PSYCHOSOMATIC MEDICINE/
CONSULTATION-LIAISON PSYCHIATRY

PROGRAM AGENDA

THURSDAY, OCTOBER 22, 2020

4:00 – 4:15 PM  Welcome and Overview  Theodore A. Stern, MD


4:35 – 4:55 PM  Catatonia, NMS, and Serotonin Syndrome  Christopher M. Celano, MD

4:55 – 5:15 PM  Neurocognitive Assessment at the Bedside  Judith Restrepo, MD

5:15 – 6:10 PM  Panel Discussion  Moderator: Theodore A. Stern, MD  Panelists: Christopher M. Celano, MD, Judith Restrepo, MD

5:45 PM  Break

6:10 – 6:30 PM  The Risk of QTc Interval Prolongation with Psychotropics  Christopher M. Celano, MD

6:30 – 6:50 PM  Seizure Disorders and Non-Epileptic Seizures  Franklin King, MD

6:50 – 7:10 PM  Factitious Illness and Malingering  Theodore A. Stern, MD

7:10 – 8:00 PM  Panel Discussion  Moderator: Theodore A. Stern, MD  Panelists: Christopher M. Celano, MD, Franklin King, MD

8:00 PM  Adjourn
FACULTY

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  Associate Director, Cardiac Psychiatry Research Program,
  Massachusetts General Hospital Psychiatrist, Massachusetts General Hospital
  Assistant Professor, Harvard Medical School

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Theodore A. Stern, MD
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  Director, Thomas P. Hackett Center for Scholarship in Psychosomatic Medicine
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  Ned H. Cassem Professor of Psychiatry in the field of Psychosomatic Medicine/Consultation
  Harvard Medical School
  Editor-in-Chief, Psychosomatics
WELCOME AND INTRODUCTION

Theodore A. Stern, MD
DELIRIUM: DIFFERENTIAL DIAGNOSIS, EVALUATION, TREATMENT, AND PREVENTION

Theodore A. Stern, MD
The Diagnosis, Treatment, and Prevention of Delirium

Theodore A. Stern, MD
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Director, Thomas P. Hackett Center for Scholarship in Psychosomatic Medicine,
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Introduction: Agitation and Delirium

- Medical and surgical inpatient floors, as well as intensive care units (ICUs), are filled with agitated and confused patients
  - While such units provide the forum for dramatic, life-saving interventions...
  - They are uniquely stressful
    - high tension
    - danger
    - high technology
    - death

General Principles

- Don’t assume psychiatric symptoms are due to a long-standing psychiatric disorder
- Don’t assume that psychiatric symptoms are a reaction to being in a critical care environment
  - Initiate a search for the underlying cause of the symptoms
  - Identify the symptoms that require treatment
  - Treat symptoms as specifically as possible
Assessment of Mental Status

- Evaluate the ABCs
  - affect
  - behavior
  - cognition

The Mental Status Examination

- Appearance & behavior
  - hypervigilant, frightened, poor eye contact, agitated, psychomotorically retarded
- Speech
  - rambling, rapid, incoherent, fluent
- Mood
  - depressed, fearful, tearful, irritable, anxious, angry, apathetic

The Mental Status Examination

- Affect
  - despondent, anxious, perplexed, blunted
- Thought
  - paranoid, loose associations, hallucinating
- Cognition
  - disoriented, decreased concentration, confused, impaired memory
Screening Tests and Tools for Assessment of Cognition

- Mini-Mental State Examination
  - MMSE
- Montreal Cognitive Assessment
  - MoCA
- Confusion Assessment Method for the ICU
  - CAM-ICU

Agitation

- Excessive, usually non-purposeful motor activity associated with internal tension
- Varies from mild restlessness to combativeness
- Can signify clinical deterioration
- “ICU psychosis” is a misleading term
  - Implies cause & effect between being in the ICU and becoming psychotic
  - Agitation, delirium, and psychosis are not the same

Delirium: Definition

- An organic brain syndrome with a clouded state of consciousness, distractibility, decreased attention, sensory misperceptions, and a fluctuating course
  - “Acute brain failure”
Delirium: Signs & Symptoms

- Clouded consciousness
- Perceptual disturbances
- Incoherent speech
- Disturbed sleep-wake cycle
- Increased or decreased activity
- Disorientation and memory impairment
  - A fluctuating course
  - Related to an organic factor

Delirium: Associated Features

- Anxiety
- Fear
- Irritability
- Depression
- Euphoria
- Apathy
  - These features may steer clinicians to make another diagnosis

Treatment...

- Since treatment is predicated on the diagnosis
  - Identify the etiology as specifically as possible
  - Be sure to rule-out life-threatening causes
Delirium: Life-Threatening Causes

- Wernicke’s encephalopathy; Withdrawal reactions
- Hypoxia; Hypoperfusion of the CNS
- Hypoglycemia
- Hypertensive encephalopathy
- Intracerebral hemorrhage; Infection
- Meningitis/encephalitis; Metabolic
- Poisoning
- Seizures

Delirium: Differential Diagnosis

- Central nervous system
  - Vascular
    - hypertensive encephalopathy, intracranial hemorrhage, vasculitis, stroke
  - Neoplastic
    - space-occupying lesions, paraneoplastic syndrome
  - Seizure
    - post-ictal state, complex partial seizures

Delirium: Differential Diagnosis

- Cardiopulmonary
  - Cardiac arrest
  - Congestive heart failure
  - Respiratory failure
  - Shock
- Infection
  - Meningitis/encephalitis
  - Sepsis
  - Sub-acute bacterial endocarditis
**Delirium: Differential Diagnosis**

- Endocrine/metabolic
  - Acid-base disturbance
  - Fluid/electrolyte imbalance
  - Diabetic ketoacidosis
  - Hypoglycemia
  - Hepatic failure
  - Renal failure
  - Thyroid dysfunction

- Intoxication/withdrawal
  - Alcohol
  - Anesthetics
  - Anticholinergics
  - Hallucinogens
  - Psychostimulants
  - Narcotics
  - Sedative-hypnotics

- Nutritional deficiency
  - Folic acid
  - Niacin (pellagra)
  - Thiamine (Wernicke’s, Korsakoff’s)
  - Vitamin B_{12} (pernicious anemia)

- Poisons
  - Carbon monoxide
  - Heavy metals (lead, mercury)
  - Toxins
Common Delirium-Inducing Drugs

- Antiarrhythmics
  - Lidocaine, mexiletine, procainamide, quinidine
- Antibiotics
  - Penicillin, rifampin
- Anticholinergics
  - Atropine

Common Delirium-Inducing Drugs

- Antihistamines
  - Non-selective: diphenhydramine, promethazine
  - H₂ blockers: cimetidine, ranitidine
- Beta-blockers
  - Propranolol
- Narcotics
  - Meperidine, pentazocine

Treatment of Agitation

- Correct metabolic and systemic abnormalities
- Eliminate drug toxicity
- Remove the offending agent(s)
- Administer appropriate antidote(s)
  - e.g., Physostigmine, naloxone, flumazenil
Treat Drug Withdrawal

- Obstacles to prompt treatment
  - Emergent admissions may result in sudden discontinuation of abused drugs
  - History of use may be difficult to establish in intubated or unconscious patients
  - Physical signs of withdrawal are non-specific
  - No laboratory tests can confirm the diagnosis

Alcohol & Sedative-Hypnotics

- Alcohol withdrawal
  - Benzodiazepines, phenobarbital, neuroleptics
- Sedative-hypnotic withdrawal
  - Symptom-onset a function of half-life; the longer the half-life the longer the latency
  - Symptom frequency and intensity greatest with half-life of 10-20 hours
  - Treatment best with a longer half-life agent

Narcotic Withdrawal

- Syndrome generally mild
  - Discomfort; delirium uncommon
  - Treatment involves replacement with a longer half-life agent of the same class
  - Clonidine is effective in reducing symptoms
Haloperidol

- A high-potency agent
- Trivial effects on heart rate, blood pressure, respiratory drive
- Often used IV despite lack of FDA approval for IV use
- Used IV it precipitates with phenytoin and heparin;
  - Flush the IV line first
- Dose used depends on symptom severity

Haloperidol

- Onset of action: 10-30 minutes
- Hypotension, if it occurs, is associated with hypovolemia
- High-dose use associated with QTc prolongation and Torsades de Pointes
- Extrapyramidal side effects are rare with IV use

Haloperidol

- Titrate the dose to the symptoms
  - If mild, use 0.5-2 mg
  - If moderate, use 5-10 mg
  - If severe, use 10 mg or more
- Repeat doses when necessary, every 15-30 minutes
- Adjust dose to clinical course
Other Neuroleptics

- Droperidol
  - More sedating than haloperidol
  - Lowers blood pressure more than haloperidol
- Chlorpromazine
  - More anticholinergic, more apt to induce hypotension, and more likely to induce arrhythmias than haloperidol

Atypical Antipsychotics

- Olanzapine
- Quetiapine
- Risperidone
- Clozapine
- Ziprasidone

Alternative Agents for Agitation...

- Dexmedetomidine
  - Highly selective alpha-2 adrenoreceptor agonist with sedative and analgesic properties
- Valproate
  - Especially when irritability or impulsivity present
- Propofol
Alternative Agents for Agitation

- Narcotics
  - Morphine typically used
- Paralytics
  - If used, sedation still required
- Benzodiazepines
  - Lorazepam
    - used PO, SL, IV; has no active metabolites
  - Midazolam
    - rapidly-acting; causes amnesia and respiratory depression

Benzodiazepines...

- Midazolam
  - half-life, 1-12 hrs; 2 mg; fast
- Oxazepam
  - half-life, 5-15 hrs; 15 mg; slow
- Lorazepam
  - half-life, 10-20 hrs; 1 mg; intermediate
- Alprazolam
  - half-life, 12-15 hrs; 0.5 mg; intermediate-fast

Benzodiazepines...

- Chlordiazepoxide
  - half-life, 5-30 hrs; 10 mg; intermediate
- Clonazepam
  - half-life, 15-50 hrs; 0.25 mg; intermediate
- Diazepam
  - half-life, 20-100 hrs; 5 mg; fast
- Flurazepam
  - half-life, 40 hrs; 5 mg; fast
- Clorazepate
  - half-life, 30-200 hrs; 7.5 mg; fast
Benzodiazepines

- Diazepam
  - IV: onset, 2-5 min; starting dose, 2-5 mg
  - PO: onset, 10-60 min; starting dose, 2-5 mg
- Lorazepam
  - IV/IM: onset, 2-20 min; starting dose, 1-2 mg
  - SL: onset, 2-20 min; starting dose, 0.5-1 mg
  - PO: onset, 2-60 min; starting dose 0.5-1 mg

Non-Pharmacological Treatment

- Re-orientation
- Adjustment of physical environment
- Reassurance
  - Determine why are the patient is anxious to guide interventions
  - Clarify misconceptions
  - Remain calm

Prevention of Delirium

- Minimize risk factors for delirium
- Monitor lab values and vital signs
  - e.g., Oxygenation, hematocrit, blood pressure, drug levels
- Administer antipsychotics prophylactically
  - Administration of olanzapine reduced incidence of post-operative delirium from 41% to 15% in elderly joint replacement patients
Conclusion

- Medically-oriented psychiatric consultants can help evaluate and manage critically ill patients as well as prevent psychiatric and neuropsychiatric symptoms
  - Psychopharmacologic skills
  - Psychotherapeutic skills
  - Medical knowledge

Selected References...


Selected References...

Thank You...

• Questions?
CATATONIA, NMS, AND SEROTONIN SYNDROME

Christopher M. Celano, MD
Catatonia, NMS, and Serotonin Syndrome

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Assistant Professor of Psychiatry, Harvard Medical School

October 22, 2020

Catatonia: How common is it?

- 7.8-9.0% prevalence rate
  - Highest rates in non-psychiatric (i.e., medical) settings and in patients undergoing ECT.
- 1.6-5.5% of all patients seen on psychiatry consultation service
  - Prevalence higher for older patients
- Up to 46% of cases may have etiology that is not primarily psychiatric


When are you called?

- Staff reports the patient is “Playing POSSUM”
- Perseveration (speech or behavior)
- Oppositionality to all requests
- Speech that trails off or is whispered
- Slowed response to questions or commands
- Undernourished (reports of decreased PO intake)
- Motionless but awake
Diagnosing Catatonia: DSM-5

DSM-5 requires 3 or more of the following:

- Catalepsy
- Waxy flexibility
- Stupor
- Agitation
- Mutism
- Negativism
- Posturing
- Mannerisms
- Stereotypies
- Grimacing
- Echolalia
- Echopraxia

American Psychiatric Association 2013

Bush-Francis Rating Scale

- Excitement
- Immobility/stupor
- Combativeness
- Autonomic Abnormality
- Impulsivity
- Mutism
- Staring
- Posturing/catalepsy
- Grimacing
- Echopraxia/echolalia
- Stereotypy
- Mannerisms
- Verbigeration
- Rigidity
- Negativism
- Waxy flexibility
- Withdrawal
- Automatic Obedience
- Mitgehen
- Gegenhalten
- Ambitendancy
- Grasp Reflex
- Perseveration

Bush 1996

Challenges with Diagnosis

- Clarifying specific symptoms can be difficult
  – Rrigidity vs. gegenhalten vs. negativism
- Inconsistency between scales
- Symptoms occur on a spectrum
- Wide variety of manifestations
Prototypes of Catatonia

- The Distant Mute
  - Mutism, immobility, interpersonal withdrawal
  - Team may be concerned this is volitional
- The Waxy Stiff
  - Catalepsy, waxy flexibility, rigidity
  - Often identified by physicians; may misattribute to psychiatric illness
- The Broken Record
  - Echophenomena, verbigeration, hyperactivity
  - Often misdiagnosed as delirium
- The Stubborn Grouch
  - Negativism, repetitive movements, excitement
  - Medical workup often not completed due to lack of cooperation.

Azarn 2013

Pathophysiology of Catatonia

- Disruption in the tracts connecting the basal ganglia and the cortex, leading to relative hypodopaminergia.
  - Dorsolateral prefrontal and anterior cingulate / medial orbitofrontal → akinetic mutism, dysautonomia
  - Lateral orbitofrontal → imitative and repetitive behaviors
  - Supplementary motor / motor / posterior parietal → rigidity, initiation and termination of movement
- Hyperactivity of the supplementary motor area and presupplementary motor area → motor control, initiation and inhibition of movement

Fricchione 2008, Walther 2019

Pathophysiology of Catatonia

- GABA and serotonin may be involved
  - The dopaminergic projections in the brain are modulated by GABA-ergic and serotonergic neurons.
  - Benzodiazepines (GABA-A agonists) are helpful
  - GABA-B agonists (baclofen) are harmful and can induce catatonia
  - Serotonergic medications also may induce catatonic symptoms.
- Glutamate may also play a role
  - Anti-NMDA receptor encephalitis can cause catatonia.
  - NMDA receptor antagonists have been used as treatments in some cases.

Mann 1986, Rogers 2019
Evaluating Catatonic Patients

- Observe patient while trying to engage in conversation.
- Scratch your head in an exaggerated manner.
- Examine the patient’s arms for cogwheeling. Move the arms with alternating lighter and heavier force.
- Move patient’s arm into different positions and observe whether they remain in position.
- Ask the patient to extend his/her arms. Place one finger beneath each hand and try to raise it slowly after stating, “Do not let me raise your arms.”

Evaluating Catatonic Patients

- Extend your hand and state, “Do not shake my hand.”
- Reach into your pocket and state, “Stick out your tongue. I want to stick a pin in it.”
- Check for grasp reflex.
- Check the chart for reports from prior 24 hours. Check for PO intake, VS, and incident.
- Observe the patient indirectly daily to observe for other catatonic symptoms.

Potential Causes of Catatonia

- Medical Illness
  - Seizures
  - CNS structural damage
  - Encephalitis (e.g., anti-NMDA) or other CNS infection
  - SLE with or without cerebritis
  - Disulfiram
  - Phencyclidine
  - Neuroleptic exposure
  - Corticosteroid exposure
  - Porphyria
  - Post-partum state
  - Iron deficiency

- Psychiatric Illness
  - MDD
  - Bipolar Disorder
  - Psychotic disorders

Carroll 1994, Denysenko 2015
**Workup for Catatonia**

- Complete Blood Count, Comprehensive Metabolic Panel
- Creatine Kinase (to look for rhabdomyolysis)
- Iron studies
- Toxicology screens
- Other bloodwork as indicated
  - Cultures
  - HIV
  - Paraneoplastic panel
  - Autoimmune studies
- Consider head CT, brain MRI, and EEG

**Catatonia vs. Delirium**

- DSM-5 states that catatonia cannot be diagnosed when symptoms are present exclusively in the setting of delirium
- Clinical practice suggests that most patients with neuromedical etiology for catatonia also have delirium
- 12-37% of patients with delirium may have features of catatonia
  - More commonly associated with hypoactive delirium and more common in women
  - Common features of catatonia include excitement, immobility, mutism, negativism, staring, withdrawal

Oldham 2015, Grover 2014

**Subtypes of Catatonia**

- DSM-5 specifies:
  - Hyperactive
  - Hypoactive
  - Mixed level of activity
- Malignant Catatonia (aka Lethal Catatonia)
  - Characterized by severe muscle rigidity, hyperthermia, and autonomic instability
    - Delirious Mania
    - Neuroleptic Malignant Syndrome
    - Serotonin Syndrome

APA 2013, Mann 1986
Management of Catatonia

- Identify the underlying cause.
  - Perform full psychiatric evaluation to identify mood or psychotic disorders.
  - Obtain collateral information about patient’s mood and behavior prior to admission.
  - Perform medical workup, especially for those with other symptoms of medical illness.
- Frequent vital signs
- Supportive care
- Remove possible culprit medications
- Initiate treatment with medications or ECT

Treatment of Catatonia: Benzodiazepines

- Intravenous lorazepam is greatly preferred
  - Quick onset of action
  - Despite a shorter half-life than other benzos, effective clinical activity may be longer because tissue distribution is less rapid and extensive
  - Also demonstrates a higher binding affinity for GABA_A receptor
- Initial dose of 2mg
  - Follow-up dose based on response and sliding scale of suspicion
- If established efficacy or diagnosis certain, continue with standing regimen
  - 8-24mg/day is typical
  - Taper very slowly after improvement

Denysenko 2015

Treatment of Catatonia: ECT

- Effective in 85-90% of cases; 60% of cases that fail medication
- Should be considered for failure to respond to lorazepam in 48-72 hours, malignant symptoms, excited subtype
- Maintenance ECT often required
Treatment of Catatonia: Alternatives

- NMDA receptor antagonists
  - Amantadine (18 cases)
    - May also have dopamine agonist activity
    - Start at 100mg daily
    - Titrate by 100mg every 3-4 days to maximum of 400mg in 2-3 divided doses
  - Memantine (9 cases)
    - Start at 5mg bid
    - Increase to 10mg bid if ineffective
- Antiepileptic medications
  - Carbamazepine (7 cases)
    - 100-1000mg daily
  - Valproic acid (5 cases)
    - 600-4000mg daily
  - Topiramate (4 cases)
    - 200mg daily

Beach 2015

Treatment of Catatonia: Alternatives

- Antipsychotic medications
  - Hypothesized to work through 5-HT1A agonism and 5-HT2A antagonism, which may lead to increased dopamine in the prefrontal cortex.
  - Aripiprazole (9 cases)
    - 3-30mg daily
  - Clozapine (9 cases)
    - 150-300mg daily
  - Olanzapine (7 cases)
    - 2.5-20mg daily
  - Risperidone (2 cases)
    - 0.5-8mg daily
  - Ziprasidone (2 cases)
    - 40-160mg daily

Beach 2015

Treatment Algorithm

- Intravenous lorazepam (initial test dose, then 6-8mg daily)
- Electroconvulsive therapy (at least 6 treatments)
- Glutamate (NMDA) antagonist (amantadine or memantine)
- Antiepileptic medication (carbamazepine or valproic acid)
- Atypical antipsychotic (aripiprazole, olanzapine, clozapine)

Beach 2015
Neuroleptic Malignant Syndrome (NMS)

- No DSM diagnostic criteria
- Expert panel criteria:
  - Exposure to dopamine antagonist (or removal of dopamine agonist) within past 72 hours
  - Hyperthermia
  - Rigidity
  - Mental status alteration
  - CK elevation (>4 times upper limit of normal)
  - Autonomic instability
  - Hypermetabolism
  - Exclusion of other medical or substance-induced causes

Guerra 2011

NMS: Complications and Treatment

- Complications
  - Rhabdomyolysis
  - Seizures
  - Respiratory failure
  - Acute kidney injury
  - Sepsis
  - Acute MI
  - Acute liver failure
  - Pulmonary embolism
- Mortality rate 5.6%
- Treatment
  - Remove offending agent
  - Similar treatment to catatonia

Modi 2015

Serotonin Syndrome (SS)

- Sometimes considered a subtype of malignant catatonia
- Symptoms:
  - Spontaneous clonus
  - Inducible clonus AND agitation or diaphoresis
  - Ocular clonus AND agitation or diaphoresis
  - Tremor AND hyperreflexia
  - Hypertonia AND hyperthermia AND ocular clonus or inducible clonus
- Classically induced by combination of MAOI with serotonergic medication
- Now more commonly seen with polypharmacy or overdose
- Clues to Serotonin Syndrome
  - Look for it in patients with antidepressant overdose
  - Look for it in any patient on >4 psychiatric medications
  - Consider it in all catatonic patients

Dunkley 2003
Treatment of Serotonin Syndrome

- Supportive treatment and wash-out is usually all that is needed
  - May use benzodiazepines to manage agitation or if catatonic symptoms are present
  - Short-acting antihypertensives
- If this is not working, can consider cyproheptadine (5-HT1A and 5-HT2A antagonist)

References


References

References

NEUROCOGNITIVE ASSESSMENT AT THE BEDSIDE

Judith Restrepo, MD
Neurocognitive Screening

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Attending in Consultation-Liaison Psychiatry – Massachusetts General Hospital
Instructor in Psychiatry – Harvard Medical School
October 2020

Screening objectives

• To guide diagnostic hypotheses & further screening/testing
• To facilitate more accurate diagnoses
• To guide appropriate treatment (medication and supportive)
• To help patients, families, and co-treating physicians understand symptoms

What is bedside neuropsychological screening?

• A judiciously employed, systematic assessment of a pt’s arousal, cognitive, perceptual, and affective statuses/capabilities

• Formal neuropsychiatric testing is for neuropsychologists
  — More rigorously quantitative
  — Less diagnostically oriented
Order of Operations

- Known medical/neurologic contributions
- Level of arousal
- Attention + Complex attention
- Language and visuospatial
- Memory
- Executive function

Hierarchy of Functions

*State-dependent vs Channel-dependent functions*

- Alertness/Arousal
  - Attention, Motivation
    - Language, Praxis, Object ID, Memory/Memories, Executive Fxn

STATE DEPENDENT ASSESSMENT
Arousal

- Maintenance of arousal is critical to assess cognition
- Importance often skinned/escapes notice
- Fluctuation can occur and this may be assessed at multiple points in time
- Three general disruptions
  - Hyperarousal
  - Hypoarousal
  - Mixed concerns (delirium)

Assessment of Arousal

- Always assume pt will not participate in exam
- Adaptation to environmental change
  - Response to verbal/visual stim
  - Move the patient (head of bed/arms legs)
- Activity
  - Maintenance of response
- Latency
  - Reaction times/consistency
- Task persistence
  - Completes tasks without direction

Level of Arousal

- Terms are often misused/misunderstood; describing state is preferred
- Common terms
  - Hyperarousal
    - Often looped in with agitation, hyperalertness, colloquial use of “manic”
  - Awake/alert
  - Somnolence/Lethargy
  - Obtunded
  - Stupor
  - Coma
Attention

- Does not exist without normal alertness
- Required for appropriate assessment for all following functions
- Considerations
  - Selective vs Sustained vs Directed
  - Attention vs Concentration vs Spatial

Assessing Attention

- Assessment often adequate by interview alone
- Many levels exist

<table>
<thead>
<tr>
<th>Initial Attention</th>
<th>Selective Attention</th>
<th>Concentration</th>
</tr>
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<tbody>
<tr>
<td>Automatic or voluntary orientation to sensory stimuli</td>
<td>Selection of stimuli from array of competing sensory stimuli</td>
<td>Maintenance of focus on stimuli to complete task</td>
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Schoenberg 2011

- Rule of thumb: bedside assessment should include vigilance, maintenance under distraction, and alternating focus

Motivation & Mood

- Aberrations of either can → false positives
- Esp. vulnerable to misinterpretation
- Assess by history & observation

- “Organic” mimics of idiopathic phenomena
  - Depression vs Apathy/Abulia
  - Blunted/inappropriate affect vs Dysprosodias
  - Affective lability vs Pathological affect

- ASK pt
- Compare spontaneous vs elicited (esp recent recall)
CHANNEL DEPENDENT FUNCTIONS

Language and Praxis

- Speech ≠ Language (dysarthrias; modalities)
  - Consider mechanics
- Fluent/Non-Fluent ≠ Sensical/Nonsensical

- Praxis
  - Many types; ideomotor screened
  - “Blow out a match,” “flip a coin,” etc.
  - Errors: inability, perseveration, vocalization, simulation w/body part

Assessing Language

- Expressive
  - Fluency
  - Articulation
  - Organization
- Receptive
  - Naming
  - Comprehension
- Repetition
- Prosody
Memory

- Includes encoding, storage and retrieval
- Intact sensory, motor, arousal and attentional skills are prerequisite
- Many individual factors affect performance
  - age, education
  - anatomy
  - material (i.e., Verbal, Visual)
- Should include recent memory and remote memory

Memory

- Content
  - Declaritive/Explicit: semantic (facts), episodic (events)
  - Implicit: procedural (skills); conditioning
- Timing
  - Immediate: working “memory”
  - Recent: min-days
  - Remote: weeks-years
- Encoding
  - Remote vs. anterograde

Assessing Memory

- Assessment must include
  - Learning
  - Immediate
  - Delayed
  - Recognition Format (is the problem with encoding or retrieval)
- Often part of extended mental status exam
  - Can include intermediate memory task
On the fly tests

- 3-Words, 3-Shapes
- Hidden $ variant
- List Recall
- Drawing Recall

3 words – 3 Shapes

Weintraub, (2013)

Executive function

- Frontal Lobes are most heavily involved (directly and indirectly)
  - Damage also impacts memory, motor, attention, language and comportment
  - Three syndromes
    - Dorsolateral
    - Orbitofrontal
    - Medial Frontal
Assessing Planning

- Collateral is often key as patients often lack awareness
- Disinhibition
  - Frontal lobe reflexes (release signs)
  - Contradictory verbal commands “don’t take this”
  - Go-no-go
- Motor and Sequencing
  - Perseveration (loops or ramparts)
  - Finger tapping
  - Luria
  - Rapid alternating movement
- Abstraction
- Organizational abilities
  - Clock

Examples of frontal-subcortical network dysfunction findings

Other channel-dependent functions

- Construction/visuospatial
  - R hemisphere & parietal – “big picture”
  - L hemisphere & frontal – details
  - Neglect ----- 2x simultaneous stimulation

- Gnosis
  - Distinguished from anoma by ability to use objects
Standardized screens

**MMSE**
- Orientation x10: Mixed function of attention, short term memory
- Registration x3: Attention
- Calculation/WORLD x5: attention/working memory
- Recall x3: Short term memory
- Language x5: name, repeat, read, write
- Construction x1
- Praxis x3

**MOCA**

Bedside screening in action

*Dementia Subtype Hypothesizing*

What’s next?

- You may be done
- Imaging
- EEG (for fine-grained delirium questions)
- Formal NPT
- Use findings to formulate questions & make predictions
References

PANEL DISCUSSION

Moderator: Theodore A. Stern, MD
Panelists: Christopher M. Celano, MD, Judith Restrepo, MD
THE RISK OF QTc INTERVAL PROLONGATION WITH PSYCHOTROPICS

Christopher M. Celano, MD
The Risk of QTc Interval Prolongation with Psychotropics

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Massachusetts General Hospital
Assistant Professor of Psychiatry, Harvard Medical School

October 22, 2020

Topics for Discussion

- QTc interval and its measurement
- Risk factors for QTc prolongation
- Relationships between psychiatric medications and QTc prolongation
- QTc monitoring in clinical practice

What is the QT interval?
How to Measure QTc

- Pick an appropriate lead on the ECG.  
  - Usually II, V2, or V3.
- Measure the QT interval.
- Measure the heart rate or RR interval.
- Calculate the QTc.

Measure the QT interval

![ECG waveform with annotations](image)

9 boxes + 10 msec
QT = 370 msec

QT intervals are HR-dependent

![Graph of QT interval vs. heart rate](image)
Measure the RR interval

17 boxes + 10 msec
RR = 690 msec

QT = 370 msec

Correction Formulae

<table>
<thead>
<tr>
<th>Method</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bazett</td>
<td>$QTc = \frac{QT}{\sqrt{RR}}$</td>
</tr>
<tr>
<td>Fridericia</td>
<td>$QTc = \frac{QT}{\sqrt{RR}}$</td>
</tr>
<tr>
<td>Framingham</td>
<td>$QTc = QT + 0.154 \times (1000 - RR)$</td>
</tr>
<tr>
<td>Hodges</td>
<td>$QTc = QT + 1.75(\text{HR} - 60)$</td>
</tr>
</tbody>
</table>

QTc Correction Methods

Patel 2016
Normal Ranges

<table>
<thead>
<tr>
<th>Rating</th>
<th>Adult Men</th>
<th>Adult Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; 430 msec</td>
<td>&lt; 450 msec</td>
</tr>
<tr>
<td>Borderline</td>
<td>431-450 msec</td>
<td>451-470 msec</td>
</tr>
<tr>
<td>Prolonged</td>
<td>&gt; 450 msec</td>
<td>&gt; 470 msec</td>
</tr>
</tbody>
</table>

However, we generally become more concerned if QTc > 500 msec.

Moss 2003

Why do we worry about QTc prolongation?

• Torsades de pointes (TdP)
  – “Twisting of the points”
  – May lead to sudden syncope or dizziness

Risk Factors for QTc Prolongation

• Female gender
• Increased age
• Congenital Long QT Syndrome
• Structural Cardiovascular Disease
• Electrolyte abnormalities
• Hepatic dysfunction
• Other medications that prolong QTc
• Metabolic inhibitors

Beach 2013
Psychiatric Medications and QTc

• Antipsychotic Medications
  – First Generation
  – Second Generation
• Antidepressants
  – SSRIs
  – Tricyclic Antidepressants
  – Atypical Antidepressants
• Other psychiatric medications

Antipsychotic medications

• Nearly all antipsychotics prolong QTc, but the degree of prolongation differs substantially among agents.
  • Haloperidol
    – In oral form, haloperidol leads to QT prolongation that is similar to aripiprazole, quetiapine, and asenapine.
    – Intravenous form may lead to higher risk of QTc prolongation, with some caveats.
    – FDA recommends cardiac monitoring for patients receiving intravenous haloperidol.


Antipsychotic Medications

• Second generation antipsychotics

![Graph showing efficacy of antipsychotics vs placebo](image)

Huhn 2019
Antipsychotic Medications

- Second generation antipsychotics
  - Highest risk: ziprasidone and iloperidone
  - Lowest risk: aripiprazole and lurasidone
- FDA warnings
  - Ziprasidone (black box)
  - Quetiapine
  - Intravenous haloperidol
- There may be a dose-response relationship for antipsychotics and QTc, but evidence is mixed.

Antipsychotic Medications and Mortality

- Both first- and second-generation antipsychotics have been linked to ventricular arrhythmias or sudden cardiac death.
  - Case-crossover study (N=17,718)
    - OR=1.53
      - Haloperidol, prochlorperazine, thioridazine, quetiapine, and risperidone were associated with increased risk.
- FDA black box warning for second-generation antipsychotics in elderly patients with dementia.
Antidepressants and QTc

- **SSRIs**
  - Initially thought to be quite safe
    - SADHART, ENRICHD, CREATE
  - FDA warnings:
    - Initial
      - Citalopram should not be prescribed at doses greater than 40mg
      - Citalopram should not be used at doses >20mg in those with liver dysfunction or over age 60
    - Revision
      - Citalopram is not recommended at doses greater than 40mg
      - Citalopram should be discontinued in anyone with QTc>500 ms


Citalopram and QTc

<table>
<thead>
<tr>
<th>Medication and dose</th>
<th>QT prolongation (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram 20mg daily</td>
<td>8.5 (6.2, 10.8)</td>
</tr>
<tr>
<td>Citalopram 40mg daily</td>
<td>12.6 (10.9, 14.3)</td>
</tr>
<tr>
<td>Citalopram 60mg daily</td>
<td>18.5 (16.0, 21.0)</td>
</tr>
<tr>
<td>Moxifloxacin 400mg daily</td>
<td>13.4 (10.9, 15.9)</td>
</tr>
</tbody>
</table>

US FDA 2011

Escitalopram and QTc

<table>
<thead>
<tr>
<th>Medication and dose</th>
<th>QT prolongation (95% CI)</th>
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</thead>
<tbody>
<tr>
<td>Escitalopram 10mg daily</td>
<td>4.5 (2.5, 6.4)</td>
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<tr>
<td>Escitalopram 20mg daily</td>
<td>6.6 (5.3, 7.9)</td>
</tr>
<tr>
<td>Escitalopram 30mg daily</td>
<td>10.7 (8.7, 12.7)</td>
</tr>
<tr>
<td>Moxifloxacin 400mg daily</td>
<td>9.0 (7.3, 10.8)</td>
</tr>
</tbody>
</table>

US FDA 2012
Effects of SSRIs on QTc

Castro 2013

<table>
<thead>
<tr>
<th>Medication</th>
<th>N</th>
<th>Difference in QTc (ms)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram</td>
<td>696</td>
<td>10.58</td>
<td>.002</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>360</td>
<td>7.27</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>135</td>
<td>4.50</td>
<td>.32</td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>27</td>
<td>-5.00</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>1486</td>
<td>-1.04</td>
<td>.67</td>
</tr>
<tr>
<td>Sertraline</td>
<td>369</td>
<td>3.00</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>SSRIs</td>
<td>3,079</td>
<td>6.10</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>TCAs</td>
<td>1,587</td>
<td>10.01</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Beach 2014

SSRIs and Ventricular Arrhythmias

- Evidence is less clear
  - Danish case-time-control study

Weeke 2012
SSRIs and Ventricular Arrhythmias

- Tennessee Medicaid Cohort Study
  - Retrospective cohort study of 54,220 patients receiving high dose citalopram (>40mg daily) or escitalopram (>20mg daily) or equivalent doses of other SSRIs.
  - Neither citalopram nor escitalopram had higher risks of sudden unexpected death or all-cause mortality than other SSRIs.
- Patient-level meta-analysis for escitalopram
  - Escitalopram led to mild 3.5msec increases in QTc, compared to placebo.
  - Rates of cardiovascular side effects were similar between escitalopram and placebo.

Ray 2017, Thase 2013

Tricyclic Antidepressants and QTc

- Tricyclic antidepressants
  - Affect sodium, calcium, and potassium channels
  - Generally are considered to be higher risk for QTc prolongation than SSRIs
  - Have other cardiovascular side effects as well

Atypical Antidepressants and QTc

- Venlafaxine
  - Minimal risk at therapeutic doses (1 case report), low risk in overdose (1%).
- Bupropion
  - Associated with QTc prolongation in overdose; possibly confounded by tachycardia
- Trazodone
  - Associated with mild QTc prolongation in overdose
- Mirtazapine
  - No clear QTc prolongation risk, though it has been associated with a higher risk of SCD or ventricular arrhythmias than paroxetine in one study
- Newest antidepressants (duloxetine, vilazodone, vortioxetine, levomilnacipran, desvenlafaxine, brexpiprazole)
  - Not associated with clinically meaningful QT prolongation

Beach 2013, Jasiak 2014, Allen 2020
Other Psychiatric Medications and QTc

- Lithium
  - Can cause QTc prolongation at levels > 1.2 mmol/L
- Anticonvulsants
  - Not associated with QTc prolongation
- Stimulants
  - Not associated with QTc prolongation
- Benzodiazepines
  - Not associated with QTc prolongation

Skills for QTc Monitoring in Practice

- Know how to calculate a QTc on an ECG.
  - Do not rely on the QTc measured by the machine.
  - Use the Fridericia or Hodge’s formula to correct for heart rate.
- Know the risk factors for QTc prolongation.
- Know which medications are higher-risk.
  - Antipsychotics: thioridazine, ziprasidone, possibly iloperidone
  - Antidepressants: citalopram, escitalopram, tricyclic antidepressants

When to monitor QTc

- Know when to monitor QTc.
  - For patients without significant risk factors and on lower-risk medications, no monitoring is needed.
  - For patients with significant risk factors or on a higher-risk medication, check QTc at baseline, then again at steady-state or when risk factors change (e.g., change in dose).
Association of Medicine and Psychiatry Algorithm

Risk Factors (individual risk score in parentheses)
- Female (1)
- Age >65 years (1)
- comorbidities (e.g., hypertension, diabetes, cardiovascular disease) (1)
- Electrical risks: QTc > 460 ms (2)
- Total Risk Score < 2
- No baseline CTC needed
- Total Risk Score ≥ 2
- Xiong 2020

Association of Medicine and Psychiatry Algorithm

- Check ECG prior to start of medication CTA
- Start lower-risk medication CTA (<30 days) if feasible, then check ECG in 3 months
- Risk score < 5: Obtain ECG in 2-4 weeks (if already on psychiatric medications)
- Risk score ≥ 5: Consider urgent/emergent cardiology referral
- CTA > 500 ms (M)
- CTA > 440 ms (F)
- Start medication; repeat ECG in 2-4 weeks. Repeat ECG when risk factors change or when increasing dose
- Asymptomatic risk factors

References

References


References


References

- Wu CS, Tsai YI, Tsai HJ. Antipsychotic Drugs and the Risk of Ventricular Arrhythmia and/or Sudden Cardiac Death: A Nation-wide Case-Crossover Study. *J Am Heart Assoc.* 2015;4(2).
Seizure Disorders and Non-Epileptic Seizures

Franklin King, MD
Seizure Disorders and Non-Epileptic Seizures

Franklin King IV, MD
Center for Neuroscience of Psychedelics
Center for Anxiety and Trauma Stress Disorders
Mass General Hospital

Overview

• Seizure Disorders
  – Definitions
  – Psychiatric symptomatology
    • Ictal, Peri-ictal, Inter-ictal
  – Treatment

• Non Epileptic Seizures
  – Diagnosis
  – Treatment

Psychiatric Symptoms in Seizure Disorders

• Psychiatric symptoms are common in all phases of seizures
• Anxiety is most common ictal phenomenon
• Depression is most common inter-ictal phenomenon
• Psychosis is associated with post-ictal phase in patients with chronic seizure disorder
Seizure Definitions

- Seizure is an abnormal paroxysmal discharge of cerebral neurons sufficient to cause clinically detectable events that are apparent to the patient or an observer
- Epilepsy is a chronic course of repeated, unprovoked seizures

Seizure Definitions

- Focal Seizure—starts in a particular part of the brain (i.e., the focus)

- Generalized Seizure—involves both hemispheres simultaneously

Seizure Definitions

- Focal Seizures (formerly called partial seizures)
  - May remain limited to focus (or particular hemisphere) or may spread to other hemisphere known as secondary generalization
  - Manifestations depend on part of brain involved
  - Described in terms of how they affect consciousness
    - Focal Seizures with impairment of consciousness or awareness (formerly complex partial seizures)
      - most common type in adults
      - frequently have associated neuropsychiatric phenomena
      - Temporal lobe epilepsy is one example
Seizure Definitions

• *Focal Seizure* manifestations
  – Sensory impairment
  – Hallucinations (gustatory, olfactory, auditory, visual or tactile)
  – Affective symptoms such as fear, anxiety & depression (rage is least common)
  – Automatisms
  – Déjà vu
  – Macropsia, micropsia, dissociation

Seizure Definitions

• *Generalized Seizures*
  – Associated with loss of consciousness or awareness
  – Range from 5-10 seconds of staring spells known as *absence seizures* (*petit mal*)
  – To the longer (3 mins) *generalized tonic clonic* (*grand mal*) which is generally followed by a post-ictal state

Psychiatric Manifestations

• Most common psychiatric manifestations differ in each of 3 seizures phases
  – *Ictal*
  – *Inter-ictal*
  – *Post-ictal*

• Differentiate from primary psychiatric diagnosis
  – proximity to seizure
  – repetitive nature (i.e., seizures generally present with similar symptomatology)
Psychiatric Manifestations

• Ictal
  – Most common with focal seizures (though may also occur with generalized)
    • Fear and anxiety are most frequent
    • Psychosis also seen (especially with TLE)
    • Important to distinguish from primary psychiatric disorder
  – Treatment is focused on underlying seizure disorder
    • Adjunctive SSRI’s, etc are not often helpful

Psychiatric Manifestations

• Post-ictal
  – Post-ictal psychosis comprises 25-30% of psychosis of epilepsy
  – Onset is average of 15-20 years after onset of epilepsy
  – Lucid interval (hours to days) followed by fluctuating:
    • Disordered thought
    • Paranoia
    • Hallucinations (auditory & visual)
    • Mania—grandiosity
    • Behavioral disturbances such as crying, laughing, disinhibition also common
  – Treatment is benzodiazepine +/- antipsychotic

Psychiatric Manifestations

• Antipsychotics with seizures
  – All lower seizure threshold
  – High potency generally less effect on seizure threshold—1st line
  – Atypicals such as risperidone are also okay
  – Clozapine is worst—generally avoid with seizures
Psychiatric Manifestations

- *Inter-ictal* (chronic)
  - Depression, anxiety and psychosis are most common
  - Rates of depression and suicide 4-5x greater in those with epilepsy
  - Risk factors include poor seizure control and focal seizure with impairment of awareness
  - Atypical features and/or dysthymia are common
  - Anxiety, panic, OCD may also be seen

Psychiatric Manifestations

- Treatments
  - AED’s
    - Lamotrigine, carbamazepine, valproate may help stabilize mood
    - Levetiracetam may cause irritability, worsen mood
    - Phenobarbital and topiramate may also worsen mood
  - Antidepressants
    - SSRI’s and TCA’s generally safe (avoid clomipramine)
    - Buspirone may lower seizure threshold
    - ECT
    - CBT and other behavioral treatments

Psychiatric Manifestations

- Virtually any psychiatric symptom can be seen with seizure
- Important to treat due to significant morbidity
Non-Epileptic Seizures

• Psychogenic non-epileptic seizures (PNES)
  – Formerly known as pseudoseizure or hysterical seizure
  – Occurs in approx 10% of patients with intractable seizures
  – ¾ are women
  – Many have history of sexual abuse
  – 25% have epileptic seizures

Non-Epileptic Seizures

• Distinguishing characteristics
  – Events occur with suggestion/provocation
  – Gradual onset and offset of symptoms
  – Responsiveness during event
  – Weeping, speaking, or yelling during the event
  – Asymmetrical clonic activity
  – Head bobbing or pelvic thrusting
  – Rapid kicking or thrashing
  – Prolonged duration of symptoms (> 3 minutes)
  – No EEG abnormalities during the event

Non-Epileptic Seizures

• Differential Diagnosis
  – General Medical Conditions
    • Transient ischemic attack (TIA)
    • Complicated migraine
    • Syncope
    • Hypoglycemia
    • Narcolepsy
    • Myoclonus (from metabolic disturbance)
  – Psychiatric Causes
    • Conversion disorder
    • Somatic symptom disorder
    • Dissociative disorder
    • Panic disorder (simulating partial seizures)
  – Volitional Deception
    • Factitious disorder (goal is to maintain the sick role)
    • Malingering (goal is to obtain secondary gain, e.g., disability income)
Non-Epileptic Seizures

• Presentation of diagnosis
  — Frame diagnosis positively (e.g., “no abnormal electrical activity, no need for AED’s”)  
  — Frame spells as functional problem  
  — Set the frame that symptoms will improve over time (less frequent, less severe, etc)  
  — Introduce the fact that stress and anxiety may make symptoms worse  
  — Acknowledge disability caused  
  — Describe treatment plan involving multiple specialities

- Non-Epileptic Seizures

• Treatment
  — Introduce as much psychiatric care as patient will allow (e.g., weekly therapy, psychoeducation, CBT)  
  — Treat adjunctive symptoms  
  — Regular appointments with neurology and PCP  
  — Regular physical exam, avoid diagnostic procedures  
  — Positive reinforcement when symptoms subside (i.e., continue treatment)  
  — Remain vigilant that epileptic seizures may be missed or may co-occur

Conclusion

• Both epileptic and non-epileptic seizures may present with psychiatric symptomatology  
• As psychiatrists, we play a key role in multiple domains:  
  — Recognizing potential epileptic seizures and referring to colleagues in neurology  
  — Treating inter-ictal and peri-ictal phenomena  
  — Diagnosing and being a key part of the treatment team in those with non-epileptic seizures
FACTITIOUS ILLNESS AND MALINGERING

Theodore A. Stern, MD
Factitious Illness and Malingering

Theodore A. Stern, MD
Chief Emeritus, Avery D. Weisman Psychiatry Consultation Service,
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Director, Office for Clinical Careers,
Massachusetts General Hospital;
Ned H. Casse Professor of Psychiatry in the field of Psychosomatic Medicine/Consultation,
Harvard Medical School;
Editor-in-Chief, Psychosomatics

Factitious Disorders: Definition

• Not real, genuine, or natural
• Characterized by:
  — Physical or psychological symptoms that are produced by the individual and are under voluntary control
• Behavior:
  — Acts have a compulsive quality

Diagnostic Categories

• Factitious disorder with psychological symptoms
• Chronic factitious disorder with physical symptoms (Munchausen’s syndrome)
• Atypical factitious disorder with physical symptoms
Factitious Disorder with Psychological Symptoms: Criteria

- Psychological symptoms are apparently under the individual’s voluntary control
- Symptoms are not explained by any other mental disorder
  — but may be superimposed on one
- The goal is to assume the “patient role”
  — it is not otherwise understandable in light of the environmental circumstances (e.g., malingering)

Factitious Disorder with Psychological Symptoms: Features

- Pan-symptomatic complex of psychological symptoms
  — worse when observed
- Claims of memory loss, hallucinations, dissociation, or suicidal ideation
- Suggestibility to addition of symptoms
- Provision of approximate answers
- Strong linkage with personality disorders and substance abuse

Factitious Disorder with Psychological Symptoms: Differential Diagnosis

- Dementia
- Psychosis
- Brief reactive psychosis
- Schizophreniform disorder
- Malingering
Chronic Factitious Disorder with Physical Symptoms

- Munchausen’s syndrome
  - First described by Asher in 1951 (Lancet)
  - Dedicated to Baron von Munchausen
- Alternative labels
  - Hospital hoboes
  - Hospital addicts
  - Malingerers
  - Kopenickades
  - Sufferers of Ahasuerus syndrome

Munchausen’s Syndrome: Characteristic Features

- Laparotomophilia migrans
- Hemmoragia histrionica
- Neurologica diabolica
- Dermatitis autogenica
- Hyperpyrexia pigmentatica

Munchausen’s Syndrome: Useful Pointers

- Multiplicity of scars
- Truculence and evasiveness
- Acute, but not entirely convincing, history
- Wallet with hospital cards
- Time of presentation that predicts care by less experienced staff
Munchausen’s Syndrome: Possible Motives (per Asher)

- Desire to be the center of attention
- Grudge against doctors and hospitals
- Desire for drugs
- Desire to escape from the police
- Desire for free room and board

Munchausen’s Syndrome: Differential Diagnosis

- True physical disorder
- Somatoform disorder
- Hysteria
- Malingering
- Schizophrenia
- Personality disorder
  - Antisocial or borderline

Munchausen’s Syndrome: Dynamics...A Need to Explain...

- Posing and pseudologia fantastica
- Medical arena for presentation
  - Physicians often central figures in childhood
  - Often works in medical profession
- Rootless wandering
  - Search for lost primary love object
- Masochistic self-injury
  - Identification with the aggressor
  - Mastery over early trauma
Munchausen’s Syndrome: Hospital Course

- Dramatic presentation
- Physicians mobilized
- Demands for attention
- Ambivalence manifest
- Hoax is discovered
- Anger erupts
- Discharge AMA without psychiatric consultation

Munchausen’s Syndrome: The MGH Experience

- General description
  - A lightning rod effect for similar cases
- Case examples
  - Gas gangrene
  - Insulinoma
  - Pheochromocytoma
  - Brain abscess

Munchausen’s Syndrome: Treatment

- Universal remedy
  - Till Eulenspiegel (1515)
- Create a rogues gallery
- Invite participation as pseudodoctors
- Apply psychotherapeutic principles
  - Be aware of countertransference
- Encourage psychiatric consultation
  - Attempt to prevent further harm
Munchausen’s Syndrome: Diagnostic Criteria

- Plausible presentation of physical symptoms
  - Under the individual’s voluntary control
  - Leading to multiple hospitalizations
- The individual’s goal is to assume the patient role
  - Not a manifestation of malingering

Moving forward: Conclusion

- Be prepared:
  - To make the diagnosis
  - To identify and manage countertransference reactions
  - To prevent further harm to the patient

Suggested References

Suggested References


Thank you..

- Questions?
- Comments?
Panel Discussion

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Panelists: Christopher M. Celano, MD, Franklin King, MD