI):** Deficits in language acquisition without hearing loss; linked to auditory cortex dysfunction.

- **Developmental Delay:** Global delays in sensory/language skills due to environmental deprivation or genetic factors.
- **Psychiatric Overlap:** Sensory/language deficits contribute to behavioral issues (e.g., irritability, social withdrawal), mimicking anxiety or ADHD.

### 3. Assessment for PMHNPs:

- **Clinical Evaluation:**
  - **Vision:** Assess milestones (e.g., tracking objects by 3 months, recognizing faces by 6 months); screen for visual fixation or strabismus.
  - **Hearing:** Evaluate responses to sounds (e.g., startle reflex, babbling by 12 months); screen for hearing loss history (e.g., recurrent otitis media).
  - **Language:** Assess milestones (e.g., first words by 12–18 months, sentences by 3 years); use parental reports for delays.
- **Standardized Tools:**
  - **Ages and Stages Questionnaires (ASQ-3):** Screens developmental milestones (vision, hearing, language) in infants/children.
  - **M-CHAT-R:** Detects ASD-related sensory/language deficits.
  - **C-SSRS:** Assesses suicidality in older children with behavioral issues tied to sensory/language delays.
- **Neuroimaging/Tests:**
  - **MRI/fMRI:** Identifies cortical abnormalities (e.g., reduced auditory cortex volume in SLI).
  - **Hearing Tests:** Audiometry for suspected hearing loss.
- **PMHNP Relevance:** Early screening identifies delays, enabling timely referrals to speech-language pathologists or developmental specialists.

### 4. Management Strategies:

- **Interventions:**
  - **Vision:** Early correction of visual impairments (e.g., glasses for amblyopia) to support visual cortex development.
  - **Hearing:** Hearing aids or cochlear implants for hearing loss; auditory training to enhance cortical processing.
  - **Language:** Speech-language therapy (SLT) to improve phonetic and syntactic skills; parent-child interaction therapy to boost linguistic input.
  - **Behavioral:** Applied Behavior Analysis (ABA) for ASD; CBT for older children with anxiety from sensory/language issues.

- **Pharmacotherapy:**
    - Limited role in sensory/language delays; used for comorbidities (e.g., methylphenidate for ADHD, SSRIs for anxiety).
    - Example: Sertraline for anxiety in older children with language delays (~40% symptom reduction).
  - **Supportive Interventions:** Parental education, early intervention programs (e.g., Head Start), and school accommodations (e.g., IEPs).
- 

## Safety Issues

### 1. Behavioral Risks:

- Sensory/language delays increase irritability, aggression (~20% higher in ASD), risking injury or social isolation.
- **Mitigation:** Implement ABA, structured environments; monitor with ASQ-3.

### 2. Suicide Risk:

- Older children with sensory/language delays may develop anxiety/depression (~10% ideation risk).
- **Mitigation:** Screen with C-SSRS; provide family support.

### 3. Medication Safety:

- Stimulants/SSRIs risk agitation in children with sensory issues; FDA black box for suicidality in <25 years.
- **Mitigation:** Start low, monitor with GAD-7/PHQ-9.

### 4. Missed Diagnosis:

- Delays mistaken for behavioral issues (e.g., ADHD, oppositional defiant disorder) delay intervention.
  - **Mitigation:** Use ASQ-3, M-CHAT-R for early screening.
- 

## High-Yield Information

### • Key Features:

- Critical periods: Vision/hearing (birth–5 years), language (infancy–7 years).

- Auditory cortex: Processes phonetic input; visual cortex: Shapes visual processing.
  - **Applications:**
    - Diagnostic: Use ASQ-3, M-CHAT-R to screen for delays.
    - Therapeutic: SLT, ABA, early intervention programs.
    - Preventive: Early sensory/language input reduces neurodevelopmental risks (~50% lower delays).
  - **Exam Pearls:**
    - Sensory/language delays mimic psychiatric disorders; screen early.
    - Questions test developmental milestones, intervention timing.
    - Critical periods are time-sensitive for neural plasticity.
- 

### Role of the PMHNP

- **Assessment:** Screen with ASQ-3, M-CHAT-R; evaluate sensory/language milestones.
  - **Intervention:** Refer to SLT, developmental specialists; manage comorbidities with pharmacotherapy.
  - **Education:** Teach families about critical periods and intervention benefits.
  - **Advocacy:** Promote early screening and access to intervention programs.
- 



**Question:** A 4-year-old male presents with delayed speech (no sentences), poor response to sounds, and irritability. Parents report normal vision but limited social interaction. Physical exam is normal, and hearing tests are pending. According to DSM-5-TR and neurodevelopmental principles, which of the following is the most appropriate next step?

- A. Start methylphenidate 5 mg daily for suspected ADHD.
- B. Continue monitoring and reassess in 6 months.
- C. Prescribe sertraline 25 mg daily for irritability.
- D. Screen with M-CHAT-R and refer for speech-language therapy.

**Answer: D. Screen with M-CHAT-R and refer for speech-language therapy:** The child's delayed speech, poor auditory response, and irritability suggest a language delay, potentially linked to autism spectrum disorder (ASD) or specific language impairment, given the critical period for language development (infancy–7 years). Screening with M-CHAT-R assesses ASD

risk, and referral for speech-language therapy (SLT) targets language deficits, improving outcomes by ~50%. This aligns with PMHNP priorities for early intervention in neurodevelopmental disorders.

**Why It's High-Yield:** Tests screening and intervention for language delays, a core PMHNP exam skill.

- **A. Start methylphenidate 5 mg daily for suspected ADHD:** Methylphenidate treats ADHD but is premature without ruling out language delays or ASD, which mimic ADHD irritability. SLT and screening are priorities. **Exam Tip:** Screen for developmental delays before assuming ADHD.
- **B. Continue monitoring and reassess in 6 months:** Delaying intervention during the critical language period risks worsening outcomes. M-CHAT-R and SLT are needed now to address delays. **Exam Tip:** Early intervention is critical for language delays.
- **C. Prescribe sertraline 25 mg daily for irritability:** Sertraline is not indicated for irritability in a 4-year-old without confirmed mood disorder; developmental delays must be addressed first. **Exam Tip:** Avoid psychotropics in young children without clear diagnosis.

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## Cognition and Insight Assessment in Psychiatric Practice: A Review for PMHNPs Overview

Cognition and insight are critical components of psychiatric assessment, providing insights into a patient's mental health status and guiding diagnosis and treatment. **Cognition** encompasses consciousness, orientation, attention, memory, and executive functioning, assessed through standardized tasks to identify deficits suggestive of neurocognitive disorders (e.g., dementia) or mood disorders (e.g., major depressive disorder). **Insight** reflects a patient's self-awareness of their mental health condition, ranging from absent to full, influencing treatment adherence and prognosis. For PMHNPs, mastering these assessments is essential for differentiating primary neurocognitive disorders from psychiatric conditions, ensuring accurate diagnosis, and optimizing patient outcomes.

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### Major Points

1. **Cognition Assessment:**
  - **Components:**
    - **Level of Consciousness:** Evaluates alertness (e.g., alert, lethargic, comatose); impaired in delirium or severe dementia.
    - **Orientation:** Assesses awareness of person, place, time, situation (e.g., "What is today's date?"); impaired in dementia or acute confusion.

- **Attention Span:** Tests focus and concentration (e.g., serial 7s, digit span); deficits in ADHD, depression, or dementia.
  - **Memory Recall:** Evaluates short-term (e.g., recall three words after 5 minutes) and long-term memory; impaired in Alzheimer's, MDD (pseudodementia).
  - **Executive Functioning:** Assesses planning, problem-solving, abstract thinking (e.g., proverb interpretation, arithmetic); deficits in frontotemporal dementia, schizophrenia.
- **Assessment Tools:**
    - **Mini-Mental State Examination (MMSE):** Scores 0–30; <24 suggests cognitive impairment.
    - **Montreal Cognitive Assessment (MoCA):** More sensitive for mild deficits; <26 indicates impairment.
    - **Clock Drawing Test:** Assesses visuospatial and executive function; abnormal in dementia.
    - **Digit Span Test:** Measures attention and working memory.
  - **Tasks:**
    - Recall recent events (e.g., “What did you eat for breakfast?”).
    - Simple arithmetic (e.g., “Subtract 7 from 100 repeatedly”).
    - Proverb interpretation (e.g., “What does ‘a rolling stone gathers no moss’ mean?”).

## 2. Insight Assessment:

- **Definition:** The patient's awareness and understanding of their mental health condition, including its symptoms, impact, and need for treatment.
- **Levels of Insight (DSM-5-TR):**
  - **Absent:** No recognition of illness (e.g., delusions in schizophrenia).
  - **Poor:** Partial awareness but minimization (e.g., bipolar disorder in mania).
  - **Good/Fair:** Acknowledges illness and need for treatment (e.g., depression with insight).
- **Assessment Methods:**
  - **Clinical Interview:** Ask, “Do you think you have a mental health problem?” or “Why do you think you're here?”
  - **Scale for Assessment of Unawareness of Mental Disorder (SUMD):** Quantifies insight across awareness, attribution, and treatment need.

- **Clinical Relevance:**
  - Poor insight predicts poor adherence (~50% lower in schizophrenia).
  - Good insight improves prognosis in depression (~40% better outcomes with therapy adherence).

### 3. Clinical Implications:

- **Neurocognitive Disorders:**
  - **Dementia (e.g., Alzheimer's, Vascular):** Memory deficits (hippocampal), executive dysfunction (frontal); poor insight common in later stages.
  - **Mild Cognitive Impairment (MCI):** Subtle deficits detectable by MoCA; insight often preserved.
- **Mood Disorders:**
  - **Major Depressive Disorder (MDD):** Pseudodementia with reversible memory/attention deficits; insight typically preserved unless psychotic.
  - **Bipolar Disorder:** Poor insight during mania; cognitive deficits (e.g., attention) in euthymic phases.
- **Psychotic Disorders:**
  - **Schizophrenia:** Absent/poor insight; executive and memory deficits due to frontal/subcortical dysfunction.
- **PMHNP Relevance:** Differentiate cognitive deficits from psychiatric vs. neurocognitive causes; assess insight to tailor interventions and predict adherence.

### 4. Management Strategies:

- **Cognition:**
  - **Neurocognitive Disorders:** Cholinesterase inhibitors (e.g., donepezil for Alzheimer's); cognitive rehabilitation for MCI.
  - **Mood Disorders:** SSRIs (e.g., sertraline for MDD); CBT to improve attention/memory.
  - **Monitoring:** Repeat MoCA/MMSE to track cognitive changes; adjust treatment based on deficits.
- **Insight:**
  - **Poor Insight:** Psychoeducation, motivational interviewing (MI) to enhance treatment acceptance.

- **Good Insight:** Leverage for therapy adherence (e.g., CBT, EMDR); involve family for support.
  - **Supportive Interventions:** Caregiver education, environmental modifications (e.g., memory aids).
- 

## Safety Issues

### 1. Suicide Risk:

- Poor insight in MDD or schizophrenia increases suicidality (~15–20% ideation risk).
- **Mitigation:** Screen with C-SSRS; implement safety plans or hospitalization.

### 2. Treatment Non-Adherence:

- Absent/poor insight in psychotic disorders leads to medication refusal (~50% non-adherence in schizophrenia).
- **Mitigation:** Use MI, long-acting injectables (e.g., aripiprazole depot).

### 3. Cognitive Impairment Risks:

- Memory/attention deficits increase medication errors or falls in elderly (~20% fall risk in dementia).
- **Mitigation:** Simplify regimens, use memory aids, assess home safety.

### 4. Misdiagnosis:

- Cognitive deficits in MDD (pseudodementia) mistaken for dementia delay appropriate treatment.
  - **Mitigation:** Use MoCA, longitudinal history to differentiate.
- 

## High-Yield Information

### • Key Features:

- Cognition: Assess consciousness, orientation, attention, memory, executive function with MoCA/MMSE.
- Insight: Ranges from absent (psychosis) to good (depression); impacts adherence.

- **Applications:**

- Diagnostic: Use MoCA, SUMD to assess cognition/insight; differentiate neurocognitive vs. psychiatric causes.
- Therapeutic: CBT, SSRIs for mood-related deficits; psychoeducation for insight.
- Preventive: Early cognitive screening reduces diagnostic delays (~30% better outcomes).

- **Exam Pearls:**

- Questions test MoCA vs. MMSE sensitivity, insight's role in adherence.
- Pseudodementia in MDD is reversible; dementia is progressive.
- Poor insight is hallmark of psychosis.

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### Role of the PMHNP

- **Assessment:** Use MoCA, MMSE, SUMD to evaluate cognition and insight; screen for suicidality.
- **Intervention:** Prescribe SSRIs, refer for CBT; use MI for poor insight.
- **Education:** Teach patients/families about cognitive deficits and insight's impact on treatment.
- **Advocacy:** Promote access to cognitive testing and integrated care.



**Question:** A 65-year-old female presents with difficulty recalling recent events, poor concentration, and sadness for 3 months. She acknowledges her low mood but denies needing treatment. MoCA score is 22, and PHQ-9 is 18. According to DSM-5-TR and cognitive assessment principles, which of the following is the most appropriate next step?

- A. Start donepezil 5 mg daily for suspected Alzheimer's disease.
- B. Continue monitoring and reassess cognition in 6 months.
- C. Initiate sertraline 25 mg daily and refer for CBT to address pseudodementia.
- D. Order an MRI to confirm hippocampal atrophy.

**Answer: C. Initiate sertraline 25 mg daily and refer for CBT to address pseudodementia:** The patient's cognitive deficits (memory, concentration) and high PHQ-9 score (18) suggest major depressive disorder (MDD) with pseudodementia, a reversible cognitive impairment linked to depression. Sertraline (25 mg daily) and CBT address mood and cognitive symptoms

(~30% improvement), aligning with PMHNP priorities for treating MDD-related cognitive deficits before assuming dementia.

**Why It's High-Yield:** Tests differentiation of pseudodementia from dementia, a core PMHNP exam skill.

- **A. Start donepezil 5 mg daily for suspected Alzheimer's disease:** Donepezil is for Alzheimer's, but cognitive deficits with prominent depression and preserved insight suggest pseudodementia, not dementia. Sertraline/CBT is priority. **Exam Tip:** Pseudodementia is reversible; dementia is progressive.
- **B. Continue monitoring and reassess cognition in 6 months:** Delaying treatment for MDD risks worsening cognitive and mood symptoms. Immediate intervention with sertraline/CBT is needed. **Exam Tip:** Pseudodementia requires prompt psychiatric treatment.
- **D. Order an MRI to confirm hippocampal atrophy:** MRI is useful for dementia but premature here, as cognitive deficits align with MDD pseudodementia. Treat mood symptoms first to assess reversibility. **Exam Tip:** Treat reversible causes (e.g., depression) before neuroimaging.

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## CIWA-Ar and COWS in Managing Substance Withdrawal

The **Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar)** and **Clinical Opiate Withdrawal Scale (COWS)** are standardized tools used to quantify the severity of alcohol and opioid withdrawal symptoms, respectively, enabling PMHNPs to develop individualized treatment plans and minimize complications. These tools are critical in psychiatric settings for patients with substance use disorders (SUDs), ensuring precise assessment and safe management. Mastery of CIWA-Ar and COWS is essential for PMHNPs, particularly for certification exams, as they underpin effective intervention in withdrawal management.

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### Major Points

1. **Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar):**
  - **Purpose:** Quantifies alcohol withdrawal severity to guide treatment, particularly benzodiazepine administration, to prevent seizures and delirium tremens.
  - **Components:**
    - Assesses 10 symptoms (scored 0–7, except orientation 0–4): Nausea/vomiting, tremor, paroxysmal sweats, anxiety, agitation, tactile/auditory/visual disturbances, headache, orientation/clouding of sensorium.
    - Total score: 0–67; mild (<10), moderate (10–15), severe (>15).

- **Administration:**
  - Conducted every 1–4 hours during acute withdrawal (first 24–72 hours).
  - Guides benzodiazepine dosing (e.g., lorazepam 1–2 mg for scores  $\geq 10$ ).
- **Clinical Use:**
  - Mild: Supportive care, hydration.
  - Moderate: Benzodiazepines (e.g., lorazepam, diazepam) to manage symptoms.
  - Severe: Hospitalization, IV benzodiazepines to prevent seizures (~5% risk in severe withdrawal).
- **PMHNP Relevance:** Use CIWA-Ar to titrate treatment, monitor for complications (e.g., seizures, delirium), and coordinate inpatient care for severe cases.

## 2. Clinical Opiate Withdrawal Scale (COWS):

- **Purpose:** Quantifies opioid withdrawal severity to guide medication-assisted treatment (MAT) and supportive care.
- **Components:**
  - Assesses 11 symptoms (scored variably): Resting pulse rate, sweating, restlessness, pupil size, bone/joint aches, runny nose/tearing, GI upset, tremor, yawning, anxiety/irritability, gooseflesh skin.
  - Total score: 0–48; mild (5–12), moderate (13–24), severe ( $>24$ ).
- **Administration:**
  - Conducted every 4–6 hours during early withdrawal (first 12–48 hours).
  - Guides MAT initiation (e.g., buprenorphine for scores  $\geq 8$ ).
- **Clinical Use:**
  - Mild: Non-opioid symptom relief (e.g., clonidine for sweating).
  - Moderate: Start buprenorphine (4–8 mg initial dose); adjunctive medications (e.g., loperamide for GI symptoms).
  - Severe: Hospitalization, buprenorphine/naloxone, or methadone for stabilization.
- **PMHNP Relevance:** Use COWS to initiate MAT, monitor withdrawal progression, and prevent complications (e.g., dehydration, relapse).

## 3. Clinical Implications:

- **Alcohol Withdrawal:**
  - Risks: Seizures, delirium tremens (~5% mortality if untreated), cardiovascular instability.
  - Management: Benzodiazepines (first-line), thiamine to prevent Wernicke's encephalopathy, hydration.
- **Opioid Withdrawal:**
  - Risks: Severe discomfort, relapse (~40% risk without MAT), dehydration.
  - Management: MAT (buprenorphine, methadone), supportive care (e.g., NSAIDs for aches).
- **DSM-5-TR Context:**
  - Alcohol withdrawal: DSM-5-TR criteria include autonomic hyperactivity, tremors, anxiety.
  - Opioid withdrawal: Criteria include dysphoria, nausea, muscle aches, yawning.
- **PMHNP Relevance:** Accurate CIWA-Ar/COWS scoring ensures tailored treatment, reducing complications and relapse risk.

#### 4. Management Strategies:

- **Pharmacotherapy:**
  - **Alcohol Withdrawal:** Lorazepam (1–4 mg every 4 hours for CIWA-Ar  $\geq 10$ ); diazepam for rapid onset in severe cases.
  - **Opioid Withdrawal:** Buprenorphine (4–16 mg/day for COWS  $\geq 8$ ); methadone (20–40 mg/day in supervised settings).
- **Psychotherapy/Supportive Interventions:**
  - Motivational interviewing (MI) to enhance treatment engagement.
  - Cognitive-behavioral therapy (CBT) to address relapse triggers (~30% reduced relapse).
  - Peer support (e.g., AA/NA) and case management for social determinants of health.
- **Monitoring:** Repeat CIWA-Ar/COWS to adjust treatment; use C-SSRS for suicidality (~15% ideation risk in withdrawal).

## Safety Issues

### 1. Withdrawal Complications:

- Alcohol: Seizures, delirium tremens (~5% risk in severe cases).
- Opioid: Relapse, overdose (~20% risk without MAT).
- **Mitigation:** Use CIWA-Ar/COWS to guide timely pharmacotherapy; hospitalize severe cases.

### 2. Suicide Risk:

- Withdrawal exacerbates depression/anxiety, increasing suicidality (~15% ideation in SUD).
- **Mitigation:** Screen with C-SSRS; provide safety plans or hospitalization.

### 3. Medication Risks:

- Benzodiazepines risk oversedation; buprenorphine/methadone risk respiratory depression.
- **Mitigation:** Monitor vital signs, titrate doses based on CIWA-Ar/COWS scores.

### 4. Misdiagnosis:

- Withdrawal symptoms (e.g., anxiety, irritability) mimic primary psychiatric disorders, delaying treatment.
- **Mitigation:** Use CIWA-Ar/COWS, toxicology to confirm withdrawal.

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## High-Yield Information

### • Key Features:

- CIWA-Ar: Scores alcohol withdrawal (0–67); guides benzodiazepine use.
- COWS: Scores opioid withdrawal (0–48); guides MAT.

### • Applications:

- Diagnostic: Use CIWA-Ar/COWS for precise withdrawal assessment.
- Therapeutic: Benzodiazepines for alcohol; buprenorphine for opioids; MI/CBT for relapse prevention.
- Preventive: Early intervention reduces complications (~50% lower seizure risk with CIWA-Ar-guided treatment).

### • Exam Pearls:

- CIWA-Ar >15 indicates severe alcohol withdrawal; COWS  $\geq$ 8 prompts MAT.
- Questions test scoring, treatment titration, and safety monitoring.
- Avoid benzodiazepines in opioid withdrawal.

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### Role of the PMHNP

- **Assessment:** Use CIWA-Ar/COWS to quantify withdrawal severity; screen for suicidality with C-SSRS.
- **Intervention:** Administer benzodiazepines/MAT based on scores; refer for CBT/MI.
- **Education:** Teach patients/families about withdrawal risks and treatment benefits.
- **Advocacy:** Promote access to MAT and inpatient detox programs.



**Question:** A 45-year-old male presents 24 hours after last alcohol use with tremors, anxiety, and nausea (CIWA-Ar score 12). He denies suicidality. Physical exam shows tachycardia. According to DSM-5-TR and clinical guidelines, which of the following is the most appropriate initial management?

- A. Start naltrexone 50 mg daily to prevent relapse.
- B. Initiate lorazepam 1 mg every 4 hours and administer thiamine 100 mg daily.
- C. Monitor symptoms and reassess in 24 hours.
- D. Refer for outpatient CBT to address anxiety.

**Answer:** B. Initiate lorazepam 1 mg every 4 hours and administer thiamine 100 mg daily.

**Rationales:** The patient's CIWA-Ar score of 12 indicates moderate alcohol withdrawal, requiring benzodiazepines (e.g., lorazepam 1 mg every 4 hours) to manage symptoms and prevent seizures (~50% risk reduction). Thiamine 100 mg daily prevents Wernicke's encephalopathy, a standard in alcohol withdrawal, aligning with PMHNP priorities for safe management.

**Why It's High-Yield:** Tests CIWA-Ar-guided treatment and safety, core PMHNP exam skills.

- **A. Start naltrexone 50 mg daily to prevent relapse:** Naltrexone is for relapse prevention post-detox, not acute withdrawal. Lorazepam and thiamine address immediate risks. **Exam Tip:** Naltrexone is inappropriate during active withdrawal.
- **C. Monitor symptoms and reassess in 24 hours:** A CIWA-Ar score of 12 requires immediate treatment to prevent seizures or delirium tremens, not delayed monitoring. **Exam Tip:** Moderate/severe CIWA-Ar scores prompt urgent intervention.
- **D. Refer for outpatient CBT to address anxiety:** CBT is effective for relapse prevention but not acute withdrawal. Lorazepam/thiamine are needed for immediate

symptom control. **Exam Tip:** Acute withdrawal prioritizes pharmacotherapy over psychotherapy.

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## Differentiating Between Dementia and Depression in the Elderly Population

Dementia and depression in elderly patients often present with overlapping symptoms, such as memory impairment, apathy, social withdrawal, and reduced daily functioning, complicating diagnosis. However, their distinct pathophysiological origins—neurodegenerative processes in dementia versus neurochemical and psychosocial factors in depression—require tailored assessment and management strategies. For PMHNPs, mastering differentiation is critical for accurate diagnosis, effective treatment, and improved patient outcomes, particularly in geriatric psychiatry where misdiagnosis can delay care and worsen prognosis.

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### Major Points

#### 1. Clinical Presentations and Overlap:

- **Dementia:**
  - **Types:** Alzheimer’s disease (AD), vascular dementia, frontotemporal dementia (FTD), Lewy body dementia (LBD).
  - **Symptoms:** Progressive cognitive decline (memory, executive function), apathy, social withdrawal, impaired activities of daily living (ADLs), disorientation, poor insight.
  - **Key Features:** Gradual (AD) or stepwise (vascular) onset; irreversible; neurological signs (e.g., gait issues in vascular dementia).
  - **Prevalence:** ~10–15% of adults  $\geq 65$ .
- **Depression (Major Depressive Disorder, MDD):**
  - **Symptoms:** Persistent sadness, anhedonia, apathy, memory complaints (pseudodementia), social withdrawal, reduced ADLs, preserved or partial insight.
  - **Key Features:** Acute or subacute onset; reversible with treatment; prominent mood symptoms (e.g., guilt, hopelessness).
  - **Prevalence:** ~10–20% of elderly, higher in medical settings.
- **Overlap:**
  - Both show memory impairment, apathy, social isolation, and ADL decline.
  - Pseudodementia in depression mimics dementia but improves with mood treatment.

- Depression is common in early dementia (~30–50%), complicating diagnosis.

## 2. Pathophysiological Origins:

### ○ Dementia:

- **Alzheimer's:** Amyloid plaques, tau tangles cause hippocampal/cortical atrophy; memory loss predominates.
- **Vascular:** Cerebrovascular damage (e.g., white matter hyperintensities, infarcts) impairs cognition.
- **FTD:** Frontal/temporal degeneration causes behavioral/executive deficits.
- **LBD:** Lewy bodies disrupt dopamine/acetylcholine, causing hallucinations, fluctuating cognition.
- **Mechanism:** Neurodegenerative, reducing neural connectivity; irreversible.

### ○ Depression:

- **Mechanism:** Dysregulation of serotonin, norepinephrine, dopamine; HPA axis hyperactivity (elevated cortisol); psychosocial stressors (e.g., loss, isolation).
- **Brain Changes:** Reversible hippocampal volume reduction (~10% in chronic MDD); no progressive neurodegeneration.

## 3. Assessment Strategies:

### ○ Clinical Interview:

- **Dementia:** Focus on cognitive history (e.g., gradual memory loss, disorientation); assess neurological signs (e.g., gait, tremors).
- **Depression:** Explore mood symptoms (e.g., sadness, guilt); assess onset, stressors, and insight.

### ○ Standardized Tools:

- **Montreal Cognitive Assessment (MoCA):** Sensitive for dementia (score <26); less impaired in depression.
- **Mini-Mental State Examination (MMSE):** Scores <24 suggest dementia; depression may show mild deficits.
- **Geriatric Depression Scale (GDS-15):** Screens for depression; scores  $\geq 5$  indicate MDD.
- **Columbia-Suicide Severity Rating Scale (C-SSRS):** Assesses suicidality (~15–20% ideation in elderly depression).

- **Neuroimaging:**
  - **Dementia:** MRI shows hippocampal atrophy (AD), white matter hyperintensities (vascular), or frontal atrophy (FTD).
  - **Depression:** Normal or mild hippocampal reduction; no progressive atrophy.
- **Other Tests:**
  - **Labs:** Rule out reversible causes (e.g., B12 deficiency, hypothyroidism).
  - **Neuropsych Testing:** Detailed cognitive profiling to differentiate dementia domains from depression's pseudodementia.
- **Key Differentiators:**
  - **Onset:** Gradual (dementia) vs. acute/subacute (depression).
  - **Insight:** Poor/absent in dementia; preserved/partial in depression.
  - **Mood:** Prominent sadness/guilt in depression; apathy more common in dementia.
  - **Reversibility:** Depression's cognitive deficits improve with treatment; dementia's do not.

#### 4. Management Strategies:

- **Dementia:**
  - **Pharmacotherapy:** Cholinesterase inhibitors (e.g., donepezil 5–10 mg/day for AD); memantine for moderate-severe AD; low-dose antipsychotics (e.g., quetiapine) for agitation (FDA black box for mortality risk).
  - **Non-Pharmacologic:** Cognitive rehabilitation, caregiver support, environmental modifications (e.g., reduce fall risks).
  - **Prognosis:** Progressive; focus on slowing decline, improving quality of life.
- **Depression:**
  - **Pharmacotherapy:** SSRIs (e.g., sertraline 25–150 mg/day, FDA-approved for MDD); avoid tricyclics in elderly due to anticholinergic effects.
  - **Psychotherapy:** Cognitive-behavioral therapy (CBT), interpersonal therapy (IPT) improve mood and cognition (~40% remission).
  - **Prognosis:** Reversible with treatment; ~60% remission with combined SSRI/CBT.
- **PMHNP Relevance:** Differentiate via MoCA/GDS-15; treat depression first to rule out pseudodementia; coordinate with neurology for dementia care.

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## Safety Issues

### 1. Suicide Risk:

- Depression in elderly increases suicidality (~20% ideation, higher in males).
- **Mitigation:** Screen with C-SSRS; hospitalize for high risk; implement safety plans.

### 2. Medication Risks:

- Antipsychotics in dementia carry FDA black box warning for mortality (~1–2% increased risk); SSRIs risk hyponatremia or falls in elderly.
- **Mitigation:** Use low-dose atypicals; monitor sodium, ECG; start SSRIs at low doses (e.g., sertraline 25 mg).

### 3. Falls and Functional Decline:

- Dementia increases fall risk (~30% annually); depression's apathy reduces ADLs, increasing frailty.
- **Mitigation:** Physical therapy, home safety assessments.

### 4. Misdiagnosis:

- Mistaking depression's pseudodementia for dementia delays effective treatment; mistaking dementia for depression risks inappropriate pharmacotherapy.
- **Mitigation:** Use MoCA, GDS-15, and longitudinal history; trial SSRI for suspected depression.

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## High-Yield Information

### • Key Features:

- **Dementia:** Progressive cognitive decline, poor insight, neurological signs (e.g., atrophy on MRI).
- **Depression:** Reversible pseudodementia, preserved insight, prominent mood symptoms.

### • Applications:

- Diagnostic: Use MoCA (<26 for dementia), GDS-15 ( $\geq 5$  for depression); MRI for structural changes.
- Therapeutic: SSRIs/CBT for depression; cholinesterase inhibitors for dementia.

- Preventive: Early diagnosis reduces complications (~30% better outcomes with correct identification).

- **Exam Pearls:**

- Questions test differentiation via onset, insight, and reversibility.
  - Pseudodementia improves with SSRI/CBT; dementia does not.
  - MRI distinguishes dementia (atrophy) from depression (normal/mild changes).
- 

## Role of the PMHNP

- **Assessment:** Use MoCA, GDS-15, and clinical interview to differentiate dementia from depression; screen for suicidality with C-SSRS.
  - **Intervention:** Initiate SSRIs/CBT for depression; refer to neurology for dementia; coordinate multidisciplinary care.
  - **Education:** Explain differences between dementia and depression to patients/families; highlight treatment reversibility for depression.
  - **Advocacy:** Promote access to geriatric mental health services and cognitive screening.
- 



**Question:** A 72-year-old female presents with memory complaints, apathy, and social withdrawal for 4 months. She reports sadness and guilt, with preserved insight. MoCA score is 23, and GDS-15 is 10. MRI is normal. According to DSM-5-TR and clinical guidelines, which of the following is the most appropriate initial management?

- A. Start donepezil 5 mg daily for suspected Alzheimer's disease.
- B. Monitor symptoms and reassess cognition in 6 months.
- C. Order PET scan to confirm dementia diagnosis.
- D. Initiate sertraline 25 mg daily and refer for CBT to address depression.

**Answer: D. Initiate sertraline 25 mg daily and refer for CBT to address depression.**

**Rationales:** The patient's memory complaints, apathy, social withdrawal, preserved insight, high GDS-15 score (10), and normal MRI suggest **major depressive disorder (MDD)** with pseudodementia. Sertraline (25 mg daily) and CBT are first-line treatments, achieving ~60% remission in elderly depression, aligning with PMHNP priorities for reversible cognitive deficits.

**Why It's High-Yield:** Tests differentiation of pseudodementia from dementia, a core PMHNP exam skill.

- **A. Start donepezil 5 mg daily for suspected Alzheimer’s disease:** Donepezil is for Alzheimer’s, but normal MRI, preserved insight, and prominent mood symptoms point to depression, not dementia. Sertraline/CBT is appropriate. **Exam Tip:** Normal MRI and insight rule out dementia.
  - **B. Monitor symptoms and reassess cognition in 6 months:** Delaying treatment for depression risks worsening mood and cognitive symptoms. Immediate intervention with sertraline/CBT is needed. **Exam Tip:** Depression’s pseudodementia requires prompt treatment.
  - **C. Order PET scan to confirm dementia diagnosis:** PET scans are used for dementia when MRI is inconclusive, but normal MRI and depression symptoms make PET unnecessary. Treat depression first. **Exam Tip:** Treat reversible causes before advanced imaging.
- 

### **The Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS) in Assessing Sleep Disorders**

The **Pittsburgh Sleep Quality Index (PSQI)** and **Epworth Sleepiness Scale (ESS)** are standardized, self-administered tools critical for assessing sleep disorders in psychiatric practice. The PSQI evaluates sleep quality across multiple dimensions over a one-month period, while the ESS quantifies daytime sleepiness, aiding in the diagnosis of conditions like insomnia, obstructive sleep apnea (OSA), and narcolepsy. For PMHNPs, these tools are essential for identifying sleep disturbances, informing diagnostic processes, tailoring interventions, and monitoring treatment outcomes, particularly given the high prevalence of sleep issues in psychiatric disorders (e.g., depression, anxiety).

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#### **Major Points**

##### **1. Pittsburgh Sleep Quality Index (PSQI):**

- **Purpose:** Assesses sleep quality over a one-month period to identify disturbances warranting intervention.
- **Components:**
  - Seven domains (scored 0–3 each): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, daytime dysfunction.
  - Total score: 0–21; >5 indicates significant sleep disturbance.
- **Administration:**
  - Self-administered questionnaire; takes ~5–10 minutes.
  - Used in outpatient/inpatient settings to screen for insomnia, OSA, or psychiatric-related sleep issues.

- **Clinical Use:**
  - **Score Interpretation:**
    - $\leq 5$ : Good sleep quality.
    - 5: Poor sleep quality;  $>10$  suggests severe disturbance requiring intervention.
  - Guides treatment (e.g., CBT for insomnia, pharmacotherapy like trazodone).
  - Monitors response to interventions (e.g., improved scores post-treatment).
- **PMHNP Relevance:** Use PSQI to identify sleep disturbances in depression (~70% prevalence), anxiety, or PTSD; tailor interventions based on specific domains (e.g., sleep latency).

## 2. Epworth Sleepiness Scale (ESS):

- **Purpose:** Quantifies daytime sleepiness to screen for disorders like OSA, narcolepsy, or hypersomnia.
- **Components:**
  - Eight everyday situations (e.g., reading, driving); scored 0–3 (0 = no chance of dozing, 3 = high chance).
  - Total score: 0–24;  $\geq 10$  indicates excessive daytime sleepiness.
- **Administration:**
  - Self-administered; takes ~3–5 minutes.
  - Used to assess functional impairment from sleepiness.
- **Clinical Use:**
  - **Score Interpretation:**
    - $<10$ : Normal.
    - 10–15: Mild to moderate sleepiness (e.g., insomnia, depression).
    - $\geq 16$ : Severe sleepiness (e.g., OSA, narcolepsy).
  - Prompts referral for polysomnography in suspected OSA/narcolepsy.
- **PMHNP Relevance:** Screen for daytime sleepiness in patients with fatigue (e.g., depression, SUD); coordinate with sleep specialists for high scores.

## 3. Clinical Implications:

- **Sleep Disorders in Psychiatry:**
  - **Insomnia:** Common in depression (~70%), anxiety (~50%), PTSD; assessed by PSQI.
  - **OSA:** Linked to depression, cognitive impairment; detected by ESS.
  - **Narcolepsy:** Excessive sleepiness, cataplexy; ESS prompts further evaluation.
- **DSM-5-TR Context:**
  - **Insomnia disorder:** Difficulty initiating/maintaining sleep, causing distress.
  - **Hypersomnolence disorder:** Excessive daytime sleepiness despite adequate sleep.
- **Comorbidities:** Sleep disturbances exacerbate psychiatric symptoms (e.g., ~30% increased depression severity).
- **PMHNP Relevance:** Use PSQI/ESS to differentiate primary sleep disorders from psychiatric-related sleep issues; guide treatment and referrals.

#### 4. Management Strategies:

- **Pharmacotherapy:**
  - **Insomnia:** Off-label trazodone (25–100 mg) or low-dose quetiapine (25–50 mg) for sleep; avoid benzodiazepines due to dependency RISK.
  - **OSA/Narcolepsy:** Refer to sleep specialists for CPAP (OSA) or stimulants (e.g., modafinil for narcolepsy).
- **Psychotherapy:**
  - **CBT for Insomnia (CBT-I):** First-line for insomnia; ~60% remission.
  - **Mindfulness-Based Therapy:** Reduces sleep latency in anxiety-related insomnia.
- **Supportive Interventions:** Sleep hygiene education (e.g., consistent bedtime), address social determinants (e.g., safe sleep environment).
- **Monitoring:** Repeat PSQI/ESS to track treatment response; use C-SSRS for suicidality (~15% ideation risk in sleep-disturbed depression).
- **Evidence:** 2025 APA guidelines endorse CBT-I as first-line for insomnia; PSQI/ESS for monitoring.

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## Safety Issues

### 1. Suicide Risk:

- Sleep disturbances increase suicidality in depression/PTSD (~15–20% ideation risk).
- **Mitigation:** Screen with C-SSRS; address sleep issues promptly.

## 2. Medication Risks:

- Trazodone risks orthostatic hypotension; quetiapine risks weight gain; benzodiazepines risk dependence.
- **Mitigation:** Start low doses; monitor BP, weight; avoid benzodiazepines.

## 3. Functional Impairment:

- Daytime sleepiness (ESS  $\geq 10$ ) increases accident risk (e.g., driving, ~20% higher in OSA).
- **Mitigation:** Advise against driving; refer for polysomnography.

## 4. Misdiagnosis:

- Sleep issues mistaken for primary depression/anxiety delay targeted treatment.
- **Mitigation:** Use PSQI/ESS to confirm sleep disorder; differentiate with PHQ-9/GAD-7.

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## High-Yield Information

### • Key Features:

- PSQI: Assesses 7 sleep quality domains (0–21);  $>5$  indicates disturbance.
- ESS: Measures daytime sleepiness (0–24);  $\geq 10$  suggests pathology.

### • Applications:

- Diagnostic: Use PSQI for insomnia; ESS for OSA/narcolepsy.
- Therapeutic: CBT-I for insomnia; CPAP/stimulants for OSA/narcolepsy.
- Preventive: Early sleep intervention reduces psychiatric exacerbation (~30% lower depression severity).

### • Exam Pearls:

- PSQI  $>5$  prompts intervention; ESS  $\geq 10$  warrants sleep study referral.
  - Questions test sleep assessment, treatment prioritization.
  - Avoid benzodiazepines for insomnia in psychiatric patients.
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## Role of the PMHNP

- **Assessment:** Use PSQI/ESS to screen for sleep disorders; integrate with PHQ-9, GAD-7 for psychiatric comorbidities.
  - **Intervention:** Prescribe trazodone/CBT-I for insomnia; refer for sleep studies in OSA/narcolepsy.
  - **Education:** Teach patients/families about sleep hygiene and disorder impacts.
  - **Advocacy:** Promote access to sleep clinics and CBT-I programs.
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**Question:** A 50-year-old female with major depressive disorder reports difficulty falling asleep and daytime fatigue. PSQI score is 12, and ESS is 14. She denies suicidality. According to DSM-5-TR and clinical guidelines, which of the following is the most appropriate initial management?

- A. Prescribe zolpidem 5 mg at bedtime for insomnia.
- B. Initiate trazodone 25 mg at bedtime and refer for CBT-I.
- C. Monitor symptoms and reassess in 4 weeks.
- D. Start modafinil 100 mg daily for daytime sleepiness.

**Answer: B. Initiate trazodone 25 mg at bedtime and refer for CBT-I:** The patient's PSQI score (12) indicates significant insomnia, and ESS (14) suggests moderate daytime sleepiness, likely related to depression and poor sleep quality. Trazodone (25 mg) is effective off-label for insomnia, and CBT-I is first-line, achieving ~60% remission. This approach addresses sleep and psychiatric symptoms, aligning with PMHNP priorities for safe, evidence-based care.

**Why It's High-Yield:** Tests PSQI/ESS-guided insomnia treatment, a core PMHNP exam skill.

- **A. Prescribe zolpidem 5 mg at bedtime for insomnia:** Zolpidem risks dependence and falls in older adults; trazodone/CBT-I are safer and preferred for depression-related insomnia. **Exam Tip:** Avoid Z-drugs in psychiatric patients with comorbidities.
- **C. Monitor symptoms and reassess in 4 weeks:** PSQI >5 and ESS ≥10 require immediate intervention to prevent worsening depression and functional impairment. Trazodone/CBT-I are proactive steps. **Exam Tip:** Significant sleep scores prompt urgent treatment.
- **D. Start modafinil 100 mg daily for daytime sleepiness:** Modafinil treats narcolepsy-related sleepiness, but ESS 14 and PSQI 12 suggest insomnia secondary to depression, not narcolepsy. Address insomnia first. **Exam Tip:** Confirm OSA/narcolepsy with sleep study before stimulants.

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