PRACTICE OF MEDICINE

PSYCHIATRY

Introduction to Clinical Psychiatry: Major Psychiatric Syndromes and their Treatment

Winter 2011

Charles DeBattista, DMH, MD
Professor of Psychiatry
Department of Psychiatry and Behavioral Sciences
Chief of Psychopharmacology and Depression Research Clinics
Director of Medical Student Education in Psychiatry
Stanford University School of Medicine

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Course Administration

Course Director: **Charles DeBattista, D.M.H., M.D.**
Professor of Psychiatry
Department of Psychiatry and Behavioral Sciences
Chief of Psychopharmacology and Depression Research Clinics
Director of Medical Student Education in Psychiatry
Stanford University School of Medicine
723-8324 phone
debattista@stanford.edu

Course Coordinators:

Tomiko Nguyen
Administrative Coordinator
725-7673
tnguyen9@stanford.edu

Quynh Dang
Administrative Assistant
725-2769
qdang@stanford.edu

Course Information

Purpose:
The course serves as an introduction to psychopathology and major treatment modalities in Psychiatry.

Course Format:
The course will be compromised of lectures, readings, and a final exam.

Lectures:
The lectures are intended to compliment the syllabus and recommended textbook. Videotapes and case vignettes will be used liberally.

Readings:
(1) Required: The course syllabus
(2) Recommended: Andreason NC, Black DW. Introductory Textbook of Psychiatry. Fourth Edition. American Psychiatric Publishing Inc. Washington DC. 2006

Small Group Sessions:

Psychiatric Interviewing will be taught in small-group format concurrently with this class. You will be assigned a site at Stanford Hospital, the Palo Alto VA, Menlo Park VA or the Psychiatry Building. Attendance at Small Group Sessions is mandatory.

POM Psychiatry
Small Group Sessions

The objectives of this course are:

1. To learn and to practice the Mental Status Examination
2. To learn and to practice taking a psychiatric history
3. To be exposed to patients with significant psychopathology
4. To review treatment interventions in psychiatry

At the end of this course, you should be able to:

1. Assess a patient for a mood disorder (especially depression)
2. Assess a patient for an anxiety disorder
3. Assess a patient for suicidality

Loosely, the format of each session is as follows:

Interview	40 minutes
Presentation	5 minutes
Case Discussion	40-50 minutes
Special Topics Discussion	10-15 minutes each

Special Topics are meant for you to cover issues related to the previous week's patient that you find interesting and helpful in managing such patients.

Examples of Special Topics to research and briefly present:

Mood disorders	Dysthymic disorder vs MDD
	Atypical depression
	Neuroendocrine abnormalities in mood disorders
	Role of neurotransmitters in mood disorders
	Premenstrual dysphoric disorder
	Treatment strategies for bipolar disorder
	Medical disorders associated with depression
Schizophrenia	Neuroanatomical features of schizophrenia
	Case management of the schizophrenic patient
	Biochemical factors in schizophrenia

Personality disorders	Borderline Personality Disorder
	Three clusters of personality disorders
Substance abuse	Detoxification of alcohol, heroin,...
	Long-term treatment and management of addiction
PTSD	Current therapies in the treatment of PTSD
OCD	Current therapies in the treatment of OCD
Somatoform disorders	Somatization disorder
	Conversion disorder
	Hypochondriasis
	Body dysmorphic disorder
	Factitious disorder
	Malingering
Eating disorders	Medical complications of eating disorders
	Biological theories of eating disorders
	Treatment strategies for anorexia nervosa
	Binge eating disorder
Child psychiatry	Attention-deficit hyperactivity disorder
	Autism
	Mood disorders in children
Cognitive disorders	Comparison of dementia and delirium
	Causes of delirium
	Causes of dementia
Social issues in psychiatry	Mental illness in homeless population
	Psychologists prescribing meds
	Views on overprescription of psych meds
	HMO coverage of psychotherapy
	Cultural differences in presentation of psych disorders

Grading:

The final grade will be composed of:

- Participation in small group
- Adequate performance on Final exam

Final Exam Date: TBD

Format: The final exam will consist of multiple choice questions and a video vignette and maybe multiple-choice questions.

Practice of Medicine – Psychiatry
Lecture Schedule
Winter 2011

Date	Topic	Lecturer	Room
Tuesday, 1/4/11 11:00 – 12:00 pm	• Introduction to Psychopathology & DSM-IV • Mood Disorders (Part I)	DeBattista	LK 120
Tuesday, 1/4/11 1:15 – 2:15 pm	• Mood Disorders (Part II)	DeBattista	LK 120
Tuesday, 1/4/11 2:15 – 3:15 pm	• Antidepressants & Mood Stabilizers (Part I)	DeBattista	LK 120
Tuesday, 1/4/11 3:15 – 5:05 pm	Small Groups (Session #1)	Various	Various
Thursday, 1/6/11 10:00 – 11:00 am	• Antidepressants & Mood Stabilizers (Part II)	DeBattista	LK 120
Thursday, 1/6/11 11:00 – 12:00 pm	• Psychotic Disorders (Part I)	Smith	LK 120
Thursday, 1/6/11 1:15 – 2:15 pm	• Psychotic Disorders (Part II)	Smith	LK 120
Thursday, 1/6/11 1:15 – 2:15 pm	• Antipsychotic	Smith	LK 120
Thursday, 1/6/10 3:15 – 5:05	Small Groups (Session #2)	Various	Various
Tuesday, 1/11/11 1:15 – 3:05 pm	• Somatoform Disorders • Dissociative Disorders	Maldonado	LK 120
Tuesday, 1/11/11 3:15 – 5:05 pm	Small Groups (Session #3)	Various	Various
Thursday, 1/13/11 1:15 – 2:15 pm	• Eating Disorders	DeBattista	LK 120
Thursday, 1/13/11 2:15 – 3:15 pm	• Anxiety Disorders	DeBattista	LK 120
Thursday, 1/13/10 3:15 – 5:05 pm	Small Groups (Session #4)	Various	Various
Tuesday, 1/18/11 11:00 – 12:00 pm	• Sedatives/Hypnotics	DeBattista	LK 120
Thursday, 1/20/11 11:00 – 12:00 pm	• Marijuana, Hallucinogens, PCP	Lembke	LK 120

RACTICE OF MEDICINE

PSYCHIATRY
Small Group Sessions

Psychiatric Interviewing

Winter Quarter 2011

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SESSION 1 (January 4, 2011):

Interviewer: PRECEPTOR:_____
Presentation to Preceptor: STUDENT #1:_____

SESSION 2 (January 6, 2010):

Interviewer: STUDENT #2:_____
Presentation to Preceptor: STUDENT #3:_____
Special Topic Discussion: STUDENT #4:_____

SESSION - Make Up (January 7 , 2010):

Interviewer: STUDENT #:_____
Presentation to Preceptor: STUDENT #:_____
Special Topic Discussion: STUDENT #:_____

SESSION 3 (January 11, 2010):

Interviewer: STUDENT #5:_____
Presentation to Preceptor: STUDENT #6:_____
Special Topic Discussion: STUDENT #7:_____

SESSION 4 (January 13, 2010):

Interviewer: STUDENT #8:_____
Presentation to Preceptor: STUDENT #9:_____
Special Topic Discussion: STUDENT #10:_____

SESSION - Make Up (January 14, 2010):

Interviewer: STUDENT #:_____
Presentation to Preceptor: STUDENT #:_____
Special Topic Discussion: STUDENT #:_____

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	HMO coverage of psychotherapy
	Cultural differences in presentation of psych disorders

Psychiatry and Behavioral Sciences

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Maps and Directions

Psychiatry Building

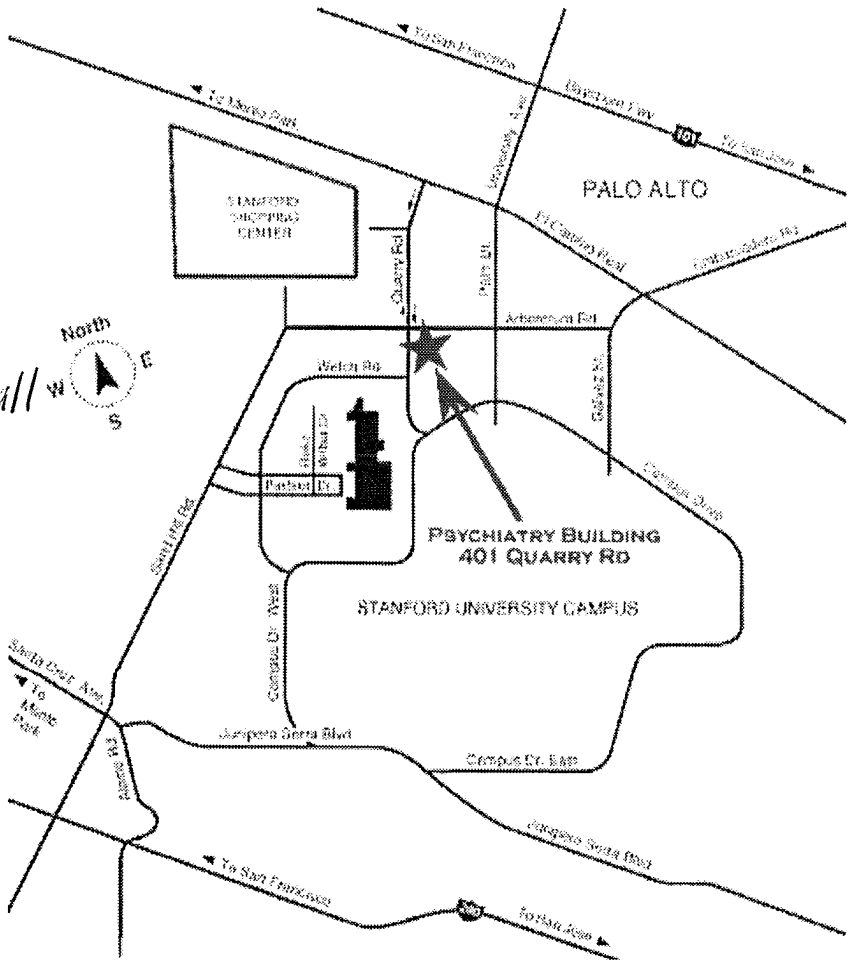
The Department of Psychiatry and School of Medicne are loacted at the northwest corner of Stanford's campus in Palo Alto, California, roughly midway between San Francisco to the north, and San Jose to the south. The San Francisco Bay Area is served by three major international airports: San Francisco, San Jose, and Oakland. Shuttle and bus service to Stanford is available from nearby airports. (NB: I-280 is better during rush hour and Oakland is not very convenient to Stanford).

MORE DIRECTIONS

- Palo Alto Veterans Administration
- Menlo Park Veterans Administration
- Stanford University Maps
- Bay Area maps on Expedia

G2 unit

Take the escalator by the gift shop to the second floor Turn left and go to the end of the hall



From San Francisco Airport: Either take US-101 SOUTH (direction San Jose) OR I-380 WEST I-280 SOUTH (San Jose) and follow the directions below.

Psychiatry and Behavioral Sciences

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From San Jose Airport: take either US-101 NORTH (San Francisco) OR I-880 SOUTH to I280 NORTH (San Francisco) and follow the directions below.

From Oakland Airport: take I-880 SOUTH (San Jose) to CA-92 WEST (Half Moon Bay) to US- SOUTH (San Jose) and follow the directions below.

Directions from US 101:
Take the University Avenue exit to the west (toward Palo Alto /Stanford). As you enter the gates Stanford the road will change names to Palm Drive. Continue on Palm Drive to Arboretum,
Turn RIGHT at the traffic light. onto Arboretum.
Turn LEFT at the next light, onto Quarry Rd.
Turn LEFT at first light (Vineyard) into parking lot (The Outpatient Psychiatry Building is located a 401 Quarry Rd., at the corner of Arboretum and Quarry Rd, across from Andronico's Market)

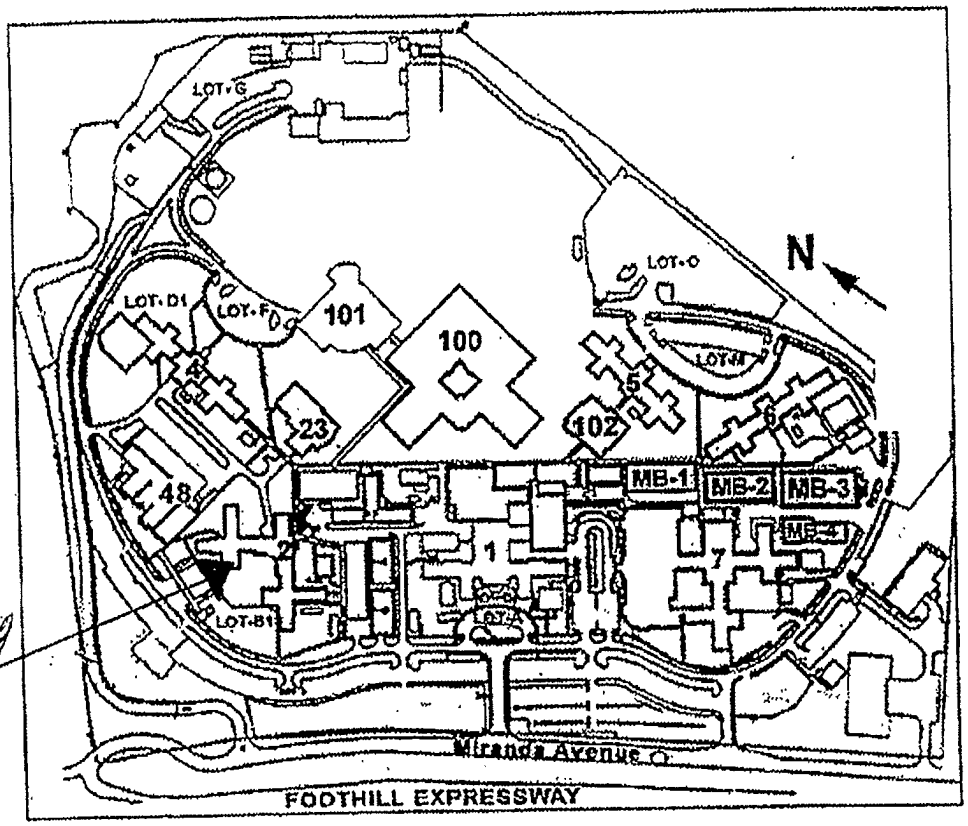
Directions from Interstate 280:
Take the Sand Hill Road exit (EAST: towards Palo Alto /Stanford) until Arbortetum
Turn RIGHT onto Arboretum
Turn RIGHT onto Quarry Rd. (first light past Crate & Barrel)
Turn LEFT at first light (Vineyard) into parking lot
(The Outpatient Psychiatry Building is located at 401 Quarry Rd., at the corner of Arboretum anc Quarry Rd, across from Andronico's Market)

Palo Alto Veterans Administration

Palo Alto Division
3801 Miranda Avenue
Palo Alto, CA 94304-1290
(650) 493-5000

The Palo Alto campus is administrative headquarters for the VA Palo Alto Health Care System (VAPAHCS). The distance between the Palo Alto Campus and other campuses is (miles): Menlc Park (7), Livermore (40), San Jose (25), and Monterey (90). As a tertiary care hospital with an annual budget of over $280 million, VAPAHCS provides state-of-the-art emergency, inpatient an outpatient treatment; complete with the latest diagnostic technology like Computerized Axial Tomography (CT), Magnetic Resonance Imaging (MRI) and Positron Emission Tomography (PE scans. Patient Care provided at the Palo Alto Campus is supported by the expertise of more thai 50 different clinical specialties. The Palo Alto VA administers an extensive $420 million research program, consistently one of the largest in the VA Health Care System. Researchers are investigating major health issues such as Alzheimer's cancer and AIDS. The Palo Alto division is home to a variety special regional treatment centers including: Brain Injury Rehabilitation, Geriat Research Education & Clinical Center, Rehabilitation Research and Development Center, Spina Cord Injury Center and Western Blind Rehab Center.

PAVA



Building 2

Palo Alto V.A.
3801 Miranda Avenue
(off Foothill Expressway)

From Stanford University, turn LEFT onto JUNIPERO SERRA.
JUNIPERO SERRA becomes FOOTHILL EXWY.
Turn LEFT onto HILLVIEW AVE.
Turn RIGHT onto MIRANDA AVE.

2B1 is located in BUILDING 2 on the first floor

2B2 is located in BUILDING 2 on the second floor.

Psychiatry and
Behavioral Sciences

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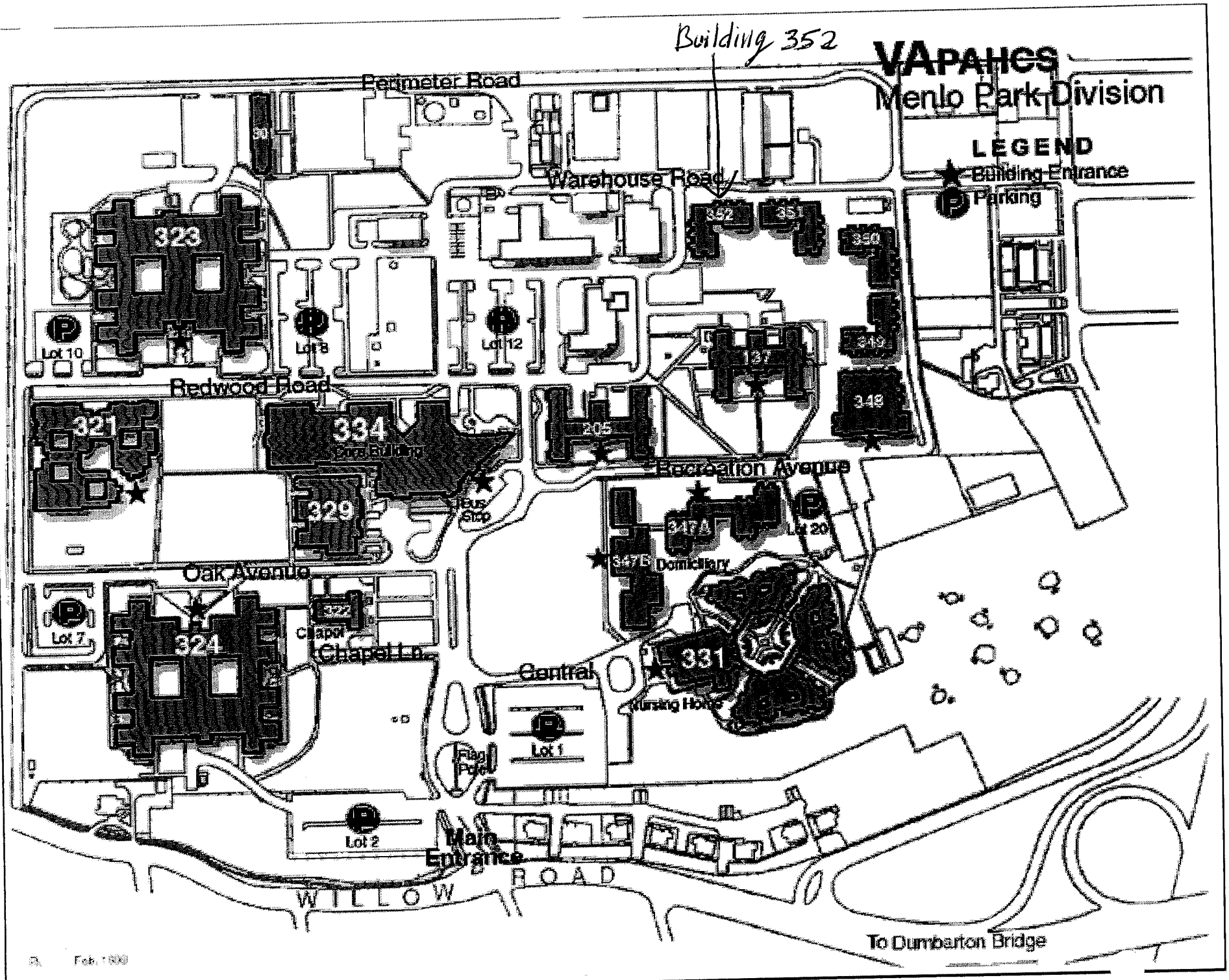
Directions From US-101:
Take Embarcadero Rd / Oregon Expressway exit
Keep LEFT at the fork in the ramp
Merge onto Oregon Expressway
Continue on Oregon Expressway, it becomes Page Mill Rd.
Turn LEFT at Foothill Expressway
Turn LEFT onto Hillview
Turn RIGHT onto Miranda Ave.
The Palo Alto VA is on your LEFT

Directions From I-280:
Take the Page Mill Rd. exit
Turn RIGHT at Foothill Expressway
Turn LEFT onto Hillview Ave.
Take immediate RIGHT onto Miranda
Ave.

Menlo Park Veterans Administration

Directions From US-101:
Take the WILLOW RD. Exit (Towards Menlo Park
The Veterans Hospital is 1/4 mile on the right of WILLOW.)

The Menlo Park Division, located on 95 beautifully landscaped acres at 795 Willow Road near Highway 101, offers a number of outpatient psychiatry, long term care and special programs providing a wide range of health services to veterans. Mental Health provides evaluation and


Building 352
VAPAHCS
Menlo Park Division
LEGEND
Building Entrance
Parking
Perimeter Road
Warehouse Road
323
Lot 10
Lot 8
Lot 12
Redwood Road
321
334
Core Building
329
Bus Stop
205
Recreation Avenue
347A
Lot 20
347B Domiciliary
Oak Avenue
Lot 7
324
322
Chapel
Chapel Ln.
Central
331
Nursing Home
Lot 1
Flag Pole
Lot 2
Main Entrance
WILLOW ROAD
To Dumbarton Bridge

Suggested Flow of Questions

Step 1: Important Interviewing Techniques

- One empathic comment during the first 5 minutes of the interview
- Progression of the interview should be from general to more specific questions
- For most patients, have the patient explain the main story until it makes sense to you

Keep in mind the following focuses

* Here and now: concentrate on current circumstances and how they may be related

* Look for precipitating stressors, such as change in relationships with family, friends, at work; financial stressors; a change in physical health; trouble attaining goals; personal safety issues

Step 2: Psychiatry Review of Systems

Minimally, your review of systems should check for depressive symptoms (SIG E CAPS), suicidality and homicidality/violence, alcohol and drug history, and cognitive deficits.

Step 3: Past Medical History

Include: Thyroid disease, sexually transmitted disease history, HIV history and risk factors, history of head injury, falls, seizure history

Step 4: Past Psychiatric History

-History of psychiatric problems (includes alcohol and drug problems)
-History of past psychiatric treatments, hospitalizations, and medications
-History of suicide attempts
-Legal history and history of violence
-History of physical and sexual abuse

Step 5: Family History of Psychiatric and Medical Problems (includes Alcohol and Drug Problems)

Step 6: Social History

-Place of birth, where one grew up
-Family members information
-Education
-Occupation
-Significant events for the patient
-Hobbies/Interests
-Tobacco Use

ALSO:
-Substance Use/Abuse
-Legal History
-Military History

Step 7: Mini Mental Status Exam

1. Suspicion of Depression:

Depressive Symptoms

SIG: E CAPS ("Prescribe Energy Capsules") plus **depressed or sad mood** is a mnemonic for the diagnostic criteria for a major depressive episode. Frequently, symptoms have a **diurnal pattern** (classically, worse in the morning than the evening).

Sleep:
Interest: (ANHEDONIA)
Guilt:
Energy:
Concentration:
Appetite:
Psychomotor retardation or agitation:
Suicidality:

Sample Screening Questions

- How has your mood been recently? How long have you felt this way?
- **SIG: E CAPS**
- Are your symptoms worse at a particular time of day?
- Have you noticed a seasonal pattern to your symptoms?
- It is not uncommon for people who are depressed to have unusual thoughts or experiences such as having unusual physical symptoms, hearing voices, or feeling suspicious. Has this happened to you?
- If patient has symptoms of psychosis, find out how long the psychotic symptoms (delusions/hallucinations) have been present and whether psychotic symptoms were present prior to or in the absence of the mood symptoms
- See Suicide Questions
- See Mania Questions
- See Alcohol and Drugs Questions (depressed when not using for some time?)
- History of Thyroid Disease/Symptoms
- Consider Cognitive Disorder Questions in the older patient
- Consider Anxiety Disorder and Obsessive-Compulsive Disorder Questions

Differential Diagnosis

- Major depression episode (unipolar vs. bipolar)
- Seasonal affective disorder
- Major depression with psychotic feature (psychotic depression)
- Dysthymia/cyclothymia
- Adjustment disorder with depressed mood
- Grief reaction

Differential Diagnosis (con't)

- Bipolar Disorders
- Schizoaffective Disorder
- Substance-induced mood disorder (due to either alcohol/drugs)
- Depression due to a General Medical Condition (thyroid disease)

Important Comorbid Disorders

- Anxiety disorders (OCD, panic disorder, social phobia)
- Personality disorders
- Substance abuse or dependence
- Dementia (in older patients)

2. Suspicion of Mania

Sample Screening Questions

- Are you feeling particularly energetic/highly productive, euphoric?
- How is your mood? Do you find yourself to be more happy than usual? more easily irritated?
- How's your sleep? Can you go several days with little or no sleep?
- Do you feel invincible or like an especially important person? Are you a religious person? Do you have a special relationship with God? Is there anyone who's after you or against you? Have you heard any voices recently? Do you find yourself planning things? Tell me more about this.
- Do people have a hard time understanding you? Do you find yourself going from one topic to another?
- Have you found yourself to be easily distracted? more focused and productive?
- Do you have racing thoughts?
- Have you been more talkative recently? Have people had trouble stopping you from talking?
- Have you found yourself taking a lot of risks recently? examples: spending money indiscriminately, buying things you later regret, going on trips on a whim, becoming promiscuous
- Depression Questions: SIG: E CAPS current and past history diurnal variation
- ETOH/Drug Questions (manic when not using for some time?)
- On any antidepressant medication recently?
- History of thyroid disease

Main Differential Diagnosis

Bipolar Spectrum Disorder (includes variants like cyclothymia)

Schizophrenia

Schizoaffective Disorder

Unipolar Depression (agitated type)

Psychotic Depression

Substance Induced Mood Disorder (ETOH/Drugs/Antidepressant medications)

Medical Condition (ie: hyperthyroidism)

Adult Attention Deficit Disorder (not in this packet)

3. Suspicion of Alcohol and Drug Use

Questions should relate to the following areas:

A) Alcohol and Drug Use are disorders of control, tolerance and withdrawal.

B) May Create/Induce/Mimic:

- -mood disorders
- -anxiety disorders
- -psychosis
- -cognitive impairment
- -non-psychiatric disorders(i.e. liver disease, anemia, seizures, arrhythmia's and many others)

C) Associated with:

- -manic episodes
- -head trauma secondary to falls/fights
- -impulsive behaviors such as suicide and homicide

D) Questions useful in eliciting an alcohol history are:

The Cage Questions

1. Have you ever tried to **cut down** on your drinking?
2. Are you **annoyed** when people ask you about your drinking?
3. Do you ever feel **guilty** about your drinking?
4. Do you ever take a morning **"eye-opener"**?

- 1 yes response: possible alcoholism
- 2-3 yes responses: high alcoholism suspicion index
- 4 yes responses: alcohol diagnosis likely

(Adapted from Ewing, JA. JAMA, 1984; 252: 1905-1907)

E) Other Questions (May adapt this section for drug use questions as well, except for alcohol's specific withdrawal syndrome):

Amount of alcohol used in one day
Frequency of use
Last use
Time period of use
Longest time gone **without any alcohol use**
Hx of **legal trouble (ie. DUI)**
Hx **blackouts**
Hx of **treatment**
Hx of **alcohol withdrawal**
-shakes/tremors
-hallucinations
-seizures
-major withdrawal or delirium tremens (DTs)

4. Suicide Questions

Note: A patient that is suicidal isn't necessarily clinically depressed. Patients with schizophrenia, drug and alcohol abuses, manias, personality disorders, adjustment disorders, and grief reaction can be suicidal, even in the absence of clinical depressive symptoms

Suicide Evaluation:

Five Parts of Suicidality

-ideation- thoughts of killing yourself

-intent- actual intention to do it

-plans- thinking of ways to kill yourself

-means- to carry plan out (i.e. gun at home)

-prior history of suicide thoughts and attempts

Risk Factors for Successful Suicide Attempts

"SAD PERSONS"

Sex (Male)

Age (<19, >45)

Depression, clinical

Previous Attempts

Ethanol (alcohol and drugs)

Rational Thinking Loss (i.e. psychotic)

Social Support System Lacking

Organized Plan

No spouse (single, separated, divorced)

Sickness (medical illness)

5. Homicide and Violence Questions

Note: If violence is in the history, consider **ruling out mania, alcohol and drugs, psychosis, dementia/delirium, and head injury** as possible etiologies.

<u>Seven Parts of Homicidality</u>

-**ideation**- thoughts of killing/harming someone

-**target**- is there a specific person(s) someone wants to kill (legally, you may have a duty to warn that person(s)

-**intent**- actual intention to do it

-**plans**- thinking of ways to kill someone

-**means**- to carry plan out (i.e. gun at home)

-**prior history** of homicidal thoughts and attempts/ history of violence

-history of **legal trouble**

6. Suspicion of Anxiety Disorder

Sample Screening Questions

- Are you usually a nervous/anxious person?
- Are you anxious all the time? Are you someone who is always worried about something?
- Do you ever have discrete episodes of anxiety? (if yes- consider panic disorder questions)

  Panic disorder is characterized by discrete, episodic panic attacks that can occur out of the blue frequently waking up someone from sleep and consist of both cognitive symptoms (i.e. fear of going crazy, of dying, that something terrible is going to happen, feeling of unreality) and physical symptoms (headaches, dizziness, shortness of breath, chest pain, palpitations, choking sensation, GI complaints, fear of losing control of one's bladder or bowels, numbness/tingling in the hands).

- Was there a specific event that led to your anxiety?
- Are there particular kinds of situations that make you anxious? Are there situations which you avoid because of anxiety?(if yes, consider social phobia, specific phobia, or agoraphobia questions)

  Social Phobia- an unrealistic fear of embarrassment or making a fool of oneself in a public setting such as public speaking, performing, urinating in a public urinal, blushing in public, eating in public.

  Specific Phobia- an unrealistic fear of a very specific situation interfering with social, occupational, or personal functioning. Fear of spiders, heights, or blood draws are examples.

  Agoraphobia- an unrealistic fear of places from which someone has no immediate escape route such as difficulty driving on bridges, a middle row seat in a movie theater, being in the middle of a crowd.

PTSD Questions:

History of out of the ordinary negative experience (combat, physical/sexual abuse) generally categorized as re-experiencing trauma, avoidance and hyperarousal.

- Have you had intrusive thoughts of the event (i.e., flashbacks, nightmares, things happening currently that reminds you of the event)?
- Are there things that you do to avoid thinking about the event?
- Does the patient have signs of hyper-arousal (i.e. startles easily, insomnia, short fuse/temper, irritability)?
- Alcohol and drug questions
- Depression questions
- Obsessive-compulsive disorder questions
- Recent med changes/ medical problems

7. Suspicion of Psychosis

Sample Screening Questions

- Have you had any unusual experiences? (general)
- Do you have thoughts that other people find difficult to understand?(general)
- Do you ever get the feeling that people are against you/ plotting against you/watching you/ trying to harm you/ trying to poison you? Is anyone after you like the FBI, CIA, Mafia? (paranoid delusions)
- Do you feel invincible or like an especially important person? (grandiose delusions)
- Are you a religious person? Do you have a special relationship with God? (religious delusions)
- Do you have concerns about your body that others have a hard time understanding? (somatic delusions)
- Do you ever hear voices when no one else is around? Could you tell me more about what the voices are like for you? What do they tell you? Do the voices ever tell you to do certain things (i.e. kill yourself) (command hallucinations)? visual, tactile (bugs crawling on you,) olfactory (i.e. smell of burning rubber), and gustatory hallucinations?
- Do you ever get any messages from the TV or radio?
- Do you feel that others pay you special attention? Are there hidden messages meant especially for you (i.e. on the TV or radio) (ideas of reference)
- Can you read other peoples' minds? Can they read your mind? (mind reading)
- Can other people hear your thoughts (thought broadcasting)
- Can you put thoughts in other peoples' heads? Could they put thoughts in your head? Can other people take away your thoughts? (thought insertion and withdrawal)
- Do you control other peoples' actions? Does anyone control your actions? (referred to as made acts)
- Alcohol and Drug questions
- Depression questions
- Mania questions
- Delirium and Dementia questions, if relevant
- Physical Disease (i.e. seizure, head trauma, brain mass, syphilis, HIV)

<u>Differential Diagnosis</u>

Schizophreniform disorder or schizophrenia
Schizoaffective disorder
Bipolar disorder
Psychotic depression
Alcohol or drug induced psychosis
Dementia
Delirium
Physical Disease- i.e. seizure disorder, head trauma, brain mass, encephalitis, neurosyphilis, and HIV

8. Suspicion of Obsessive-Compulsive Disorder

Common Obsessions

1. Contamination illness
2. Violent images
3. Fear of harming others/self
4. Perverse forbidden sexual thoughts, images, or impulses
5. Symmetry/exactness
6. Somatic Concerns (about one's body/body part(s))
7. Religious

Common Compulsions

1. Checking
2. Cleaning/washing
3. Counting
4. Hoarding/collecting
5. Ordering/arranging
6. Repeating

Sample Screening Questions

- Do you have any repetitive behaviors such as hand washing, checking doors to make sure its locked, checking the stove to make sure it is off, or counting behaviors?
- Are there any repetitive thoughts that are difficult for you to get out of your mind? such as avoiding something for fear of contamination (dirt, germs or chemicals)? repetitive sexual thoughts? repetitive thoughts that you or someone else may be harmed?
- Drug questions esp. history of amphetamine use
- Depression / SIG E CAPS questions
- Anxiety Disorder Questions
- Eating Disorder Questions (not in packet)

Differential Diagnosis

Drugs intoxication, i.e., amphetamines
Psychosis
Eating Disorders (Not in Packet)
Body Dysmorphic Disorder (Not in Packet)

High Comorbidity

Depression
Anxiety Disorders
Tourette's disease

9. Suspicion of Somatoform Disorder: Physical Complaints without a sensible physical basis

Sample Screening Questions

- Have you had a lot of physical problems in your life? Have these problems prompted you to see a doctor or doctors on many occasions? Have they significantly interfered with your life?
- Have doctors had a difficult time diagnosing your condition?
- How old were you when these symptoms began?
- Do a thorough review of systems especially Pain, GI, Sexual, and Neurologic symptoms
- Do a good Past Medical History/ Medication History
- Depression questions/ SIG E CAPS
- Anxiety disorder questions
- Alcohol and Drug questions
- Psychosis questions
- Find out if there was a history of physical or sexual abuse

Differential Diagnosis

- Somatoform Disorders:
  - Somatization disorder
  - Somatoform Pain disorder
  - Hypochondria's
- Factitious disorder
- Malingering (i.e. consider drug-seeking for pain meds, financial gain, getting out of work, wanting disability, wanting a place to stay for the night, etc.)
- Major depression
- Anxiety disorder
- Psychotic disorder
- Alcohol and Drugs
- Physical Disease

10. Disorders of Cognition (Delirium and Dementia)

"DEMENTIAM" mnemonic for remembering the reversible causes of delirium and dementia

Drugs including medications/**D**epression ("Pseudodementia")

Endocrine (thyroid disease, hyper/hypo-glycemia, Cushing's disease, Addison's disease)

Metabolic (increased/decreased CO2, hypoxia, hepatic encephalopathy, uremia from renal failure, increased/decreased Na, increased/decreased Ca, increased/decreased Phosphorus, increased/decreased Mg)

Ears/Eyes (vision/hearing impairments)

Nutritional deficiencies (B12 or thiamin deficiency)/Normal Pressure Hydrocephalus (dementia, ataxia, urinary incontinence)

Trauma/Tumor

Infection (i.e. HIV, syphilis)/Infarct (Stroke, MI)

Alcohol

Miscellaneous (i.e. epilepsy, collagen-vascular diseases (i.e. lupus), Wilson's disease)

Screening Questions (for individual or caregiver)

- Have you been having difficulty with your memory recently?
- Description of the cognitive impairment
- Length of time cognitive impairment has been going on
- How did the cognitive impairment(s) come on and change over time (suddenly, gradually, stepwise fashion (like a series of small strokes)
- Fluctuations of impairment with the time of day (i.e. is there a pattern)
- Ability to do one's own ADLs
- Behavior Problems/Personality Changes
- Wandering Problems
- Depression questions/ SIG E CAPS
- Psychosis questions, especially tactile hallucinations (bugs crawling on you), visual hallucinations and auditory hallucinations
- Neurologic Symptoms/Review of Systems
- Questions relating to the reversible causes of Dementia/Delirium

Mini Mental Status Examination (MMSE)*

POINTS

ORIENTATION (1 point each)

TIME: What is the Year ___
Season ___
Date ___
Month ___
Day of the Week ___ ___ of 5

PLACE: What is the Name of this place ___
Floor we are on ___
City ___
State ___
County ___ ___ of 5

IMMEDIATE RECALL (and REGISTRATION)

Name three different objects. Then ask the patient all three after you have said them. Ask patient to remember the words because you will ask them to name then again in a few minutes. Repeat until all three are learned. (1point each)

Baby Pen Boat ___ of 3

ATTENTION (1 point per letter or number correct)

Spell "WORLD" backwards.
-OR- Serial 7's: Subtract 7 from 100, then keep subtracting 7 from the answer you get. ___ of 5

DELAYED RECALL (1 point each)

Name the three objects repeated above (Baby, Pen, Boat) ___ of 3

NAMING (1 point each)

Show two different objects and ask the patient to name them.
e.g. – PENCIL WATCH ___ of 2

REPETITION

Repeat the following: "NO IF'S, AND'S, OR BUT'S" ___ of 1

THREE STAGE COMMAND ___ of 1

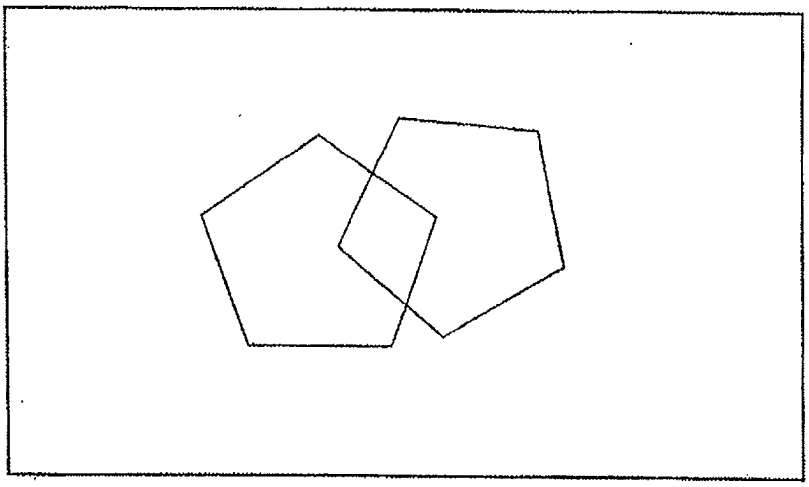
"Take a paper in your right hand, fold it in half, and then put it on the floor."

READING

Read the following silently and do what it says:
CLOSE YOUR EYES ___ of 1

COPYING

On a sheet of paper, copy the following diagram: ___ of 1



WRITING

On a sheet of paper, write a complete sentence. ___ of 1

Total = _____
(Maximum = 30)

Ranges and guidelines**:
Lowest quartile cut-off score by age range:

Age	MMSE score
40-49	29
50-79	28
80-89	26
90-	not normalized

*The MMSE is adapted from: Folstein MF, Folstein SE, and McHugh PR. "Mini-mental State:" A practical method for grading the cognitive states of patients for the clinician. *Journal of Psychiatric Research*, 1975, 12:189-198.

**From Bleecker et al., (1988). *Neurology*. 38:1565-1568.

Introduction to Psychiatry & DSM-IV

Introduction to Psychiatry

Charles DeBattista, M.D.

Prefatory Remarks

This course deals with a dimension of human suffering with which most of you have had limited contact so far. Psychiatric disorders may be as disabling and life threatening as any disorders you will encounter in your medical careers. The ravages of malignancies, diabetes, heart disease, lethal infectious disease are all horrible things, but in this course, we will discuss disorders that really cut to the core of who people are: their perceptions, their emotions, their behaviors.

The primary objective of this course is to overview psychopathology. We're going to learn about the major psychiatric syndromes such as schizophrenia, major depression, and bipolar disorder. The course will also present an introduction to psychiatric treatment, including pharmaceuticals, psychotherapy, ECT and other strategies that you'll see firsthand as you get to the clerkships. An important objective is for you to learn to take a psychiatric history and complete a mental status exam. The mental status exam is to psychiatry is what the general physical exam is to medicine. It is a critical tool and your first exposure to clinical medicine: how to evaluate somebody when you see them.

If you want to get a sense of who's going to be taking care of most psychiatric patients, look in the mirror. Primary care physicians and other non-psychiatric specialists manage most psychiatric conditions. It's the

relatively rare or compromised patient who ends up in the hands of psychiatrists; mostly, patients will be treated by internists, obstetrician/ gynecologists, pediatricians, family doctors, and so on. Of all the medicines you will prescribe in your careers, psychotropic medications will be among the most common. Of the 10 most commonly prescribed drugs in medicine currently, 4 are psychotropic agents. In this course, you will receive a general overview of the major classes of CNS drugs, including antidepressants, antipsychotics, mood stabilizers, and anxiolytics, with the understanding that you are concurrently learning CNS pharmacology. We also introduce you to the role of psychotherapy (talk therapy).

This course also prepares you for the behavioral science and psychiatry sections of Part I and II of the Boards. The USMLE is a general licensing exam, and expects that you have a certain fundamental knowledge applicable across specialties. The Boards emphasizes what is *common* and what is *catastrophic*. Part of the reason psychiatry is a preclinical course is because the National Medical Board considers knowledge of psychiatric treatment and psychopathology fundamental to first year clerks.

At the end of this course, the Final Exam will consist of Board-type questions. Since this is a clinical course, part of the final is also clinical. You will be asked to comment on the mental status, differential diagnosis, and treatment plan for one or more patients presented in video vignettes.

Resources

We highly recommend the Andreason text. It's designed for primary care physicians, so the material will also be meaningful to you in later years.

In this course we're also going to talk a lot about the DSM-IV. We do not require people to buy this heavy text, so we have included sections that correspond to the pathology we're talking about in the Syllabus. At the back of each section, we have included the DSM-IV diagnostic criteria for each of the major diseases we covered. Also, in the back of the syllabus you'll find a glossary of terms. The old exams and syllabus will be put up on the CWP.

Definition of a mental disorder

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) consists of standardized criteria used to define psychiatric disorders. The DSM-IV states that a mental disorder is a set of "psychological, emotional or behavioral symptoms that are associated with distress, disability or the increased risk of disability or death." A mental disorder occurs within a certain context: a situational context, a cultural context, and so on. For example, what represents psychopathology in North America might not represent psychopathology in Asia. The DSM-IV uses a descriptive approach, with characteristic signs and symptoms required to be present before a diagnosis is made. The use of specific criteria increases the reliability of the diagnosis process.

About 30% of people who walk into primary care clinics are there with a primary or co-morbid psychiatric problem: depression, anxiety, and so on. Being "upset" doesn't represent psychopathology. Let's say you're a Packer fan, and are so demoralized by a game you can't get out of bed. Does that represent psychopathology? Depends how often the Packers lose! If you miss the first day of work with every lost game, you could lose your job.

What if you didn't get out of bed for six months after a loss? That does represent significant disability and a level of distress far beyond the norms. There are patients who haven't gotten out of bed for ten years, and patients who have never left the house in twenty years. Such patients represent a significant level of disability.

Sadness is not necessarily a pathologic state. Anxiety is not necessarily a pathologic state. We all experience them as part of normal human experience. Imagine what life would be like if you never had any anxiety: you'd never show up for class, you might flunk out of school. Anxiety is an integral part of the human experience and one of many important motivating variables. What makes an emotion pathologic is the level of distress, disability or life threatening behavior it involves. If you're so sad you want to kill yourself, that's a problem.

To review then, we define **psychiatric illnesses** as disorders of emotional and/or behavioral functioning in which there is **disability** (i.e., significant impairment in an important area of functioning) and/or **distress** (i.e., painful symptoms). This definition allows us to distinguish sadness and grief (considered normal) from the state of major depression (considered abnormal and worthy of clinical intervention). Put another way, while all of us, at one time or another, feel emotional pain or distress, we consider these fluctuations in mental experience and functioning to be abnormal when they include disability and/or distress.

Classification of Disorders

Every major period since antiquity has had some way of trying to classify psychiatric problems. Indeed, descriptions have not changed dramatically since the time of Hippocrates. In the United States, the classification had its origins in census reasons, when the government was interested in how idiocy and lunacy were compromising the census. Forty years later there were other categories: mania, melancholy, and senility. The first cohesive attempt was made after WWII, when the military wanted to have a finer classification system in order to exclude certain people from service. The first Diagnostic and Statistical Manual of Mental Disorders (DSM-I) covered schizophrenia, mania, depression, and some anxiety disorders. Those original 40 pages of are now expanded to 900 pages. The DSM-I included the dominant Freudian psychoanalysis of the 1940's and 50's; this was removed by the DSM-III version. The DSM-IV lists seventeen major categories of mental disorders.

Making a diagnosis allows doctors to predict a certain prognosis, a certain course of disease. Repeated observations often make the diagnosis more valid. For instance, when a patient comes into the ER with psychosis, it is often unclear whether stimulant abuse or schizophrenia or mania or some other process is at play. Over time, depending on course and history, you may be able to make a diagnosis with a fair amount of validity. An illness carries a certain prognosis as well as a set of treatments that will work and not work. Diagnostic schemas are always evolving. Some definitions are controversial; some descriptions may not constitute a psychiatric disorder the medical community can agree on.

Up until now, to make a psychiatric diagnosis, doctors have relied on their own subjective observations as well as patient self-report. Not many outside tools exist to help make the diagnosis, but new tools will come along, including biologic markers and functional imagery. For instance, PET images of patients with depression show the brain to be hypermetabolic in the frontal areas.

The two most widely used psychiatric diagnostic tools are the mental status exam and the DSM-IV. We briefly mention the mental status exam here and we'll cover it in greater detail in a subsequent lecture. In addition, in this course you'll be asked to incorporate much of the mental status exam in weekly interview sessions.

The DSM-IV contains the accepted diagnostic criteria and nomenclature for all recognized psychiatric disorders. It is very useful to have standard definitions of psychiatric illnesses for clinical and for research purposes. The DSM-IV relies on a *descriptive* approach. The typical symptoms and features of each disorder are used to distinguish one from another; the etiology (cause) of the disorder is generally not part of the diagnostic scheme.

Another feature of the DSM-IV is that it categorizes disorders into groups that share some basic features in common. We will cover all of the major umbrella categories in this class, as noted in the class schedule (i.e., mood, psychotic, anxiety, somatoform, cognitive, substance use, and eating disorders).

The prevalence of psychiatric disorders is highly dependent on the setting in question; for example, certain illnesses are much more commonly seen in outpatient medical practices than in the general population.

Rank order of frequency of diagnosis, by setting

General population (by community survey)	**Outpatient medical office**	**Inpatient General Medicine Service**	**Inpatient Psychiatric Service**
Substance abuse	Anxiety Disorders	Mood Disorders	Mood Disorders
Anxiety Disorders	Mood Disorders	Adjustment Disorders	Schizophrenia
Mood Disorders	Substance Abuse	Substance Abuse	Substance Abuse
Schizophrenia	Somatoform Disorders	Delirium	Cognitive Disorders

The DSM-IV also incorporates a system for coding disorders on a "multiaxial" system. This system is rarely used outside of psychiatry, but is important to know because it devolves from the *biopsychosocial model* of disease. The biopsychosocial model is applicable to all illnesses (not just psychiatric ones) and states that disease always takes place in the context of three major, interconnected systems of human life. The anatomic, structural and molecular basis of the disease make up the *biological* system; psychodynamic factors, personality, and motivation make up the *psychological* system; and cultural, environmental, and familial factors make up the *social* system. The multiaxial coding method is meant to include all three elements of the biopsychosocial model into account when formulating a patient's problem.

There are five axes, all of which should be included for each patient (if you use this system):

Axis I:	Clinical psychiatric disorders
Axis II:	Personality disorders and mental retardation
Axis III:	General medical disorders
Axis IV:	Psychosocial and environmental problems
Axis V:	Global assessment of functioning (GAF – rated from 1-100)

Psychiatric Training

Psychiatry Residency is three years after a one-year internship. The internship must consist of 2-4 months of neurology and 4-6 months of internal medicine. During residency, required experiences include at least 9 months of inpatient psychiatry, 1 year of outpatient psychiatry, and rotations in consulting to the medical and surgical services, community psychiatry, and child psychiatry. The US graduates only about 900 board eligible psychiatrist/year now. Solo practice tends to be more feasible in psychiatry than most specialties. Psychiatrists can be generalists or can work in many different subspecialties, including child psychiatry, forensic psychiatry, substance abuse, geriatric psychiatry, and psychopharmacology.

Psychiatry at Stanford

The major Psychiatry rotations at Stanford that you may encounter are:

Stanford Hospital:

G2 Inpatient open unit

- Mixed medical/psychiatric problems
- Geriatric psychiatry

H2 Inpatient locked unit

- Schizophrenia, psychotic disorders, suicidality

Consultation/Liaison (C/L) Psychiatric problems of hospitalized patients

- On other wards, such as surgery or medicine

Palo Alto VA:

2B1 Acute psychiatry, locked unit

- Schizophrenia, psychotic disorders, suicidality

2B2 Less acute psychiatry, locked unit

- Mood disorders, PTSD, personality disorders

At most psychiatry wards, you should expect a high incidence of substance abuse (alcohol, methamphetamine, heroin, crack, cocaine, marijuana, nicotine). At the VA especially, you may also encounter a high incidence of PTSD.

A note on "locked wards"
***Not all patients in the locked wards are detained involuntarily; however, below is a short description of the basis of psychiatric hold:

Involuntary detainment (on the basis of a psychiatric disorder)

- Danger to self (suicidal, self-mutilative)
- Danger to others (intent to do harm or evidence to indicate that patient may be an imminent danger to others)
- Grave disability (inability to provide for food, clothing, and shelter because of a mental illness)

"Psychiatric Hold" -- 3 days *for observation only* (5150). No treatment is allowed without the consent of the patient unless it is a life-threatening emergency. A 5150 can be employed for any of the 3 reasons mentioned above for involuntary detainment.

A hearing must take place within 72 hours to determine if the hold can be extended for **up to 14 days (5250)**. The patient is entitled to representation by a patient advocate or attorney. The patient may be temporarily conserved by the state if cause to uphold the 5250 as gravely disabled is upheld and patient's condition is chronic. Only two consecutive 5250's (each with a hearing every 14 days) are allowed for danger to self or others. At that time a potentially violent patient can undergo a formal court hearing to determine if they can be held for up to 180 days as a danger to others. There are few recourses for holding the imminently suicidal patient after two consecutive 5250's.

Mental Status Exam

The Mental Status Exam

Charles DeBattista, M.D.

David L. Smith, M.D.

The diagnosis of psychiatric disorders is primarily based on a careful history in combination with a Mental Status Exam (MSE). The MSE is the tool used by psychiatrists to elicit and describe abnormalities of emotion, behavior, communication, and perception; it is the corollary in psychiatry to the physical exam in general medicine. Being able to use and to describe the MSE is a skill that you will need to learn and to practice during this course and beyond. It allows you to describe what a patient is like using a standardized format and vocabulary.

The Mental Status Exam has ten sections. Some sections are purely observational - the relevant data are obtained just by watching and listening to the patient - while other sections can be described only if the clinician actively asks appropriate questions.

Outline of the MSE

1.	Appearance	**6.**	Thought Process
2.	Behavior	**7.**	Thought Content
3.	Speech	**8.**	Perceptions
4.	Mood	**9.**	Insight/Judgment
5.	Affect	**10.**	Cognition

Appearance

This is an observational category and generally is a description of the patient's physical characteristics:

Sex
Age (apparent age and chronological age)
Race or ethnic background
Position (sitting down, standing up, lying on the floor)
Posture (stiff or stooped/hunched over)
Dress (casual, sloppy, disheveled, formal, seductive)
Grooming (well groomed, messy, makeup, body odor)
Attentiveness (are they distractible, can they pay attention
Facial expression (crying, smiling, grimacing, in pain)
Other features (bandages, handicaps, scars, etc)

When a clinician comments on an aspect of appearance, it is not necessarily abnormal, just remarkable. Psychiatric illnesses are really patterns, or sets of remarkable features that coalesce into a diagnosis, so you should comment on anything you find unusual.

For instance, a patient in the ER wore a tie with fire engines that had actual flashing red lights (he was manic). A female bipolar patient might wear too much makeup when manic, and then not wear any when depressed.

Behavior

Whereas appearance is like a snapshot in a single moment, behavior is more like a video over a period of time. Behavior is also an observational category.

Elements of Behavior
Eye contact
Manner and Attitude
Kinetics

Eye contact is an important element of behavior. Natural eye contact is mostly direct, with occasional glances away. Staring directly at you the whole interview, or never meeting your eyes, is remarkable.

Manner and Attitude refer to how someone is dealing with the task of the interview at hand, how they're relating to you. Examples of abnormalities include hostility and seductiveness, neither of which is appropriate.

Kinetics refers to body movements and gesticulations. Some people can be hyperkinetic, or *psychomotor agitated.* This refers to excessive movements that appear to be related to a psychological state. *Psychomotor retardation* is the opposite state, where a patient - for example, someone suffering from depression -- moves very slowly, is inwardly turned and doesn't look up, doesn't gesture, etc.

Speech

In this section the way the patient uses speech is examined.

Elements of Speech
Rate
Rhythm
Volume
Amount
Articulation
Spontaneity

You should pay attention to patients' voices while you are talking to them. How fast are they talking? Is the rate rapid or slow? Do they have *pressured speech,* which is when the stream of the patient's speech is difficult to interrupt?

Rhythm refers to the melody or musicality of speech. Most people have an up-and-down intonation or melody in their speech. Occasionally, people have monotonous speech without much variety or intonation.

You also want to comment on volume (loudness or softness) and the amount (a lot or very little) of speech. If it seems unusual to you, it probably is and is worth remarking upon in the MSE.

Articulation refers to how words are pronounced.
Spontaneity refers to the natural flow of speech without having to prepare one's words beforehand.

Mood

Mood is the internal experience of an emotional state.

Mood can only be assessed by asking the patient a question such as: "How do you feel?" You can quote the patient in the MSE. For example, "I feel lousy," or "I feel terrific!" The person's internal report is the definitive response. *You cannot guess mood; you have to ask directly.*

Affect

Affect is the external representation of an emotional state. It is not necessarily the same as the mood, and can be quite different from, or even incongruous with mood.

Elements of Affect
Range
Stability
Intensity
Appropriateness/Congruity

Consider the following metaphor: patients are pianists with an emotional piano at their disposal. The way they play the piano (their affect) reflects how they express their internal emotional state. One element of affect is its range. In an average psychiatric interview a patient will have many opportunities to express his or her emotions. An average person would be expected to demonstrate a full range: laughter at jokes, sadness when sad topics are brought up, similarly for anger, anxiety. A patient with a *restricted range* may be angry for the whole hour, unwaveringly; here, only one aspect of the emotional range is shown. In the terms of the piano metaphor, range

refers to the diversity of keys that patients use; are they single-theme players, or are they using all the possible keys available to them?

Stability refers to how they shift across the keyboard. Normal would be organized progression from one theme to another, each developed to a reasonable extent before the transition. Abnormal would be *lability:* going from one part to another too quickly, with diminished emotional coherence. A *fixed* affect is when the same emotional note is played over and over again.

Intensity, using our metaphor, refers to how strongly the patient pushes the keys. Affect can be overly intense. For example, if a patient is extremely angry over a 25-cent increase in laundry prices, for instance or another situation not normally thought of as very severely upsetting. *Blunted* intensity is also possible; for example, a patient who says, "Yeah, my mom died yesterday, it was pretty sad, I went to the funeral." If the emotional notes are so bland that they can't be distinguished, the patient is said to have a flat affect. Flat affect reflects a near robotic absence of emotional expression.

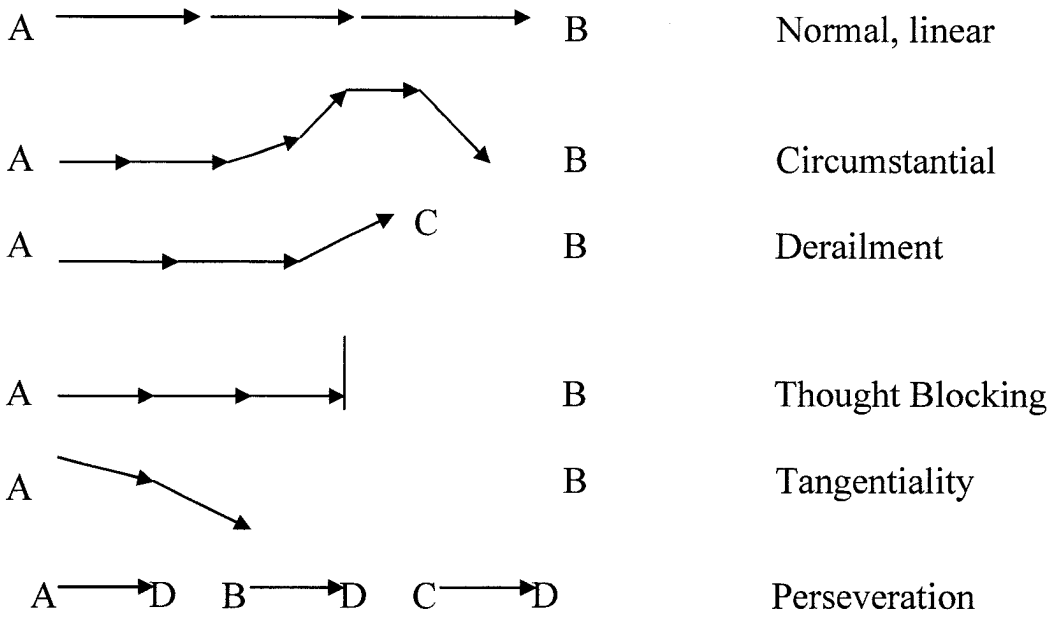
The fourth dimension of affect is the appropriateness or congruity of affect to the underlying mood. For example, a patient may talk about something sad but laugh while discussing it. This is very common, which is why you **must ask about mood and not assume based on affect.**

Thought Process

Thought Process refers to how patients form and convey their ideas to you.

A *formal thought disorder* is when there is a significant abnormality of the way thoughts are formed. A normal process should be *linear* and *logical;* for example, if you ask a patient a question they should be able to express themselves from point A to point B in a straight line, with each idea along the way logically connected to the ones before. A significant deviation from this line is potentially abnormal.

Thought Processes - linearity



Circumstantial (circumferential): The thought process is remarkable for excessive, irrelevant material, but point B is reached eventually.

Derailment: An abrupt transition from the initial train of thought to another topic, not logically linked together.

Thought Blocking: This occurs when a patient starts speaking and then just abruptly stops. He or she doesn't recognize the stop until you ask them to continue.

Tangentiality: The train of thought goes off in a different direction and never comes to point B.

Perseveration: When a patient repeals a word or idea over and over again even when it is clearly not applicable to the current question. For example: "How old are you?" "62..." "What year is it?" "62." "Are you married?" "62," etc.

Logicality refers to how well each idea is connected to the next in sequence. When patients have this type of thought disorder gaps may appear in their train of thought and you will have trouble following how they are moving from one idea to the next.

Flight of Ideas is when thoughts are shifted in a quick and abrupt way but there are understandable (if inappropriate) links between topics based on themes, plays on words, or even external stimuli.

Looseness of Associations is when gaps are present to such an extent that it becomes difficult to understand what the patient is vying to convey. In more extreme forms, this looseness is called word salad (an incomprehensible string of words or phrases) or incoherence (garbled speech where even individual words are often lost).

Neologisms occur when a patient invents a word that is not in dictionary and has special, idiosyncratic meaning to them. For example, a patient used the word "doctorb" to refer to nice doctors.

Clanging occurs when a rhyme or sound of a word triggers the train of thought or ideas. For example, "I'm so mad I'm glad I'm bad, watch out, Dad."

Thought Content

The following areas of thought content often need to be asked directly to the patient.

Elements of Thought Content
Suicidal ideation
Homicidal ideation
Obsessions
Ruminations
Paranoid ideation
Delusions

The various aspects of thought content will be covered in the relevant chapters dealing with disorders of thought content. One essential point: *a patient will not become suicidal because you ask them about it, and you cannot help a suicidal patient unless you know they are suicidal.*

Perceptions

Abnormalities of perception can be in any of the five senses.

Illusions are misperceptions of real stimuli. For example, a patient hears her mom's voice in a real train whistle.

Hallucinations are sensory perceptions in the absence of actual external stimuli. Examples hearing voices in an empty room, seeing bugs crawling on the ceiling, etc.

Insight/judgment

Insight describes to what extent the patient understands that they have an illness or problem.

Judgment is a reflection of the patient's ability to make decisions that are in his or her best interest.

Cognition

Cognition - intellectual functioning - has many facets. Each of these areas is discussed in detail in the Cognitive Disorders chapter.

Elements of Cognition
Attention/Concentration
Orientation
Memory
Language
Visuospatial skills
Executive Functioning

Cognition (intellectual functioning) has many facets.

Attention is the ability to attend to a specific task. Attention is tested on the MSE in several ways. Digit span, the ability to repeat a seven-digit number, requires both attention and immediate memory. Serial sevens, subtracting 7 serially from 100, also is a test of attention.

Orientation refers to the ability of the patient to know who and where they are. Orientation is evaluated by asking the patient their name, the date, day of the week, where the interview is taking place, where they live, etc.

Memory may be broken down into immediate, recent and remote. Immediate recall is tested having the patient repeat a number of set words. Recent memory is evaluated by having the patient remember 3 words at 1 and 5 minutes. Remote memory is evaluated by asking the patient to recall distant events that they should know based on their educational level such as the last 5 presidents.

Visuospatial skills are often evaluated by having the patient repeat a drawing of intersecting pentagrams or other geometric figures.

Executive Functioning refers to the cognitive ability to plan, organize, and sequence data.

Each of these areas is discussed in more detail in the Cognitive Disorders chapter, and a frequently used cognitive test called the Mini-Mental Status Exam (MMSE) will be introduced then.

Summary of the Mental Status Exam

1. Appearance
- -Sex, Age, Race/Ethnicity
- -Position, Posture, Dress, Grooming, Attentiveness, Facial expression, Other features

2. Behavior
- -Eye Contact, Manner and Attitude, Kinetics (psychomotor agitation/retardation)

3. Speech
- -Rate – slow, rapid, pressured
- -Rhythm – monotonous (always up or always down)
- -Volume, Amount, Articulation, Spontaneity

4. Mood – internal experience of emotion – *must ask directly*

5. Affect – external representation of mood – *observed*
- -Range – normal, restricted
- -Stability – labile, fixed
- -Intensity – blunted, flat, overly intense
- -Appropriateness/Congruity

6. Thought Process
- -Linearity – circumstantial, derailing, thought blocking, tangential, perseverative
- -Logicality – flight of ideas, looseness of association (word salad, incoherence), neologism, clanging (rhyming)

7. Thought Content
- -Suicidal/homicidal ideation, obsessions, ruminations, paranoid ideation, delusions

8. Perceptions (auditory, visual, touch, taste, smell)
- -Illusions – misperception of real stimuli
- -Hallucinations – sensory perceptions in ABSENCE of external stimuli

9. Insight/Judgment
- -Insight – extent to which patient understands that they have an illness or problem
- -Judgment – patient's ability to make decisions in his/her best interest

10. Cognition

Tested on the Mini-Mental Status Exam (MMSE)*:
- -Attention/Concentration (serial subtraction)
- -Orientation (in time, place, person)
- -Memory (learning, immediate recall, delayed recall)
- -Language (naming, repetition, comprehension, reading, writing)
- -Visuospatial Skills (apraxia detected by inability to copy a diagram)

Cognitive functions not tested on the Mini-Mental Status Exam:
- -Agnosia – inability to recognize objects, people, sounds, smells, etc
- -Executive functioning (drive, programming, synthesis, etc.)
- -Motor skills

* For details of the Mini-Mental Status Exam, refer to the cognitive disorders chapter.

Psychotic Disorders

David L. Smith, M.D.

The Case of the Man Hunted by Aliens

A 23 year-old unmarried man is brought into the ER by the police. His neighbors had called the police because he had been acting strange and shouting at them if they came close to his apartment. He had duct-taped all his windows over and covered his ceiling and floor with aluminum foil. When the police picked him up he yelled "Don't take me out! They'll know I'm here!"

In the ER his vital signs were BP: 135/85; HR: 85; T: 99.0. His PE was remarkable for being somewhat thin but otherwise there were no significant findings on general physical. Neurological exam was nonfocal. His labs showed normal electrolytes, hepatic function tests, complete blood count, and a negative toxicology screen.

His MSE was remarkable for the following. A/B: disheveled male with unkempt hair and dirty clothes. He appeared suspicious of the interviewer. He was psychomotor agitated and paced during the interview. His eye contact was poor and he frequently stared at the ceiling. Speech: fluent, loud. Mood: "Upset" Affect: blunted TP: The patient was frequently tangential and occasionally had thought blocking. His answers to questions were often illogical with looseness of associations. Perceptions: The patient reported hearing voices telling him that he was going to be abducted by aliens and tortured. TC: He denied suicidal or homicidal thoughts. He did report that he felt the television and radio were being used to spy on him by aliens and that they intended to capture him. He believed several of his neighbors were actually aliens in disguise. He reported that he had a transmitter implanted in his tooth by the aliens while he was sleeping so the aliens could track him down. Insight: poor. Judgment: poor. Cognition: level of consciousness, memory, orientation, and language skills were all intact. The patient had difficulty with abstraction. When asked how a car and a bus are similar he replied, "they flatten the asphalt."

What is the most likely diagnosis? What is the differential diagnosis?

What symptoms would be classified as positive symptoms? Negative symptoms? Thought disorder?

What would an MRI of the brain show?

Does this illness have a genetic basis?

What is the most appropriate class of medications to treat this illness?

Schizophrenia

Introduction

Our current understanding of schizophrenia is that it is a constellation of symptoms that clearly has a biological, brain-based origin. Schizophrenia is a common illness (the estimated prevalence is approximately **1%** of the adult population worldwide) that is associated with significant suffering and distress for affected individuals and their families as well as with important public health and economic consequences for society in general. The etiology (or more accurately, etiologies) of schizophrenia is not known, but the factors that predispose an individual to develop schizophrenia are becoming better understood.

Psychosis and the Three Symptom Clusters of Schizophrenia

Psychosis, defined narrowly, is a failure in reality testing, an inability to tell the real from the unreal. Psychosis is a symptom, not a diagnosis; when a patient presents with psychosis the next step is to determine why. Schizophrenia is the prototypical psychotic disorder, although many other conditions can present with psychotic symptoms (see the last section of this chapter).

The definition of psychosis is sometimes broadened to include any severe deficit in the processing of perception, emotion, and/or communication. This larger definition is useful for understanding the varied symptoms of schizophrenia, which can be organized into three categories.

Positive Symptoms	Negative Symptoms	Thought Disorder
Hallucinations (auditory most common) Delusions (ideas of reference, persecution, grandeur) Disorganized behavior	Flat affect Alogia Social withdrawal Avolition	Loosening of associations Thought alienation Poverty of thought

Positive symptoms are behaviors or experiences that people with schizophrenia have that are not considered part of normal functioning. Examples are *hallucinations, delusions,* and *disorganized behavior* (for example, shouting in public or grossly inappropriate attire).

*N.B. A word on Hallucinations vs. Illusions vs. Delusions

Hallucinations are completely imagined (hearing voices that are not there)… Vs.

Illusions are real events that are misinterpreted (hearing wind blow through trees that makes sounds which are mistaken for voices)… Vs.

Delusions are fixed, false beliefs ("these trees are absolutely out to get me")

Negative symptoms are essentially the result of a lack or of a reduction of normally expressed behaviors. Examples of negative symptoms include flattened affect, *alogia* (absent or diminished spontaneous speech), and *avolition* (absent or diminished motivation).

The third main symptom cluster includes **thought disorder** symptoms. A formal thought disorder (i.e., a disorder in the way thoughts are formed) is any consistently expressed abnormality in the linearity or logicality of thoughts and ideas.

History and Phenomenology of Schizophrenia

The way schizophrenia has been understood has changed over time. The main reason for this is that schizophrenia is a disorder that is diagnosed phenomenologically (by the presence of an arbitrarily determined set of symptoms) rather than by a specific test. **Emil Kraeplin** was the first researcher to develop a consistent set of symptoms for diagnosing schizophrenia, although the term he used for the disorder was *dementia praecox*, which means "early dementia." Kraeplin thought that the most salient aspect of the illness was the *negative symptom cluster*, as well as what he thought was an inevitable deterioration in his patients' clinical status over time. He thought that positive symptoms were only accessory components of the illness.

Eugin Bleuler actually coined the term schizophrenia, which means "split mind" (but has nothing to do with so-called multiple personality disorder). Perhaps truer to his intended meaning would be "fractured mind." Bleuler thought that the presence of a *thought disorder* was the most important aspect of diagnosing schizophrenia, although he also noted the importance of negative symptoms in his "*Four A's of Schizophrenia:*" loose *associations* (i.e., thought disorder), *autism* (in this context, an inappropriately egocentric worldview), *ambivalence* (an inability to make decisions), and inappropriate *affect*.

Kurt Schneider thought these two earlier ways of diagnosing schizophrenia were too subjective and looked for specific symptoms that he thought were pathognomonic of schizophrenia (pathognomonic symptoms

are those that definitively indicate one and only one diagnosis). He focused mostly on the positive symptoms of schizophrenia, and his list of symptoms is called *Schneider's First Rank Symptoms* (examples of 1st rank symptoms are the hallucination of two or more voices arguing with each other or of a voice commenting on the individual's actions).

The **DSM-IV** incorporates some of the concepts of all three of the above researchers. There are five symptom criteria for DSM-IV diagnosed schizophrenia: *hallucinations, delusions* (see chart below for examples), *grossly disorganized behavior, any negative symptom,* and *"disorganized speech"* (another way of saying thought disorder). Note that all three symptom clusters are represented (although three of the five are positive symptoms). *To be diagnosed with schizophrenia an individual must have two or more of the above five symptoms for at least six months with a corresponding decrease in social and/or occupational functioning.* Also note that there is no mention of deterioration as a definitive feature of schizophrenia.

There are other clinical aspects of the illness that are often seen in people with schizophrenia, but are not part of the diagnostic criteria. *Neurological soft signs* (which, as opposed to *hard signs,* can not be localized to a dysfunction in a specific, discrete brain region) are evidenced by poor coordination, difficulty with fine motor skills, and primitive reflexes (blink reflex, grasp reflex, palmomental reflex, etc.). *Neuropsychological deficits* are often seen in schizophrenic patients, particularly in problems with executive functioning, concept formation, abstraction, and the ability to change sets and rules.

DSM-IV Criteria for Schizophrenia

2 or more of the following:

- Hallucinations
- Delusions
- Grossly disorganized behavior
- Negative symptoms
- Disorganized speech (i.e., a formal thought disorder)

Duration greater than six months

Corresponding decrease in social and/or occupational functioning

Types of Delusions

Persecutory	Delusional belief that one is in danger, being followed or monitored, harassed, or conspired against
Grandiose	Delusional belief of special power, talent, abilities, or identity
Delusions of control	Delusional belief that one's own actions, behavior, or feelings are not under personal control or won doing, but are imposed by an outside force
Guilty	Delusional belief of responsibility for tragedy or disaster to which there is no personal connection and/or of deserving retribution or punishment
Jealous	With little or no evidence, the person believes his or her sexual partner is unfaithful
Mind reading	Delusional belief that people can read one's mind or know one's thoughts
Delusions of reference	Delusional belief that ordinary, insignificant comments, objects, or events refer to or have special meaning for the patient.
Erotomanic	Delusional belief that one is loved, perhaps secretly, by another person (usually a person of high status).
Somatic	Delusional belief that one's body is diseased or changed (i.e., a man who is convinced he is pregnant)
Delusions of replacement	Delusional belief that someone important to the patient has been replaced by a double. Also called *Capgras delusions*
Nihilistic	Delusional belief that the person, a part of the person's body, or the world does not exist
Thought broadcasting	Delusional belief that as thoughts occur they escape from the person's head and can be heard by others
Thought insertion	Delusional belief that thoughts are not one's own but have been placed there by someone of something else
Thought withdrawal	Delusional belief that one's thoughts have been removed or taken away by someone or something from the outside

Epidemiology

Important epidemiological data regarding schizophrenia are summarized in the table below.

Variable	Finding
General Prevalence	1-1.5%
Suicide Rate	50% attempt suicide at some point 10-15% die because of suicide
Substance Abuse	75% use cigarettes 30-50% use alcohol 5-10% use cocaine
Gender and Age	Male = female in general prevalence But males have earlier onset Onset: Males (age 17-27), Females (17-37) And females have better outcome
Socioeconomic Status (SES)	↑ prevalence in lower SES groups Estimated 30-60% homeless have schizophrenia
Cultural Variables	↑ rate in recent immigrants Prognosis more benign in less developed countries
Season of birth	↑ risk seen in people born in Winter, early Spring (reversed in the Southern Hemisphere!)
Pregnancy and Birth Complications (PBCs)	Any PBC is assoc. with ↑ risk of schizophrenia, especially maternal flu in the 2nd trimester or malnutrition during the 1st trimester

The increased prevalence of schizophrenia in lower SES groups has two potential explanations. The first is called the *downward drift hypothesis* that states that people with schizophrenia are more likely to have poor occupational functioning and thus to end up in a lower SES as they age. The *social causation hypothesis* argues that living in a lower SES is associated

with a higher degree of stress and is thus more likely to lead to development of schizophrenia in at-risk individuals. Both processes likely contribute to this epidemiological finding.

Etiology

Schizophrenia almost certainly has many etiologies. In other words, there are many factors that can contribute to someone's risk of developing the characteristic set of behavioral and perceptual abnormalities seen in the disorder, and these factors are not necessarily the same for every schizophrenic patient.

Until recently, the major debate about the etiology of schizophrenia concerned which model better explains its development and course, a **neurodevelopmental model** or a **neurodegenerative model.** The neurobiological data discussed above (in particular, the evidence of abnormal brain structure at the onset of the illness and the absence of gliosis) are much more consistent with a neurodevelopmental model (early derangements in the way the brain develops which later lead to the expression of the symptoms) than with the neurodegenerative model (a normally developed brain begins to deteriorate at some point after development is completed). A neurodevelopmental model also better explains the increased risk of developing schizophrenia in infants whose mothers experienced Pregnancy and Birth Complications (PBCs) and for those born in Winter (presumably because of an increased risk of exposure to certain viruses or other infections during critical periods of brain development).

Neurobiology - The field of neuroimaging has contributed much to our understanding that schizophrenia is a disease of the brain. The classic imaging findings were with computerized tomography (CT or CAT scan) technology, which uses x-rays. CT studies have repeatedly demonstrated increased ventricular size and decreased cortical volume in schizophrenics at the onset of the illness. The data are inconsistent regarding whether there is further, progressive volume loss after diagnosis.

In recent years there have been intriguing **neuropathological findings** associated with schizophrenia. There has never been evidence of *gliosis* (scarring), which is almost always noted in conditions that lead to necrotic brain cell loss (as in Parkinson's disease or Huntington's disease). This suggests that the abnormal cortical volume loss in schizophrenia occurred early in development, when non-necrotic mechanisms for reduction in cell volume are normal. The two leading explanations posit an abnormal increase in *apoptosis* (programmed cell death) or in *synaptic pruning* (which leads to loss of dendrites but not cell bodies).

On the level of **pathophysiology**, the classic explanation for schizophrenia is the **Dopamine Hypothesis**, which essentially explains the illness as the result of too much dopamine activity in the brain. Evidence for this model is that neurochemical agents that block dopamine activity reduce psychosis, agents which increase dopamine activity can be psychotogenic, and that increased levels of homovanillic acid (HVA; a metabolite of dopamine) are correlated with increased levels of psychosis in schizophrenics. This explanation is now considered overly simplistic, although excessive dopamine neurotransmission is probably a factor in the

illness. Abnormalities in other neurotransmitter systems have also been implicated in schizophrenia, particularly the serotonin (5-HT) system (LSD, which is 5-HT_2 agonist can induce psychosis) and the glutamate and GABA systems (which are the major excitatory and inhibitory amino acid neurotransmitter systems in the brain, respectively).

Neuroanatomical models of schizophrenia concentrate on presumed abnormalities in the functioning of certain brain regions; the two areas that have received the most attention are the *limbic system* and the *frontal lobes.* The limbic system is important in the regulation of emotions and perceptions (as well as having memory functions) and it is hypothesized that the positive symptoms of schizophrenia may be related to dysfunction in this area. The frontal lobes are responsible for much of the brain's *executive functioning* skills like planning, organizing, motivation, and drive; it is thought that the negative symptoms of schizophrenia may be related to dysfunction in these areas. Two dopamine tracts in the brain, the mesolimbic tract, and the mesocortical tract, project dopaminergic neurons to the limbic system and the frontal lobes, respectively. These dopamine tracts may play significant roles in the pathogenesis of the positives symptoms (from the mesolimbic tract) and the negative symptoms (from the mesocortical tract) in schizophrenia.

Historically there has been a lot of attention paid to the **psychosocial** aspects of schizophrenia. One essential point: *faulty parenting does not cause schizophrenia.* A concept previously put forth was the idea of *double bind*, where it was thought children who were exposed to conflicting parental messages regarding behavior, attitudes, and feelings responded by

withdrawing into psychosis in order to escape the supposed unsolvable emotional confusion. There is no evidence that double bind situations play any role in the development of schizophrenia. Another, more useful, concept is that of *expressed emotion (EE)*, which is when intense criticism, hostility and intrusiveness are overexpressed in a family system, not to be confused with normal expression of emotion. Although high EE environments do not cause schizophrenia, they have been associated with higher relapse rates for schizophrenic patients.

Genetics

Schizophrenia has a strong genetic component, although there are probably many genes that confer a vulnerability to developing the disorder. The evidence for a genetic basis is seen most clearly in the concordance rates between family members (i.e., what is the chance of a certain degree relative of a schizophrenic patient also having schizophrenia). These data are summarized below:

Relative with Schizophrenia	**% Concordance**
(General population)	(1-1.5%)
Non-twin sibling	8%
One parent	12%
Dizygotic twin	12%
Both parents	40%
Monozygotic twin	47%*

*There is no change in concordance for MZ twins raised together or apart

Course and Prognosis

The typical age of onset for schizophrenia is in late adolescence (late teens, early 20s), and males have an earlier average age of onset than females. The onset of the disorder – also termed the "first psychotic break" – may or may not be associated with a stressor in the patient's life. There is typically a period of gradual recovery after the first break, but without a return to baseline functioning. As schizophrenic patients age positive symptoms typically become less pronounced while negative symptoms often worsen.

About 20-30% of schizophrenics can be termed "high-functioning" in that they live independently and have jobs and families. Over 50% of patients however have courses marked by repeated hospitalizations, exacerbations of symptoms, and episodes of suicidality.

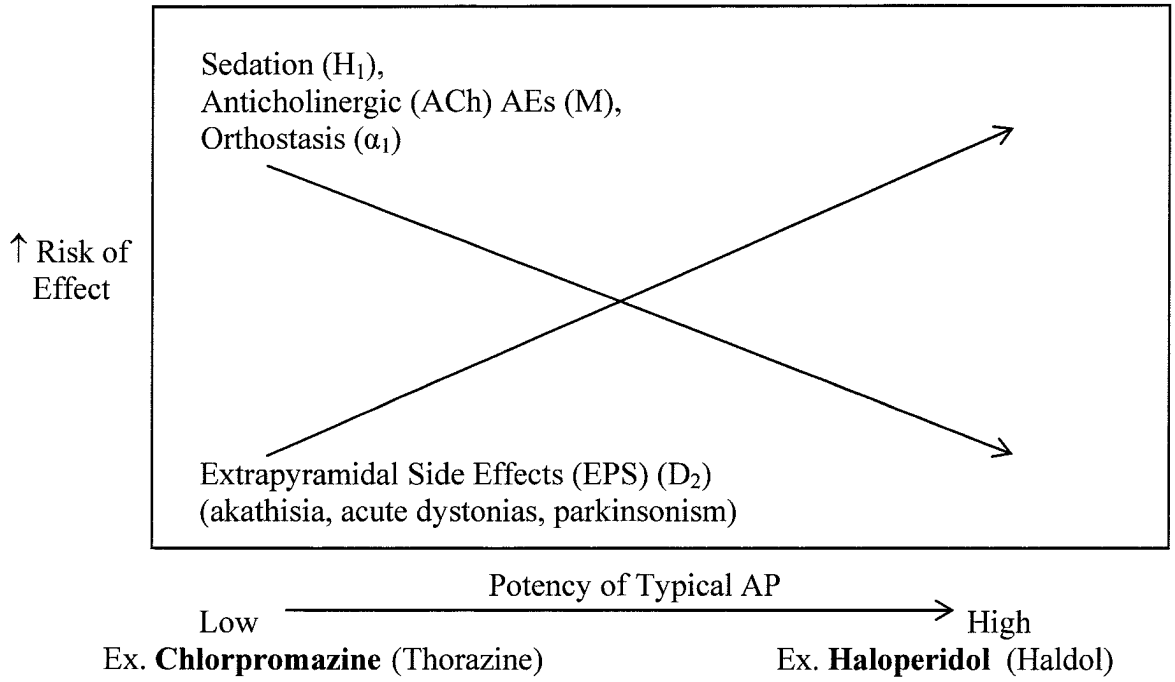
Treatment

The cornerstone of treatment for schizophrenia is the use of **antipsychotic medications**. In this section we will expand on the basic information covered in the review of psychopharmacology lecture. *You will be expected to remember only the medication names in bold.*

Typical Antipsychotics

Typical antipsychotics (APs) all have high degrees of dopamine D_2 antagonism at therapeutic doses, which is thought to be responsible for their

clinical benefit. They are all generally more effective for the positive symptoms of schizophrenia than for the negative symptoms. The most important differentiation between typical APs is their relative potency, which is correlated with different side effect profiles on two opposing gradients:



In other words, low potency typical APs are more sedating, cause more ACh adverse effects (AEs), are more likely to cause orthostasis, and are less likely to cause EPS than high potency APs. The reason for these two, inverted gradients has to do with receptor profiles (see next chart).

Atypical Antipsychotics

Atypical APs are considered atypical because at clinical doses they are all less likely to cause EPS than are typical APs, even low potency typicals. **Clozapine** is the prototypical agent in this class. It does not cause EPS at all.

It also has several other properties that distinguish it from typicals: efficacy in treatment-refractory schizophrenia, a lower risk of tardive dyskinesia, no association with an increase in prolactin release, and demonstrated ability to reduce negative symptoms. The other atypical APs have not been shown (and probably do not) share all these properties with clozapine; they also do carry some degree of risk for EPS (but less than typicals).

It is not known why atypical APs are different in the risk for EPS than typical APs, but one leading theory holds that their modulation of the serotonin system (specifically 5-HT_2 antagonism) helps reduce EPS. The receptor profiles of the different types of APs are summarized below. Clozapine carries the risk of several idiosyncratic AEs that are not well explained by its known receptor profile, as seen in the chart.

Antipsychotics: Receptor Profiles

	D_2	5-HT_2		H_1	M (ACh)	α_1	Idiosyncratic
Typicals:							
Low potency (i.e., **chlorpromazine**)	++			++	++	++	
High potency (i.e., **haloperidol**)	++			+	+	+	
Atypicals:							
clozapine (Clozaril)	+/-	+		++	++	++	Agranulocytosis, Sialorrhea, ↑ Seizure risk
olanzapine (Zyprexa)	+	+		+	+	+	
risperidone (Risperdal)	+	+				+	
quetiapine (Seroquel)	+	+		+		+	
ziprasidone (Geodon)	+	+				+	

Action on these receptors leads to "nuisance" AEs as covered in the psychopharmacology lecture. The more strongly an agent interacts with these receptors, the more likely it is to cause the corresponding AE.

<u>D_2 antagonism</u> can lead to the development of many side effects, which are most easily remembered by reviewing the major dopaminergic tracts in the brain. Remember: atypicals are much less likely to cause these D_2 related side effects than typicals (possibly because of the <u>5-HT_2 antagonism</u>).

D_2 related side effects

DA Tract	*Related side effects (of antagonism)*
Tuberoinfundibular	Elevated prolactin, temperature dysregulation
Nigrostriatal	Acute EPS (Extrapyramidal side effects): akathisia (motor restlessness) acute dystonias (muscle spasms) parkinsonism (akinesia, tremor, and rigidity) Other: Tardive dyskinesia (abnormal, involuntary muscle movements – typically in the mouth/face) NMS (Neuroleptic malignant syndrome)
Mesolimbic	Reduced psychosis (this is good)

Most clinicians consider atypical APs the treatments of first choice for schizophrenia because of their superior side effect profile (however, they are much more expensive than most typical APs). One other potential disadvantage of atypicals is that they currently only are available in oral forms, while two of the typical APs – **haloperidol decanoate** and fluphenazine decanoate -- are available in *a long-acting intramuscular depot formulation.* For patients who will not or do not want to take pills these formulations can be quite helpful.

Reference list of antipsychotics

		Typicals	**Atypicals**
Potency:			
(High)	100:1	pimozide	**clozapine** (Clozaril)
	50:1	**haloperidol** (Haldol)	**olanzapine** (Zyprexa)
		fluphenazine	**risperidone** (Risperdal)
	20:1	thiothixene	quetiapine (Seroquel)
		trifluoperazine	ziprasidone (Geodon)
	10:1	perphenazine	aripriprazole (Abilify)
		loxapine	
		molindone	
		piperacetazine	
	2:1	mesoridazine	
(Low)	1:1	**chlorpromazine** (Thorazine)	
		thioridazine	
		chlorprothixene	

Psychosocial Treatment

Non-medication treatment modalities are also essential to the comprehensive care of schizophrenic patients. In institutional systems (like county mental health systems or the VA system) *case management* is a way of ensuring that patients have their psychiatric, social work, and medical needs integrated. *Psychotherapy* (both group and individual) can be helpful for many patients. The style of therapy is generally supportive (vs. insight-oriented) and focuses on problem-solving current life problems, improving reality testing, and education regarding the illness. *Family therapy* also can be useful to increase the family members' understanding of the illness and to explain issues like the value of reducing "expressed emotion."

Other Psychotic Disorders

Several other disorders are associated with psychosis. One way to organize them is based on whether or not they have a prominent affective component.

Psychotic disorders without significant affective component:

Schizophreniform disorder: The symptom criteria are the same as for schizophrenia, but the duration of illness must be 1-6 months. The prognosis is generally positive.

Brief psychotic disorder: The symptom criteria are the same as for schizophrenia, but the duration of illness must be less than one month. This diagnosis also has a good prognosis.

Psychosis secondary to medical condition or substance: The most common etiology in this category is stimulant intoxication. There are many other conditions that can cause psychosis, like temporal lobe epilepsy, frontal lobe tumors, endocrine abnormalities, and many others.

Delusional Disorder: The hallmark of this illness is the presence of one or more non-bizarre delusions that lead to a decrease in the patient's occupational or social functioning. Unlike schizophrenia, there are no other symptoms of psychosis (hallucinations, thought disorder, negative symptoms).

Psychotic disorders with significant affective component:

Bipolar disorder: The criteria for bipolar disorder are reviewed in the mood disorders chapter. Remember that psychosis can be present in either the manic or depressed phase of the illness. "Post-partum psychosis" is most often related to an exacerbation of underlying bipolar disorder; *this is a psychiatric emergency, as the infant can be in imminent danger.*

Schizoaffective disorder: A patient with this disorder meets the symptom criteria for schizophrenia, but also has had at least one concurrent major mood episode (major depressive or manic); delusions and/or hallucinations must be present for at least two weeks when there is an absence of major mood symptoms.

Disorders that do not lead to true psychosis, but can be confusing to remember:

Schizoid personality disorder: This personality disorder is associated with a pervasive detachment from social relationships and a restricted range of emotional expression. The symptoms are similar in quality, but not as

severe, as the *negative symptoms* of schizophrenia. There are no delusions or hallucinations present (*positive symptoms)*.

Schizotypal personality disorder: This personality disorder is associated with a pervasive pattern of social and interpersonal deficits and by cognitive distortions and eccentricities of behavior. The symptoms are similar in quality, but not as severe, as the *positive symptoms* of schizophrenia. Although patients often have odd beliefs and superstitions, they do not have actual delusions or hallucinations.

Return to the case

The most appropriate diagnosis is schizophrenia (assuming the symptoms can be traced back for at least six months). The differential diagnosis of first-onset psychosis is other psychiatric disorders associated with psychosis (mania, sever bipolar or unipolar depression, schizoaffective disorder, delusional disorder), psychosis secondary to substance use (particularly stimulant intoxication), and psychosis secondary to medical/neurological diseases (such as brain tumors, seizure disorders, endocrinopathies, certain CNS infections, etc., etc.). Positive symptoms in this case are auditory hallucinations, persecutory and bizarre delusions, delusions of reference, delusions of replacement, disorganized behavior (i.e., taping his room, staring at the ceiling). The most prominent negative symptom is blunted affect. Thought disorder symptoms are loose associations, tangentiality, and thought blocking.

An MRI would most likely be normal. Although there is a statistically significant difference in brain volume for populations of schizophrenics compared to populations of controls, in any individual the changes are usually too subtle to be noted radiologically. Schizophrenia does have a genetic basis, but is not a single gene disorder. The most appropriate psychopharmacologic intervention is an antipsychotic medication (current practice is to use atypical antipsychotics first-line).

Mood Disorders I & II

Mood Disorders

Charles DeBattista, D.M.H., M.D.

The Case of the Surgeon Turned Apostle

A patient with a history of a presumed depressive episode treated effectively with a Selective Serotonin Reuptake Inhibitor (SSRI) now presents 2 years later after being picked up by the police. The patient is 28 yo W male surgery resident found wandering naked through campus with bible in hand espousing religious ideas and suggesting that he was the 13th apostle. He was noted to have bizarre posturing and speech was largely unintelligible.

He had been working late on a paper for the past 2 weeks and had presented it 5 days PTA (prior to admission) in Australia. Since returning home, he hand been somewhat jet lagged and was only sleeping about 4 hours/night. His colleagues noted that he had been particularly excited about this particular paper which was well received but nothing else unusual was noted. Pt was off on the weekend and was supposed to report to the OR at 7:00 am Monday . The attending surgeon attempted to contact the patient at home unsuccessfully. The locked psychiatric service called the surgery service at 1 p.m. to inform them of the patient's hospitalization.

Past Psychiatric Hx: One episode of depression treated with SSRI for 6 months then discontinued approximately 20 months earlier.
Medical Hx: Hypertension (HTN)
Medications: unspecified antihypertensive
Habits: Occasional EtOH. No Recreational Drugs
Family: Paternal Uncle hospitalized for unclear psychiatric condition
Paternal uncle completed suicide
EtOH in other paternal uncle, maternal aunt, brother
Vitals: BP =145/90, rr=16, Pulse=96
Labs; Chem 20, CBC (Complete blood count), TFTs (Thyroid Function Tests), UA (urine analysis) all WNL (Within Normal Limits)
Tox screen positive for THC (cannabis)

MSE: at admission
Lethargic, appears disoriented
Speech: largely mute
Kinetics: appears catatonic with periods of agitation. Also waxy flexibility
Thought process: unable to evaluate. By hx tangential with loose associations
Thought content: by hx delusions of grandeur, religious delusions. Appears to be responding to internal stimulation.
Judgment: Poor; Insight: Poor
Cognition: unable to evaluate
SI (Suicidal ideation), HI (homicidal ideation); no evidence

What is the most likely diagnosis?

What is the significance of the previous depressive episode? How common is it for a depressive episode to precede a psychotic episode?

How may antihypertensives contribute to a mood disorder?

What is the evidence for genetic transmission of mood disorders?

What is the significance of a positive THC drug screen in this patient?

What psychiatric conditions may present with catatonia?

Are there other lab tests that should be done now?

What are the acute treatment options? What about long term management?

General

Mood disorders have been described by healers and physicians since antiquity. Among the earliest descriptions of what we now called major depression are from Egyptian scholars 3500 years ago. The Greek physicians borrowed from the Egyptians, and Arab scholars from the Greeks. Hippocrates described major depression in some detail; he termed it Melancholia and believed it was a biological illness caused by an oversecretion of black bile. If a patient had too much black bile the patient became melancholic: withdrawn, isolative, pessimistic, oversleeping. Areteous of Cappadocia described "melancholy" as the beginning and a part of mania, connecting periods of depression with agitation and excitement. Galen linked depression to anxiety and aggression. In a classic western text, The Anatomy of Melancholy, Burton (1621) described depression as not only a biologic illness but one that was genetically transmitted. He also associated depression with disturbances in the sleep cycle and recommended certain kinds of treatment including chastity and the avoidance of alcohol.

Mood disorders are very common in the general population, yet they are underdiagnosed. Mood disorders are debilitating and sometimes lethal illnesses that we must learn to identify as there are many effective, FDA-approved treatments for both Major Depression and Bipolar Disorder.

Understanding depression requires defining a few themes central to the discussion of psychological status. Emotion is a mental state that tends to be characterized by a particular feeling. It is transient and has physiologic sequelae. Anger, sadness and anxiety are emotions, and they can change

from moment to moment. Mood suggests a more enduring emotional state; the analogy is that emotion is to weather as mood is to climate. Mood must be elicited directly from the patient (ie. The patient describes his mood as "sad"). Affect is what you see, what you describe when you see the patient (The patient appears agitated and tearful). Temperament is a trait that is enduring; it may be evident from birth.

In describing people's temperaments, we typically speak of four general temperaments: dysthymia, euthymia, hyperthymia, and cyclothymia. **Dysthymia** is an enduring type of low mood, low self-esteem, and mild disturbances in sleep and appetite. It is often reported as being there "forever". The diagnosis of Dysthymic Disorder is made after at least two years of depressed mood for more days than not, with symptoms that fall short of a major depressive episode. That is, Dysthymic Disorder is characterized by feelings of helplessness that do not escalate to the point of despair or suicide. **Euthymia** refers to a normal mood, neither high nor low. **Hyperthymia**, a term not frequently used, describes a consistently elevated mood. Many successful, socially effective people may be hyperthymic. These people tend to tend to be persistently cheerful, energetic and motivated. In some cases, hyperthymia may be on the spectrum of manic depressive illness but it may also represent a normal and useful temperament. **Cyclothymia** is probably a variant of bipolar disorder, and is characterized by at least two years of numerous periods of 'highs' (that do not meet criteria for a manic episode) as well as numerous periods of depressive symptoms (that do not meet the criteria for a major depressive episode).

DSM-IV Diagnostic Criteria for Dysthymic Disorder

*The differentiation of dysthymic disorder from major depression can be difficult. Key features to dysthymia are a milder, moderate depressed mood, and a chronic (greater than 2 years) course.

1. Depressed mood for most of the day, for more days than not, for at least two years.
2. While depressed, there must be present 2 or more of the following: poor appetite or over-eating, insomnia or hypersomnia, low energy/fatigue, low self-esteem, poor concentration or difficulty making decisions, feelings of hopelessness.
3. During the two year period, the patient has never been without the symptoms in number 1 or 2 for more than 2 months at a time.
4. There has never been a Major Depressive Disorder, Manic Episode, Mixed Episode, Hypomanic Episode or Cyclothymic Disorder.
5. The symptoms must cause significant impairment or distress.

Major Depressive Disorder

DSM-IV Diagnostic Criteria for Major Depressive Disorder

Major Depressive Disorder is characterized by at least two weeks of either depressed mood (most of the day, nearly every day) or anhedonia, a markedly diminished interest or pleasure in daily activities, plus at least four additional symptoms:

-weight loss/gain

-insomnia

-psychomotor agitation or retardation

-fatigue or loss of energy

-feelings of worthlessness, excessive or inappropriate guilt

-diminished ability to concentrate

-recurrent thoughts of death

A helpful mnemonic to remember the symptoms of depression: SIGECAPS. Sleep (too much/little) Interest (decreased), Guilt, Energy (poor) Concentration (diminished) Appetite (high/low) Psychomotor (agitation/ retardation), Suicidal Thoughts.

Patients may describe apathy and irritability rather than a depressed mood: in fact, denial of depressed mood is common. The classic type of sleep disturbance in depression is an early morning awakening, where the patient can't get back to sleep. On the other hand, some patients will sleep twenty hours a day (hypersomnia). It is not unusual for patients to feel as if they are slowed down in a depressed state: speech will be slower, thoughts forming slower; they won't move or walk as fast. Fatigue or anergy can be mild or profound. Many depressed patients present with the primary complaint that they have no energy, "I had zest but now I don't..." Feelings of worthlessness and guilt can take on delusional proportions. A patient may come to believe that he or she is an evil person who is responsible for the sins of the world. There may be a diminished ability to think or concentrate: this is also a common reason to seek help: "I can't concentrate, can't read a book, follow an article, can't follow plot of movie." Often, depressed patients will show deficits in executive functioning including the ability to make decisions. Even simple decisions may become overwhelming. The

cognitive deficits of depression, along with fatigue, are the leading causes of disability in depressed patients.

Thoughts of death are quite common in major depression. Sometime, these thoughts are of a passive quality ("It would not be a bad thing if I never woke up again"). Other times, the patient may have active thought that they should end their lives. Often these active thought are associated with a depressive rationale ("My family would be better off without me. I am just a burden"). Depressed patient often feel bad that they've survived to see another day. Major depression is a *potentially lethal illness*: 15% percent of patients with hospitalized for a major depression will kill themselves in the course of their lifetimes. Since depression is not an uncommon problem, this adds up to many suicides every year. The implications for physicians are clear – depressed patients must be identified and those identified should be treated.

The Epidemiology of Major Depression

A gender difference in major depression has been noted in virtually all cultures examined. Women suffer form major depression at about twice the rate of men. The average lifetime prevalence of depression in the U.S. is 17%; 26% for women, and 12% for men. However, it is unclear whether this difference is related to other factors, such as increased help-seeking behavior by women for depressive symptoms, or by the tendency for men to cope with depressive symptoms through alcohol abuse. Physicians are at least as vulnerable to depression as general population and may be at greater risk. The mean age for a first episode is in the thirties. Demographic

differences show that rates tend to be higher in urban areas. No racial significance has been noted. However, it has been demonstrated that lower socio-economic status, recent immigration status, and being married (for women only; for men marriage is appears to be protective) are also independent risk factors for major depression.

In primary care settings, rates of depression may be high as 30%. Often, people present to doctors feeling bad and depression is an important reason they may not feel well. Depression is often underdiagnosed in primary care settings because patients rarely present with a complaint of being depressed. Rather, a depressed patient may present primarily with complaints of memory problems, energy loss, or sleep disturbance. Depression can be quite chronic. At least 20% of depressive episodes last more than 2 years and 80-90 % of patient who experience one depressive episode will experience multiple episodes. Depression can progress if untreated to a more chronic, recurrent, and refractory illness. In fact, depression ranks among the most common causes of absenteeism and vocational disability in the developed world.

Subtypes

There are five basic subtypes of depression: Psychotic Depression, Atypical Depression, Melancholic Depression, Seasonal Depression, and Postpartum Depression. Note that these last two, Seasonal and Postpartum depression are time-specific, occurring during certain time frames.

Psychotic Depression accounts for 5-25% of depressive episodes, especially in geriatric patients. In addition to meeting full criteria for a major depressive episode, these patients have psychotic symptoms such as nihilistic, somatic and paranoid delusions. In addition, they may also hear voices telling them they are a bad person or that they should be dead. Psychotic depression tends to be less responsive to standard antidepressant treatment alone and often requires an antipsychotic or ECT.

Atypical Depression refers to a type of depression that is characterized by the presence of mood reactivity. These "reactive" atypical depressives may have a transient improvement in mood when something good happens. However, they tend to quickly slip back into a more persistently low mood. In addition, patients with atypical depression may have carbohydrate cravings and gain substantial amounts of weight as opposed to the weight loss and anorexia that characterizes most severe depressive episodes. Rather than having insomnia, patient with atypical depression tend to sleep too much. They may be in bed and sleep 12 hours a day or more. Atypical depression may respond preferentially to MAOIs.

Melancholic Depression is characterized by a lack of mood reactivity; the depressed mood is pathologically and persistently low. Thus, the low mood is not brightened by positive events. In addition, melancholic depression tend to have more severe vegetative symptoms. This may include profound psychomotor retardation, anorexia that is severe that the patient may require a feeding tube, and a prominent diurnal variation in which patients tend to feel most horrible but symptoms might improve as the

evening approaches. Melancholic depression is thought to be more responsive to TCAs or SNRIs (venlafaxine) than to SSRIs.

Seasonal depression or seasonal affective disorder tends to be characterized by low mood as the days get shorter. As the spring comes on many patients recover; some may actually become manic. As one might anticipate, seasonal depression is more common in northern latitudes. One of the treatments is phototherapy with a lightbox. Since seasonal depression is often associated with bipolar disorder, mood stabilizers are also sometimes needed.

Postpartum Depression is a depression than occurs within a few months of delivery. It is not unusual to begin within days of the birth. The post-partum period is the single most vulnerable time in a woman's life for the development of a depressive episode. An estimated 50-85% of women have postpartum blues, which is characterized by crying spells, sadness, confusion, anxiety, and insomnia. Full-blown post partum depression is thought to occur in about 10% of new moms. These patients often fail to care for their baby or themselves and may have psychotic symptoms. Psychotic features in a post partum depression represent a medical emergency and are frequently associated with both suicide and infanticide.

Medical Conditions Associated with Depressive Symptoms

There are a number of medical conditions that present with the symptoms of depression. In order to effectively diagnose and treat patients,

one must be thorough in identifying any medical conditions that may be associated with depression:

Cushing's syndrome (hypercortisolemia), Hypo/hyperthyroidism, Hypo-parathyroidism, Diabetes Mellitus - hyperglycemia, frontal strokes, Vitamin B12 deficiency, Brain tumors, Parkinson's Disease, Viral Infection (Flu, HIV). (Note that all of these pathologies have been covered in HH&D - be vigilant in recognizing depression associated with these diseases!) Additionally, chronic disease of any type may be associated with depression.

Biological Factors in Major Depression

Although the exact etiology of depression is yet to be determined, multiple factors, including biological factors, are involved in the presentation of depression.

Genetic Vulnerability: having a first degree relative with depression, you have a four times higher risk than the general population; if you have a monozygotic twin with depression, concordance is as high as fifty percent. The more severe and recurrent the twin's depression the more likely the other twin will have a severe mood disorder. See Table 7-5

The Biogenic Amine Hypothesis states that depression is caused by deficiency of certain amines, like serotonin and norepinephrine. Most antidepressants do enhance central amines; if patients are given a drug like reserpine, which depletes monoamines (NE, dopamine, serotonin), those patients are more likely to get depressed. Virtually every available

antidepressant works on serotonin, norepinephrine, or dopamine, but there are other neurotransmitters we are just discovering, including peptide neurotransmitters. It has been difficult to demonstrate a monamine deficiency in most depressed patients. Thus, it is likely that monoamine antidepressant have some uniform downstream effect such as turning on the genes that transcribe neurotropic factors such BDNF.

Noradrenergic Theory of Depression

Data Supporting the Theory
1. Approximately 15% of patients treated for hypertension with reserpine develop depression.
2. Drugs that enhance noradrenergic (norepinephrine) functioning (stimulants) are often found to have some antidepressant properties.

Data Against the Theory
1. Drugs that interfere with noradrenergic transmission, or block catecholamine synthesis, do not regularly produce depression in most subjects.
2. Blockade of norepinephrine reuptake or MAO inhibition by antidepressant drugs occurs within hours or a few days after administration of the antidepressant, but the clinical effects do not usually appear until after 2 to 4 weeks of treatment.
3. Attempts to demonstrate abnormalities in norepinephrine activity by measuring plasma, urine, and CSF concentrations of norepinephrine and its major metabolite 3-methoxy-4-hydroxy-phenylglycol (MHPG) have produced conflicting data.

Data Supporting Serotonin Dysfunction in Major Depression

1. Decreased concentrations of brain serotonin and CSF 5-HIAA in many depressed patients.
2. Most antidepressant agents have been shown to increase the efficacy of central serotonin neurotransmission.
3.Reduction in both central and peripheral 5-HT reuptake sites has been found in depressed subjects.
4. Neuroendocrine challenges have demonstrated that the postsynaptic serotonin-mediated stimulation of prolactin is blunted in depressed patients.

Hypothalamic Pituitary Axis Abnormalities. Depression appears to be a chronic stress state. As such, major depression is associated with a relative hypercortisolemia, an increase in CRF levels, and a tendency to result in false positives on the dexamethasone suppression test. (Dexamethasone, a corticosteroid, should suppress hypercortisolemia caused by a secondary upregulation of ACTH, e.g. pituitary tumor/Cushing's Disease, since cortiocsteroids inhibit ACTH release via negative feedback; if the patient's hypercortisolemia is primary/from hypersecretion of cortisol by the adrenal cortex, Dexamethasone cannot suppress the hypercortisolemia.) Also note that exogenous glucocorticoid therapy (e.g., prednisone) may be associated with depression and hypomania.

There a numerous glucocorticoid receptors throughout the cortex. The hippocampus is particularly rich in glucocorticoid receptors appears to be sensitive to chronic stress states. Anti-glucocorticoid drugs may be effective in the treatment of some severe forms of depression.

Neuroendocrine Abnormalities in Depression

1. Hyperactivity of the hypothalamic-pituitary axis, resulting in elevated plasma cortisol levels, nonsuppression of cortisol following dexamethosone (DST), hypersecretion of corticotropin-releasing factor.
2. Blunting (decreased physiological responsiveness) of the normally expected increase in plasma concentrations of thyroid stimulating hormone (TSH) following infusion of thyrotropin-releasing hormone (TRH).
3. Blunting of the normally expected increase in plasma growth hormone in response to alpha-2 adrenergic receptor agonist stimulation.
4. Blunting of serotonin-mediated increase in plasma prolactin.

The Neurotrophic Deficiency Hypothesis of Depression. An increasingly convincing hypothesison the etiology of depression is that depression and other chronic stress states may result from chronic suppression of neurotrophic factors such as Brain Derived Neurotrophic Factor (BDNF). Increased levels of cortisol may mediate this suppression of BDNF. As cortisol levels rise, BDNF levels drop. The suppression of BDNF results in impaired neurogenesis, neuroplasticity, and synaptic resilience in the hippocampus. As a result, the hippocampus has been noted to undergo atrophy that varies as a function of the duration of an untreated depressive episode. The longer the untreated depression, the greater the atrophy. All studied antidepressants appear to enhance BDNF and facilitate neurogenesis. ECT may more dramatically enhance neurogenesis than antidepressants.

Functional Imaging studies show that dorsolateral prefrontal cortex is less active in depressed patients than it is in those who are not depressed. Conversely, the ventromedial cortex tends to be overactive in functional imaging studies. Resolution of the depression is associated with normalizing activity in the dorsolateral and ventromedial prefrontal cortex.

Sleep Abnormalities: shorter/decreased REM latency is probably most consistent sleep finding.

Sleep Abnormalities in Depression

Non-REM Changes in Depression
1. Prolonged sleep latency (the period of time between going to bed and falling asleep).
2. Shortened REM latency (the period of time from the onset of sleep to the first REM period)
3. Increased wakefulness
4. Decreased arousal threshold
5. Early morning awakening
6. Reduced stages 3 and 4 sleep

REM Sleep Changes in Depression
1. Shorter REM latency (i.e. 30-60 minutes instead of the normal average of 60-90 minutes)
2. A redistribution of REM sleep to the first half of the night rather than the second half.

Psychotherapy for Depression

Psychotherapy is as effective for the treatment of mild to moderate depression as is pharmacotherapy. It is not likely that psychotherapy alone is an effective option for more severe forms of depression such as psychotic depression or those depressive episodes characterized by catatonia or severe cachexia. The combination of psychotherapy with medications appears to be more effective for the treatment of depression than either one modality alone. Biological and psychotherapeutic treatments may approach a depression from different angles. Psychotherapy has been demonstrated to first effect areas such as the dorsolateral prefrontal cortex which tends to be hypoactive in depression. In addition, psychotherapy is known to help a

patient deal with stress and normalizing the stress response lowers cortisol, which in turn, raises BDNF levels.

Psychodynamic school: (Freudian) Freud thought depression is caused by object loss, especially the loss of a love object. If a patient had ambivalence toward the love object, for example a lost mother, the patient was thought to be more likely to get depressed. Therapists today do think that loss is an important psychological variable in depression and loss is almost invariably stressful. Other theoreticians such as Menninger conceived of depression as an anger inwards: a self-aggression. Suicide, then, was a murderous impulse turned inwards. Interpersonal theories attribute depression to a social dysequilibrium (problems in a patient's social world). To treat depression, interpersonal therapists will focus on social dysequilibrium. Thus, this form of psychotherapy tends emphasize problems in relationships and a resolution of those problems. Cognitive School: CBT (Cognitive Behavior Therapy) focuses on the idea that cognitive problems are important in the etiology of depression. Depressed patients tend to have numerous cognitive distortions. For example, in response to a perceived failure, a patient may conclude that they are worthless and that they are a burden to their families. In CBT, a therapist will teach a patient to challenge these negative and overreaching conclusions. In addition, behavioral strategies aimed at mobilizing the patient may be prescribed. CBT is the best studied form of psychotherapy for the treatment of depression.

Antidepressants

Antidepressants are among the most commonly prescribed medications in western medicine and represent a 13 billion dollar/year industry. Antidepressants have a number of things in common; they are successful in 60-80% of patients, and no antidepressant has been shown to be more effective than another. There are at least two dozen on the market; for the purposes of this class, you only need to remember three or four. All of them work on *monoamines*. Furthermore, *none work immediately*. Rather, they take 4-8 weeks to show maximum efficacy. Examine the prototypes for each class of antidepressant, and understand the side effects and toxic interactions for each.

MAOIs: Monoamine Oxidase Inhibitors

Prototype: Phenelzine (Nardil)

Rarely used now but sometimes used in treatment resistant depression and Parkinson's Disease. They block the breakdown of monoamines by inhibiting monoamine oxidase (which metabolizes monoamines). Serious drug interactions including **serotonin syndrome** (delirium, diaphoresis (profuse sweating) tremulous, myoclonic jerking, coma, seizure, and often death). In combination with serotonergic drugs and hypertensive crisis (associated with dietary tyramine which acts as a false pressor). Common side effects include hypotension, sexual side effects and weight gain.

SSRIs: Selective Serotonin Reuptake Inhibitors.

Prototype: Fluoxetine (Prozac).

The most commonly used antidepressant agents in the world. Their popularity rests on ease of use, nonlethality in overdose , and wide spectrum of activity. They are also FDA-approved in the treatment of panic disorder, social phobia, OCD, PTSD, Premenstrual Dysphoria and other conditions. They work by selectively blocking the reuptake of serotonin in the synaptic cleft via inhibition of the serotonin transporter. The most common side effects are GI upset, CNS activation, diaphoresis, and sexual side effects.

TCAs; Tricyclic Antidepresants

Prototype: Imipramine (Tofranil)

The TCAs are less commonly used now because they are dangerous in OD. (About 1500 mg of impramine is sufficient to induce a lethal arrhythmia.) They block serotonin and norepinephrine reuptake. They are also quite antimuscarinic (dry mouth, constipation, urinary retention etc), antihistaminic (H-1 and H-2 antagonists) and are associated with sedation and weight gain. In addition they block alpha-2 receptors and cause postural hypotension. Commonly used for chronic pain, enuresis, and migraine prophylaxis.

Atypicals:

Prototype: Bupropion (Wellbutrin)

Monoamine effects used to be thought to be dopamine reuptake inhibition, more likely noradrenergic agonist. Common AEs (Adverse Effects) include CNS activation, insomnia, tremor, and weight loss. There is a small risk of seizures with the drug.

SNRI: Selective serotonin and norepinephrine reuptake inhibitor

Prototype: Venlafaxine (Effexor)

Indicated in GAD, MDD and soon in neuropathic pain. AEs much like the SSRIs except may also induce hypertension

When would antidepressants be used instead of psychotherapy or combination therapy?

Evidence shows that the optimal therapy for depression combines antidepressants with psychotherapy. However, there are a number of cases in which antidepressants are used instead of psychotherapy. The first such instance is when a patient has had a previously good response to an antidepressant. The same drug may be suggested during a subsequent severe episode. Also, if a patient can't engage with a therapist, or if a patient is psychotic, catatonic or cachectic (thin and wasted from not eating) as a function of depression, psychotherapy alone would not be recommended, and antidepressants are indicated. Suicidality normally suggests a level of severity that may warrant pharmacological intervention. Psychotherapy is not inexpensive, and may not be covered by insurance plans; therefore, not everybody has the resources for this treatment option. Furthermore, some California counties have few mental health professionals so availability may be another frustration. On the other hand, psychotherapy may be used independent of pharmacological agents for milder depression or due to the patient's preference to avoid medication.

When and why use electroconvulsive therapy (ECT)?

ECT is still commonly used in the US (500,000 procedures per year). It is often used in the treatment of resistant depression or mania that is unresponsive to other strategies. ECT has a high efficacy, and is considered to be **single most effective treatment** for severe depression. In addition there are people who respond to ECT that respond to nothing else. However, besides the stigma still associated with it, ECT has the universal side effect of anterograde/ retrograde memory loss, which is usually a short term loss around the period of the seizure. Rarely, deficits can last for months afterward, and very rarely can last permanently. ECT has a low mortality rate. Treatment is usually given in six to twelve sessions, with treatments every other day over the course of two to four weeks. Despite more than 60 years of investigation, the mechanism of ECT is not completely understood. Induced seizures are an ancient treatment of mood disorders; they seem to increase monoamines, affects the HPA axis. Yet no clear mechanism has been found. A variety of mechanisms probably contribute to the therapy's efficacy.

Other Brain Stimulation Treatments of Depression

While there is an unfortunate history of the use of ablative brain procedures in the treatment of psychiatric disorders, in recent years, a number of reversible treatments that involve the direct or indirect electrical stimulation of the brain have been introduced. In 2005, vagus-nerve stimulation (VNS) was FDA approved for the treatment of resistant depression. This treatment uses a pacemaker type device implanted under

the left clavicle to send electrical impulses, via wire, to the left vagus nerve. The vagus nerve, which is at least 80% afferent on the left, collects sensory information from throughout the body and relay it to various parts of the cortex including those areas that regulate mood. Thus, stimulating the nerve with a unidirectional electrical impulse is an indirect way of stimulating various parts of the brain. This treatment works slowly and about 30% of highly resistant depressed patients respond after about 1 year. This is about the same rate of response seen with VNS in resistant epilepsy for which it is also approved. Another type of brain stimulation therapy is transcranial magnetic stimulation (TMS) which may be approved in 2007. This treatment uses a focused electromagnetic field to stimulate the dorsolateral prefrontal cortex. It is an outpatient procedure and does not require anesthesia. TMS also is not associated with cognitive problems but appears less effective than ECT. Finally, deep brain stimulation (DBS) which is approved for the treatment of some movement disorders, such as the tremor associated Parkinson's disease, has shown some promise in the treatment of severe, refractory depression that has been unresponsive to all other treatments. In this neurosurgical procedure, bilateral leads are implanted directly into the anterior cingulated or parts of the limbic circuit to directly stimulate tracts that are known to be important in mood. The leads are connected to a small implanted generator. Early studies have suggested that there may be an immediate, dramatic, and persistent antidepressant effect of DBS in some patients. Multi-center studies are currently underway.

Bipolar Disorder

Bipolar disorder is characterized by both periods of major depression alternating with mania or hypomania.

A Manic Episode is a distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least one week. In many ways, mania is the mirror image of depression. Instead of being down, patients are up to the point of uncontrolled euphoria an excitement with inflated self-esteem or grandiosity, often delusional. Manic patients tend to display a decreased need for sleep (for example, going for days without sleep.) They exhibit a pressure to keep talking: they need to get words out. Racing thoughts are common, with a fluidity of thoughts and insights that a patient may find initially wonderful. Increased distractibility, increased activity or agitation, excessive involvement in pleasurable activities are common. Chronic mania can last for years, but this is very rare. The average manic episode lasts for days to weeks, not more; patients burn out easily.

A helpful mnemonic for the main features of a Manic Episode is "PooR JuDGES." P=Pressured speech, R=Racing thoughts, J=poor Judgment, D=Distractibility, G=Grandiosity, E=Energy (increased), S=Sleep (decreased need).

DSM-IV Diagnostic Criteria for Bipolar Disorder, Manic Episode

A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least one week, of sufficient severity to cause impairment in social or occupational functioning.

During the period of mood disturbance, three (or more) of the following symptoms must have persisted (four if the mood is only irritable) and have been present to a significant degree:

- inflated self-esteem or grandiosity
- decreased need for sleep (e.g. feeling rested after 3 hr sleep)
- more talkative than usual, pressure to keep talking
- flight of ideas, subjective experience of racing thoughts
- distractibility
- increase in goal-directed activity or psychomotor agitation
- excessive involvement in pleasurable (but risky) activities (e.g. buying sprees, sexual indiscretions, foolish investments).

Additionally, there must be no evidence of a physical or substance-induced etiology or the presence of another major mental disorder to account for the patient's symptoms.

Hypomania

Mania is on a spectrum with hypomania. Hypomania is defined as an elevated/irritable mood that is a grade below mania in terms of its severity. Hypomanic episodes are distinct periods of persistently elevated, expansive or irritable mood similar to manic episodes, with the difference being shorter duration, lasting at least four days, instead of one week. Also, they do not result in hospitalization or psychosis.

DSM-IV Diagnostic Criteria for Hypomanic Episodes

* The criteria are identical to the criteria for Manic Episodes. The only difference is the time course - in Hypomania, symptoms must be present for at least 4 days.

Stages of Mania

Mania can progress from a relatively mild form to an absolutely dysphoric, psychotic, out-of-control form. It often starts with euphoria, progressing to dysphoria (a mood of general dissatisfaction, restlessness, depression, and anxiety) to frank panic. The early stages of mania tend to be characterized by elevated mood, feeling on top of the world, etc., but as the episode progresses, patients may start feeling worse. Soon, they're out of control, in two thirds of cases progressing to psychosis, including hallucinations and delusions.

Mania Associated Symptoms

Thought disorder is common in mania: flight of ideas, tangentiality are common occurrences during manic episodes (more common in mania than in schizophrenia). Psychosis, including delusions and hallucinations, occur in up to 90% of manic episodes. Manic patients may also be hypersexual. There is also a high incidence of violence and sociopathy in manic states. The association between creative genius and bipolar illness has been noted for decades. Some of our most acclaimed artists, writers, and

scientists are known to have suffered from bipolar disorder. Hypomania has also been associated with political, religious, and military leadership.

Etiology of Mania

Mania may be precipitated by a disruption of sleep cycles, or sleep deprivation in susceptible patients. Bipolar patients who travel crossing time zones should make sure to sleep! There is clearer genetic data for bipolar disorder than any psychiatric condition. Concordance rates in monozygotic twins raised apart since infancy shows a concordance rate as high as 80%. Psychosocial stresses can precipitate manic episodes, but are neither sufficient nor necessary to bring on a manic episode. There is a seasonal variation to manic episodes and they appear to be somewhat more likely to occur in the spring or summer as the photoperiod of the day increases. Any effective antidepressant can precipitate mania in a susceptible patient.

Bipolar I Disorder

Bipolar I Disorder is characterized by one or more Manic or Mixed Episodes, usually accompanied by Major Depressive episodes. Patients typically have at least one episode of mania, and in greater than 90% percent of cases they also have episodes of depression. Thus, bipolar reflects both a depressive and manic pole.

Bipolar II Disorder

Bipolar II Disorder (female > male) is characterized by one or more Major Depressive Episodes, accompanied by at least one Hypomanic Episode. There must not have been a manic episode or a Mixed episode. Symptoms must cause clinically significant distress or impairment in social, occupation, or other important areas of function, and not be better accounted for by Schizoaffective disorder or a psychotic or delusional disorder.

Mixed Episode

In a Mixed Episode, criteria are met for both a manic episode and for a major depressive episode nearly every day during at least a one-week period. Mixed states are simultaneous, with mania co-incident with depression, (e.g. depressed mood but with grandiose, racing thoughts). The mood disturbance is sufficiently severe to cause marked impairment in occupational functions in or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

Bipolar Epidemiology

Bipolar illness has an *early onset* (teens to 30), with most cases diagnosed by age 22 in this country. The first hospitalization occurs, on average, at age 26, with the first episode in men more likely to be mania, while in women it is more likely to be depression. A bipolar patient may first present with depressive episode and then subsequently have a manic break in

their 40's. Suicide also not uncommon, 20-30% will make suicide attempts, while 10% to 15% complete the attempt. There is a mean of 4 cycles per decade. Females are more likely to rapidly cycle.

As time goes on, there is a tendency for the disease to worsen. Initially, bipolar disorders will present as minor depression triggered by significant stressors. However, later in the course of the disorder, the depressions are more severe and triggered by smaller, less significant stressors (or, symptoms can even arise spontaneously).

The lifetime prevalence of Bipolar Disorder is 1%, the same as for Schizophrenia. Among Bipolar Patients: only 5% have only manic episodes. 95% will have both manic and depressive episodes.

Treatment of Bipolar Illness

Medications

Bipolar Disorder requires chronic treatment in order to control symptoms. Pharmacotherapy is almost always employed in the treatment of Bipolar Disorder. In general, the drugs are much better at preventing /treating the mania than the depression. Most bipolar medications are remarkable for their tendency to cause weight gain; however, lamictal and newer atypical antipsychotics are not associated with weight gain. The weight gain is the number one reason patients stop taking the medication, while the second most common reason to stop taking medication is the markedly diminished cognitive abilities that frequently occur. In addition,

lithium and some anticonvulsants are teratogenic, and must be avoided in first trimester of pregnancy.

In the past, the most commonly prescribed medication to treat Bipolar Disorder was **Lithium Carbonate**. Lithium Carbonate (Lithobid, Eskalith) is just an ion. Not all patients respond to it but up 70% of patients with bipolar type 1 respond. Lithium decreases the risk of subsequent episodes by a factor of two; it also reduces the number of depressive cycles. It may take seven days or longer for lithium to treat acute mania. The adverse effects of lithium treatment include tremor, gastric upset, and cognitive problems. In toxicity, lithium can be lethal, therefore it is important to monitor blood levels. Generally, chronic treatment is required. In recent years, atypical antipsychotics, and anticonvulsants such as valproate, carbamazepine, and lamotrigine are being increasingly prescribed for bipolar disorder.

Anticonvulsants

Depakote (Valproic Acid) – can cause hepatic irritation, sedation, tremor, weight gain, dyspepsia, rare but fatal hepatotoxicity.

Tegretol (carbamazepine) – can cause ataxia, sedation, weight gain and a catastrophic aplastic anemia, which is lethal

Neurontin (gabapentin) – not established in the treatment of bipolar disorder but well tolerated

Lamictal (lamotrigine) – FDA approved for the maintenance treatment of bipolar in 2003. Associated with Stevens-Johnson syndrome (very rare) and 10% rate of other rash.

Antipsychotics

With the exception of clozapine, all atypical antipsychotics are now used for the acute treatment of mania; olanzapine and aripiprazole are also approved for the maintenance of bipolar disorder (preventing relapse of depression or mania). Symbyax (a combination of fluoxetine and olanzapine) is the first drug approved for use in the acute treatment of bipolar depression. Quetiapine, another atypical antipsychotic is now approved as a monotherapy for the treatment of bipolar depression. While lithium and anticonvulsants may take a week or longer to treat mania, antipsychotics may begin to help in the ER. Thus, antipsychotics are the most agent to be first prescribed in the hospital.

Benzodiazepines (Valium, Librium, Ativan) extremely useful as adjunctive agents to help with sleep, anxiety, and agitation.

Psychotherapy

In Bipolar Illness, psychotherapy focuses on education, stress management, and family and group therapy. The illness is often extremely disruptive to families, since family members with the illness may be losing money, having affairs, drinking, and so on. There is no evidence that psychotherapy alone can prevent manic episodes or depressive cycles; thus, it is not an option of monotherapy. Rather, it is generally used in combination with medications to treat Bipolar Disorder.

Mood Disorders: Major Clinical Features

Diagnosis

	Major depressive episode	Minor depressive episode	Mania	Hypomania
Major Depressive Disorder	X			
Dysthymic Disorder		X		
Bipolar I Disorder	X		X	X
Bipolar II Disorder	X			X
Cyclothymic Disorder		X		X

Overview of Psychiatric Treatments

Overview of Psychiatric Treatments

David L. Smith, M.D.

Introduction

There are many different treatments available for psychiatric illnesses. A very useful way of thinking about and organizing the types of treatments is the **biopsychosocial model**. The biopsychosocial model describes all illnesses (not just psychiatric ones) as influenced by and connected to three interwoven systems of human life. The anatomic, structural and molecular basis of a disease make up the *biological* system; psychodynamic factors, personality, and motivation make up the *psychological* system; and cultural, environmental, and familial factors make up the *social* system. Psychiatric treatments can be primarily biological, psychological, or social; for most patients, a comprehensive treatment plan considers all three areas.

Biological Treatments in Psychiatry

Introduction to Psychopharmacology

Psychiatric medications can be broadly classified into four groups -- **antidepressants, mood stabilizers, antipsychotics,** and **anxiolytics** -- and this is the primary indication for the medications in each group. However, most medications are also useful for a variety of other symptoms. In the charts below are listed the generic names of the medications, along with the trade names (in parentheses) of the more commonly used and/or well-known agents. *You are expected to remember only the names of medications or medication classes in bold.*

At an introductory level the most important feature of a psychotropic medication to understand is its neurotransmitter **receptor profile**; this helps explain its mechanism of action, its spectrum of effect, and its side effect profile. To start, certain receptors are important to know because they are largely related to unwanted (side) effects of many different psychotropics:

Receptors important to know because of *side effects*

Receptor	*Side Effects Associated with Antagonism (receptor blockade)*
H_1 (Histamine)	Sedation, weight gain
M (Acetylcholine, Muscarinic type - especially important in the parasympathetic nervous system)	Anticholinergic side effects: confusion, blurry vision, dry mouth, tachycardia, constipation, urinary retention, dry skin
α_1 (Alpha adrenergic)	Orthostasis (dizziness on change of position)
Na-channel (quinidine-like effect)	↑ Cardiac conduction time (arrhythmias)

Antidepressants

The history of the group of medications called antidepressants (ADs) goes back to the early 1950s when an antituberculosis medication with MAOI properties, iproniazid, was found incidentally to help with depressed mood. Since that time many AD agents have been developed, but it was not until the introduction of Prozac (generic – fluoxetine) in 1988 that there was widespread availability of relatively safe, tolerable, and easy to use ADs.

All antidepressants (ADs) share four important properties:

1. They all increase the activity of one or more of the three monoamine neurotransmitter systems: serotonin (5-HT), norepinephrine (NE), and dopamine (DA).
2. They all have equal efficacy in major depression (approximately 60-70%).
3. They all have equal speed of onset (about 2-4 weeks).
4. None are addictive or lead to drug tolerance/dependence.

Most common indication for an AD is in the treatment of **major depressive disorder**, especially when the symptoms are moderate or severe. All ADs also appear useful for the treatment of dysthymia. Many **anxiety disorders** respond to ADs; however, the different classes of ADs vary significantly in their anti-anxiety profiles.

Antidepressants: Spectrum of Indications

	Depression	**Dysthymia**	**Panic**	**GAD**	**OCD**	**Social Phobia**
SSRIs **Prozac**, et al	√	√	√	√?	√	√
SNRIs						
venlafaxine (Effexor)	√	√	√?	√	√?	√?
Duloxetine (Cymbalta)	√	√				
Other						
bupropion (Wellbutrin)	√	√				
nefazodone (Serzone)	√	√				
mirtazapine (Remeron)	√	√				
MAOIs	√	√				√
TCAs entire class [**clomipramine**]	√ √	√ √	√ √		√	

SSRI = Selective serotonin reuptake inhibitor; SNRI = Serotonin and norepinephrine reuptake inhibitor; MAOI = Monoamine oxidase inhibitor; TCA = Tricyclic antidepressant; GAD = Generalized anxiety disorder; OCD = Obsessive-compulsive disorder

The ADs can be separated into different classes based on their receptor profiles. Within each class ADs are generally quite similar in terms of spectrum of effect and potential side effects:

Antidepressants: Receptor Profiles

Therapeutic Affects					**Side Effects (Associated with Antagonism)**				
	5HT	NE	DA		H_1	M (ACh)	$\propto_1$	Na-channel (quinidine-like)	Idiosyncratic
SSRIs: **Fluoxetine** (Prozac), **sertraline**, **paroxetine**, **citalopram**, fluvoxamine	√								
TCAs: imipramine amitriptyline desipramine nortriptyline [**clomipramine**]	√	√ √			+ +	+ +	+ +	+ +	
MAOIs: phenelzine tranylcypromine	√	√	√				+		Serotonin Syndrome; Hypertensive Crisis
Newer:									
venlafaxine (Effexor)	√	√							
bupropion (Wellbutrin)		√?	√						↑Seizure risk
nefazodone (Serzone)	√*						+		
mirtazapine (Remeron)	√*	√			+				

(√) denotes Therapeutic Action

Increased monoamine activity (although good for depression) can also cause side effects:

	Side Effects related to ↑ activity
5-HT	GI upset, **sexual dysfunction***, CNS activation (insomnia, jitteriness, anxiety, restlessness, headache), sedation
NE	GI upset, CNS activation
DA	GI upset, CNS activation

*nefazodone and mirtazapine interact with 5-HT hetero- and autoreceptors ($5\text{-}HT_2$ receptors to be precise); they are much less likely to cause sexual dysfunction than SSRIs.

Receptor blockade can also cause side effects:

Receptor	*Side Effects Associated with Antagonism(receptor blockade)*
H_1 (Histamine)	Sedation, weight gain
M (Acetylcholine, Muscarinic -important in the parasympathetic nervous system)	Anticholinergic side effects: confusion, blurry vision, dry mouth, tachycardia, constipation, urinary reten-tion, dry skin
$\propto_1$ (Alpha adrenergic)	Orthostasis
Na-channel (quinidine-like)	↑ Cardiac conduction time (arrhythmias)

Mood Stabilizers

Mood stabilizers (unlike antipsychotics, antidepressants, and benzodiazepines) are a very heterogeneous group of medications. Their receptor effects and mechanisms of action are complex and poorly understood. Clinicians must rely more heavily on straight memorization to know these medications well (although currently there are only three used widely).

Mood stabilizers are primarily indicated for **bipolar disorder**, particularly the manic phase of the illness. They are occasionally used for the treatment of agitation or irritability caused by other psychiatric disorders.

Most side effects of mood stabilizers tend to be dose-dependent; that is, the higher the dose, the worse the side effect load. Mood stabilizers also differ in their dangerousness in overdose, known as the *therapeutic index* (the lower the index the more dangerous).

Mood Stabilizers

	Side Effect Profile
Lithium	CNS: cognitive impairment, tremor GI: nausea, diarrhea Renal: polyuria, polydipsia (excessive drinking) Other: acne, hypothyroidism, weight gain, leukocytosis, teratogenicity **Therapeutic index: low**
valproic acid (Depakote)	CNS: sedation, tremor GI: nausea, diarrhea Hepatic: elevated liver enzymes, hepatotoxicity Other: hair loss, thrombocytopenia, weight gain, teratogenicity **Therapeutic index: high**
carbamazepine (Tegretol)	CNS: sedation, ataxia, diplopia, tremor GI: nausea, diarrhea Heme: leukopenia, bone marrow suppression Other: elevated liver enzymes, hyponatremia, skin reactions, teratogenicity **Therapeutic index: moderate**
Lamotrigine (Lamictal)	CNS: Sedative, cognitive deficit Others: Rash – Steven's Johnson Syndrome

Antipsychotics

Antipsychotic medications will be covered in detail in the psychotic disorders lecture. A brief overview will be given in this section.

The first antipsychotic (AP) medication (chlorpromazine – Thorazine) was discovered in 1952. It was actually used initially as an adjunctive medication to general anesthesia; it was only incidentally found to have antipsychotic properties.

The primary indication for this class of medications is for the treatment of **schizophrenia**. They are also useful for other primary and secondary psychotic disorders, for symptomatic treatment of delirium, and for emergency treatment of severe agitation.

There are two archaic terms for antipsychotics, whose use we discourage. The oldest term is "major tranquilizer" which was meant to distinguish them from the benzodiazepines, or "minor tranquilizers." However, many APs are not particularly sedating. The second term is "neuroleptic" which was meant to imply that these medications cause neurological side effects. However, the newer APs are much less likely to cause these side effects than the older agents. The one property shared by the whole class is that they treat psychosis; thus antipsychotics is the preferred term.

The most recent advancement in the field of antipsychotic pharmacology is the development of the newer, atypical APs, such as clozapine, olanzapine and risperidone. As opposed to the older, typical APs like chlorpromazine and haloperidol, the atypicals are less like to cause uncomfortable neurological side effects. However, these atypical agents are more likely to cause metabolic symptoms including weight gain, dyslipidemias, and diabetes. The typical APs are all potent dopamine (D_2) antagonists; atypical APs have more complex receptor profiles, and generally appear to combine dopamine and serotonin modulation.

Reference list of antipsychotics

		Typicals	Atypicals
Potency (only refers to Typicals):			
(High)	100:1	pimozide	**clozapine** (Clozaril)
	50:1	**haloperidol** (Haldol)	**olanzapine** (Zyprexa)
	10:1	fluphenazine	
		risperidone (Risperdal)	
	20:1	thiothixene	**quetiapine** (Seroquel)
		trifluoperazine	ziprasidone (Geodon)
			aripiprizole (Abilify)
			paliperidone (Invega)
	10:1	perphenazine	
		loxapine	
		molindone	
		piperacetazine	
	2:1	mesoridazine	
(Low)	1:1	**chlorpromazine** (Thorazine)	
		thioridazine	
		chlorprothixene	

Anxiolytics

Benzodiazepines

Benzodiazepines (BZDs) are a homogeneous group of medications. They all have the same mechanism of action: they bind to the BZD receptor locus of the chloride channel complex, which increases its responsiveness to GABA (the major inhibitory amino acid neurotransmitter in the brain).

BZDs all have three main properties (all due to increased GABA activity and all with relatively fast onset):

1. Anxiolysis/sedation
2. Anticonvulsant properties
3. Muscle relaxant properties

BZDs also all have three potential problems with chronic use, which arise from the brain's homeostatic response to their presence:

1. Tolerance
2. Withdrawal
3. Abuse potential

The choice of a BZD is mainly driven by their varying kinetic properties (half-life, speed of onset) and presence or absence of active metabolites).

Short half-life

Long half-life

Benzodiazepines

	Speed of Onset	Active Metabolites?
triazolam	Moderate	No
oxazepam	Slow	No
alprazolam (Xanax)	Fast	Yes
temazepam	Moderate	No
lorazepam (Ativan)	Fast	No
chlordiazepoxide	Moderate	Yes
clonazepam	Moderate	Yes
diazepam (Valium)	Very fast	Yes
flurazepam	Moderate	Yes

Buspirone

Buspirone is a unique anti-anxiety medication similar in many ways to antidepressants. It has serotonin effects, is not addictive, and takes several weeks to work. It has a benign side effect profile (similar to SSRIs). It has been demonstrated to be effective only for **generalized anxiety disorder** and is probably not useful (at least as monotherapy) for other anxiety disorders.

Electroconvulsive Therapy (ECT)

ECT involves the use of electricity to cause a global brain seizure under controlled conditions. ECT was developed in the late 1930s and is still recommended and used for appropriate patients. The major indications for ECT are *severe, debilitating mood disorders* (i.e., depression or mania) which are *not responsive to usual treatments* like medications and psychotherapy. The significant advantages of ECT compared to other treatments for mood disorders are faster speed and higher efficacy. ECT is performed under general anesthesia with the patient unconscious and temporarily paralyzed (so as to eliminate the unnecessary motor convulsion that accompanies uncontrolled seizures).

ECT generally has a low risk of medical or anesthetic complications, even in frail or elderly patients. The major limiting side effect of ECT is memory and general cognitive dysfunction, especially during the acute series of treatment. Fortunately, most cognitive effects of ECT are transient and resolve shortly after a course of treatment is completed, and more modern ECT techniques (most notably the use of non-dominant unilateral ECT) have been developed that carry a reduced risk of cognitive side effects. The other significant disadvantage to ECT is the relatively high relapse rate after successful treatment. Most recent evidence suggests that post-ECT medications and/or the use of maintenance ECT (single ECT treatments at bimonthly or monthly intervals) significantly reduce early relapse rates.

Other Devices: Currently Vagus Nerve Stimulation (VNS) is approved for the treatment of resistant depression. Transcranial magnetic stimulation

(TMS) is expected to receive FDA approval for resistant depression. Deep Brain stimulation (DBS) is under active investigation in the treatment of severe, refractory depression. (See mood disorders section of the syllabus).

Psychological Treatments

The main psychological treatment for psychiatric disorders is psychotherapy, or "talk therapy." Although there are many different schools and models for psychotherapy (the major ones will be described below), all psychotherapies do share some basic principles and assumptions.

In general, contact with other human beings can relieve distress, change behavior, and shift attitudes about ourselves and the world around us. Psychotherapy is, at its core, a means by which a therapist attempts to provide new interpersonal experiences for another human being so as to increase that person's ability to cope with and manage distress and to improve his or her ability to create and maintain emotionally satisfying relationships.

The principles in common to all psychotherapies are that:

1. The patient brings to psychotherapy an expectation that help is possible
2. The therapy will attempt to give the patient a schema for *making sense* of complex and confusing mental processes
3. The therapist offers the expectation that positive change is possible
4. The therapy will offer a safe place for taking risks

5. The therapy will expand the patient's horizons and increase his or her options

General Psychodynamics

Psychodynamics is, SIMPLY put, the study of mental forces and drives and how they motivate behavior. Most of the basic principles of the psychodynamic model of the human mind were articulated and described by Sigmund Freud (1856-1939) and, although many of the model's tenets and conclusions are controversial, it has profoundly influenced not only the field of psychiatry but also intellectual and artistic thinking in general in the 20th century. Psychoanalysis is the treatment modality that most directly springs from psychodynamics, but even more recently developed psychotherapies like cognitive-behavioral and interpersonal therapy (which have very different models and vocabularies) are best understood after a basic introduction to psychodynamics.

Freud developed two, compatible, models of the human mind. The first is the **topographic model** which separates thoughts, feelings, and perceptions into *conscious, preconscious,* and *unconscious* spaces in the mind. In the *conscious* mind are things we are aware of easily and directly; the *preconscious* are ideas and feelings we recognize only when we focus on them (for example, thinking about and being frightened of a big earthquake); the *unconscious* mind includes impulses, ideas, and fantasies that reside outside the reach of the conscious mind (although Freud thought the unconscious mind could be accessed under special circumstances, such as through dreams).

The second model is called the **tripartite model** which describes three major forces in the mind. The *id* is comprised of basic, primitive drives which Freud felt could all be explained by either a desire for increased pleasure (he called this "libido," but did not limit it to sexual pleasure) or a desire to be aggressive or destructive. The *ego* is a mediator function of the mind which balances and modifies the drives of the id with everyday reality and with the inhibitions of the superego. Examples of ego functions are to delay gratification or to substitute more acceptable for less acceptable pleasures. In the jargon of psychodynamics, the ego is where psychological **defense mechanisms** are developed and used in the mind. (Defense mechanisms are unconscious techniques we use to reduce anxiety and relieve psychological distress). Freud also saw most intellectual functions, like comprehension, judgment, and language, as part of the ego. The *superego* is the "conscience" of the mind, the externally imposed values, mores, and attitudes we take on from our families, societies, and cultures. Examples are societal prohibitions (like "stealing and cheating are bad") or family attitudes (like "you have to make a lot of money to be secure and happy"). Each person's superego is highly individual. Freud did not think of any of these three forces as good or bad; rather he saw them as metaphors to explain the varied responses of humans to stress and conflict, sometimes in mature and psychologically healthy ways, sometimes in less mature, maladaptive ways.

Freud argued that the id is innate, but the ego and superego develop during childhood and adolescence. His description for this process is called **psychosexual development**; essentially he feels that the libidinal and aggressive drives are met with and negotiated in different ways as a child

grows leading towards full emotional maturity. He breaks psychosexual development into distinct "stages:"

1. oral (0-18 months); when self and other are distinguished
2. anal (1.5-2.5 years); when basic physical self-control and independence, and ability to share are learned
3. phallic (2.5-4 years); when autonomous exploration, ability to take pride in accomplishments, and stable sense of self-worth develop
4. oedipal (4-6 years); when desire for special relationship with opposite sex parent and jealousy and hostility towards same sex parent develops leading to the ability to mediate pleasurable desires and manage aggressive and hostile emotions
5. latency (6-12 years); when complex social skills and rules are learned, intellectual and physical skills mastered
6. adolescence (12-adulthood); when sexual maturity and personal identity develop. Individual responsibility for actions assumed. Abstract thinking skills solidified

Freud felt that most adult psychopathology could be explained by incomplete or disrupted development during one of these childhood stages. Although the concept is useful, this explanation for adult psychiatric illnesses – at least for major Axis I psychiatric disorders like schizophrenia, major depression, bipolar disorder, and obsessive-compulsive disorder -- is no longer accepted.

Somatoform Disorders

Somatoform Disorders

Jose R. Maldonado, M.D.

Case

A 32 year old female transfers to a new gastroenterologist with complaints of abdominal pain that has been unrelieved by medications. She transferred from her previous physician of 2 years because she was not helping. Workups had included an upper and lower GI series, ultrasound, colonoscopy, laparoscopy and exploratory surgery with no definitive findings. She had been treated empirically with H-2 antagonists, antacids, and even narcotics with modest benefit.

In obtaining medical records from the previous physician, the new gastroenterologist discovers extensive outpatient and ER visits for a variety of complaints including chest pain, back pain, irregular menses, and headaches. The chest pain was severe enough that it was worked up with EKGs, and echocardiogram, and a cardiac catheterization.

In reviewing the history with the patient, the gastroenterologist discovers that the patient is currently undergoing a divorce and custody battle with significant financial stress. She recommends against further medical workup, since she believes the workup has been more than adequate, and instead advises the patient seek a counselor to help with the stress. The patient "fires" the gastroenterologist and makes an appointment with a new specialist for the following week.

What is the differential diagnosis?

What is the most likely diagnosis?

How would this disorder be distinguished from a factitious disorder?

What are the major medical risks from this disorder?

What treatment approaches would be advisable?

Introduction

Somatoform Disorders can be broadly defined as illnesses marked by physical symptoms that are clinically significant in causing distress or functional impairment, but cannot be fully explained by a general medical condition or organic cause. Instead, the somatic symptoms have a psychological basis. Somatization is defined as a process by which an individual consciously or unconsciously uses bodily symptoms for psychological purposes or personal gain. The concept of alexithymia is often used to explain the process of somatization. Alexithymia is defined as an impaired ability to recognize mood and to describe these emotions verbally. In other words, these patients are not "psychologically-minded" and express emotions through physical means. An example of alexithymia would be a child who is afraid of going to school who presents with chronic stomach pain.

An important aspect of somatoform disorders to consider is the extent of consciousness. Different disorders are characterized by different degrees to which symptoms are consciously produced. For example, patients with Somatization Disorder, Hypochondriasis, Pain Disorder, and Body Dysmorphic Disorder do not know that they are producing these symptoms psychologically – they actually believe that they have a physical problem. Similarly, they are *not delusional* either. In contrast, patients with Factitious Disorders and Malingering know that they are pretending to have these symptoms in order to receive some secondary gain. It is therefore quite important to distinguish between the unconscious somatoform disorders and the conscious Factitious Disorder and Malingering.

Regardless of the underlying level of consciousness as to the nature of their disorders, the presentation of a somatoform disorder demands a "thorough diagnostic investigation and therapeutic approach that focuses on the psychosocial history while formulating the extent of the patient's disease, the magnitude of the illness, and the degree to which the patient is suffering" (Stoudemire, 1998). In treating these patients, one must rule out true medical illnesses. Somatoform Disorders can co-exist with actual medical illnesses, and often co-exist with other psychiatric disorders. In addition, it is worth noting that some medical processes are difficult to diagnose and patients can be misdiagnosed with a Somatoform Disorder.

Somatoform Disorders are remarkably common, with a greater prevalence in the primary care setting. This carries important implications for all physicians, as we are likely to see someone with one of these disorders, regardless of specialty. Somatoform patients are frequently misdiagnosed and often receive extensive unnecessary medical tests and unnecessary and potentially iatrogenic procedures and treatments. These come at significant costs to the patient, the health care system and society as a whole.

Prevalence of Somatoform Disorders

As stated before, the prevalence of these disorders is remarkably high, with reported figures ranging between 5 - 40% of patient visits. In general, Somatoform Disorders are more common in women than men, with the exception of hypochondriasis, which is gender-equal. Prevalence of Somatization Disorder is 0.2% - 2% in women and under 0.2% in men. Conversion Disorder is about 1 - 3% of all patients referred to mental health

clinics. Hypochondriasis is present in 4 - 9% of all patients, with equal gender distribution. Pain disorder is common, but the actual prevalence is unknown. The prevalence of Body Dysmorphic Disorder is unknown.

The DSM-IV recognizes seven Somatoform Disorders

Somatization Disorder
Conversion Disorder
Hypochondriasis
Pain Disorder
Body Dysmorphic Disorder
Undifferentiated Somatoform Disorder
Somatoform Disorder, NOS (not otherwise specified)

General Differential Diagnosis of Somatoform Disorders

Organic Disease (lupus, brain tumor, MS, hypoglycemia, thyroid, etc.)
Factitious Disorder
Malingering
Psychotic Disorder with somatic delusions

General Treatment Guidelines for Somatoform Disorders

Patients with Somatoform Disorders are difficult to both diagnose and treat. It takes a high level of suspicion and awareness to recognize many of these patients. Once identified, many will be reluctant to receive psychological treatment. One must always consider that the disorder may be

primarily or secondarily associated with an underlying psychiatric syndrome, disorder, or stressor that has significance with respect to the presentation. Because of this, the physician must evaluate the patient's psychosocial history and evaluate their support system. The cornerstone of effective treatment lies in establishing a strong patient-physician relationship by scheduling regular appointments and offering ample reassurance. One should emphasize management of the physical symptoms, rather than a cure as the goal of treatment. Pharmacology is generally not an effective treatment, unless there is another psychiatric illness. In contrast, psychotherapy, hypnosis and relaxation therapy are useful treatments. Unfortunately, in many disorders, symptoms tend to return after treatment.

Somatization Disorder

Somatization Disorder is a chronic psychiatric disorder characterized by a pattern of multiple and recurrent physical symptoms for which a medical explanation is not present, OR, when a medical condition is present, the level of complaint or impairment is in excess of what would be expected based on physical findings. The patients' symptoms are presumably a response to or an expression of a preceding psychosocial stressor(s). Patients with Somatization Disorder commonly present with a long and complicated medical history, excessive medical treatment-seeking behavior, and a chronic pattern of multiple physical symptoms that are not feigned or under voluntary control (which distinguishes Somatization Disorder from Factitious Disorder or Malingering).

DSM-IV Diagnostic Criteria for Somatization Disorder

A. A history of multiple physical complaints that begin before age 30 and occur over a period of several years and results in treatment being sought or significant impairment in social, occupational, or other areas of functioning.

B. Each of the following criteria must have been met, with individual symptoms occurring at any time during the course of the disturbance:

1. **Four** pain symptoms: A history of pain in four different sites of function. (e.g., head, abdomen, back, joints, chest)
2. **Two** Gastrointestinal symptoms: A history of two GI symptoms other than pain. (e.g., nausea, bloating, vomiting, diarrhea, food intolerance)
3. **One** sexual symptom: A history of at least one sexual or reproductive symptom other than pain. (indifference, irregular menses, erectile dysfunction)
4. **One** pseudoneurological symptom.

C. The patient must exhibit either:

1) After appropriate investigation, each of the symptoms in Criterion (B) cannot be fully explained by a known medical condition or the direct effect of a substance.

2) When there is a related general medical condition, the physical complaints or resulting social or occupational impairment are in excess of what would be expected from the history, physical examination, and laboratory findings.

D. The symptoms are not intentionally produced or feigned.

*The most important diagnostic feature of Somatization Disorder is recurrent, multiple somatic complaints for a duration of several years, with the seeking

of medical attention for these symptoms. Thus, it is important for all physicians to be conscious of this disorder, as it is likely to present at some time in your career, regardless of specialty.

Course and Treatment of Somatization Disorder

Somatization Disorder usually presents by adolescence, and is a chronic, usually lifelong illness. As stated above, the diagnostic criteria must be met by age 30. Stressful life events will often cause an increase in symptoms and symptoms severity.

Unfortunately, there are many complications in the treatment of Somatization Disorder. Somatizing patients have little initial insight into their condition, and usually seek medical attention without recognizing a link between the physical symptoms and psychosocial disorder/stressors. These patients' medical history is characterized by excessive utilization of the health care system and "doctor-shopping" - the tendency of patients, who are unsatisfied with their physician, to move from physician to physician, in search of one who can better treat their physical symptoms. In addition, because these patients see themselves as being physically ill, they are relatively resistant to psychiatric treatment. In fact, the majority of somatizing patients reject psychiatric intervention. Thus, doctor-patient conflicts are quite common. In addition, because the psychiatric etiology of the disorder is often missed, the somatizing patient is often subjected to unnecessary exposure to diagnostic and treatment modalities, some of which can have iatrogenic side effects. (* N.B. Iatrogenic: Denoting response to medical or surgical treatment, induced by the treatment itself; usually describes unfavorable responses.)

Given these many complications, it is clear that the cornerstone of treatment of Somatization Disorder is the development of a trusting doctor-patient relationship. Treatment should be centered on the management of the psychiatric aspects of the disorder, rather than on a disease cure. Physicians should schedule regular appointments, be thorough in explaining all diagnostic results and treatment options, and extensively reassure the patient. One should make every attempt to reduce the amount of testing and invasive treatments and instead strive to open the door to the discussion of psychological problems and mental health consultation. A cure is seldom achieved, but the recurrent symptoms can be diminished and ideally exchanged for a dependence on a clinic or physician.

Conversion Disorder

Conversion symptoms refer to symptoms/deficits in motor or sensory function that suggest some medical disorder, but cannot be explained by any physiological mechanism. Rather, the symptoms are associated with psychological factors. The initial loss of motor or sensory function is usually abrupt and dramatic, follows a psychological stressor/event, and often has symbolic significance. Conversion Disorder is usually seen in ambulatory settings or the emergency departments, and is very common in medical practice. It is estimated that 20-25% of patients in a general medical setting exhibit conversion disorder symptoms. The course is often short-lived and generally responds to therapeutic modalities that even offer the suggestion of cure.

DSM-IV Diagnostic Criteria for Conversion Disorder

A. One or more symptoms or deficits affecting voluntary motor or sensory function that suggest a neurological or other general medical condition. [e.g. motor paralysis, seizures, lump in throat, paresthesias (abnormal sensations), anesthesias, and visual problems (e.g. blindness, tunnel vision)]
B. Psychological factors are judged to be associated with the symptoms or deficits because conflicts or other stressors precede initiation or exacerbation of the symptoms or deficits.
C. The symptom/deficit is not intentionally produced or feigned.
D. After appropriate investigation, the symptom cannot be fully explained by a general medical condition, or by the direct effect of a substance, or a culturally sanctioned behavior or experience.
E. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.
F. The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of Somatization Disorder, and is not better accounted for by another mental disorder.

Basis of Conversion Disorder

Conversions can be best thought of as an adaptation to a difficult or frustrating life experience. Freud's concept of conversion was that the conversion resulted from the substitution of a somatic symptom for a repressed idea or some form of psychological conflict. While this Freudian

concept is congruent with the presentation of some patients, all conversion symptoms will serve at least one of four functions. (1) Conversion may be a means to permit expression of a forbidden wish or impulse. (2) Conversion may also be a way to impose punishment on oneself via the disabling symptom, for a forbidden wish or wrongdoing. (3) Conversion may also allow the patient to remove him/herself from an overwhelming life-threatening or traumatic situation (primary gain - keeping internal conflicts from outside the patient's awareness). (4) Finally, conversions function to allow one to assume the sick role to allow gratification of dependency needs (secondary gain - patient accrues the advantages and benefits that result from the sick role).

Conversion Disorder Demographics

Conversion Disorders are very common, prevalent in as many as 20-25% of general medical patients. The disorder is more prevalent in women, with a 1:2-5 M:F ratio. The onset may occur at any age, but is most common in adolescence and early childhood, with a mean age of onset of 17 in women, 25 in men. There is a higher incidence among lower socioeconomic groups, rural populations, and low levels of education.

Conversion Disorder Comorbidity

Conversion Disorder is almost always associated with another disorder, either psychiatric or neurological. The seven major comorbid psychiatric disorders: Major Depression, Somatization Disorder (Briquet's Syndrome), Anxiety Disorders, Alcohol Abuse, Dissociative Disorders, Depersonalization Disorder, Personality Disorder (usually, but not exclusively, Histrionic and

Dependent Personality Disorders, in women; Antisocial Personality Disorder, in men).

In addition to psychiatric disorders, conversion disorder is often comorbid with neurological disorders. One major common comorbity is head trauma (found to exist with Conversion Disorder in 25% of men, 11% of women). In addition, 65-70% of patients with Conversion Disorder showed evidence of preceding or co-existing neurologic disorders, most commonly: seizure disorders, CNS tumors, Multiple Sclerosis, and intellectual subnormality.

Clinical Presentation of Conversion Disorder

By definition, patients with Conversion Disorder present with some sort of motor or sensory deficit. These frequently present as sensory disturbances such as anesthesia, paresthesia, visual problems or movement disorders. Movement disorders can be quite varied and include: tremors of limbs, head, and trunk, stereotyped tics and jerks, body spasms, paresis, paralysis, dissociation of function, dysphagia, or aphonia. It is significant to note that despite these motor abnormalities, reflexes remain normal, there are no fasciculations or muscle atrophy, and EMG findings are normal. This is a clear indication that the motor abnormalities are due not to a physiological disorder, but rather a psychological one. Patients with Conversion Disorder also frequently present with convulsive phenomena or psychosis.

Conversion Disorders often present with other classic associated features. Chief among these is that the conversion symptoms will usually

conform to the patient's concept of disease rather than to expected pathophysiological or anatomical mechanisms. In addition, as part of the definition, there must be an absence of a medical cause or sufficient explanation and the identification of a psychological cause (a "smoking gun" - the psychological event or stressor that initiated or exacerbated the somatic symptom). Conversion patients may exhibit an indifferent attitude toward their somatic symptom, and have no interest in what the symptom means or in resolving the symptom ("La Belle Indifference"). Conversion symptoms are often identified with some sort of symbolic significance (a loss of vision in a man who had an accident because he didn't see a truck coming). Finally, conversion symptoms are frequently modeled after an important figure in the patient's life.

Differential Diagnosis of Conversion Disorder

Unidentified Organic Process
Co-morbid Psychiatric Illness (see above)
Factitious Disorder
Malingering

Treatment of Conversion Disorder

In treating Conversion Disorder, it is important to understand that a symptom whose psychological meaning has not been understood, or that still serves a purpose, will not be easily extinguished. Every symptom presented by the patient has a function or meaning that must be understood and respected. Patients will not give up a symptom until they feel strong enough to

do without it. Patients always do better when there is a powerful incentive for a symptom free state. Therapy for Conversion Disorder involves some form of psychotherapy. The purpose of therapy is to teach patients how much control they have over their symptoms, their bodies and their lives. A physician should provide patients with permissive suggestions that allow patients to give up their symptoms whenever they feel they are ready to deal without them. The following are the main forms of psychotherapy utilized in the treatment of Conversion Disorder: Psychoanalysis, Behavioral Therapy, Conventional Psychotherapy, Medication Facilitated Interview, Hypnosis Facilitated Psychotherapy, and Psychopharmacology.

Hypnosis

Hypnosis facilitated psychotherapy is especially useful in patients with Conversion Disorder, as they are usually highly hypnotizable. In fact, it may be that the use of hypnosis is exquisitely connected to the patient's symptoms: "Patients suffering from a conversion disorder may indeed be using their own hypnotic capacity to dissociate in order to displace the uncomfortable feelings or affects into a chosen body part which then becomes dysfunctional." (Maldonado, 1996) Thus, hypnosis is a reasonable and often effective adjuvant to medical treatment. In the course of therapy, we attempt to teach patients how to produce a trance state, and later, how to recreate and control the symptoms of conversion. Therefore, the goal is one of teaching the patients that the control of their symptoms resides in themselves, not in external factors or people.

Hypnosis is used sequentially - first for symptom exploration, then for symptom alteration, which may involve symptom substitution or symptom extinction. In this manner, one transfers the control to the patient and attempts to maximize their functioning. In addition, the use of hypnosis may provide patients with a "face-saving" opportunity when their symptoms are no longer needed. While hypnotic exploration of unconscious symptoms is highly useful, one must exercise caution in its practice. One must remember that the conversion symptoms serve some function, and that their presence and utility must be respected. To do so, there are three "golden rules" of hypnosis that a therapist should follow:

1. Never eliminate a symptom without first understanding its value and purpose.
2. Never take away a symptom before having provided the patient with a better defense.
3. Always obtain the patient's agreement before exploring and/or removing a symptom.

*Complications and Warnings Concerning the Treatment of Conversion Disorder

Patients suffering from Conversion Disorder may also suffer other "real" medical diseases. These patients may be labeled as "fakers", malingers, or just not taken seriously. Studies suggest that a considerable number of patients diagnosed with Conversion Disorder go on to develop a disease process at some time in the future that may explain the pathological findings presented initially.

Hypochondriasis

Hypochondriasis is defined as the fear of having a disease or the belief that one has a disease based on a misinterpretation of physical signs or symptoms, but in the absence of any identifiable organic pathology and despite medical evaluation and physician reassurances to the contrary. It is essentially an exaggerated preoccupation with illness. Note that Hypochondriasis differs from Conversion & Somatization Disorders, in that Hypochondriasis is characterized not only by a bodily complaint without pathology, but also by a mistaken conviction or fear of having a disease.

DSM-IV Diagnostic Criteria for Hypochondriasis

A. Preoccupation with fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms.
B. The preoccupation persists despite appropriate medical evaluation and reassurance.
C. The belief in criteria A is not of delusional intensity.
D. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
E. The duration of the disturbance is at least 6 months.
F. The preoccupation is not better accounted for by Generalized Anxiety Disorder, Obsessive-compulsive Disorder, a Major Depressive Disorder, Separation Anxiety, or another Somatoform Disorder.

Etiology of Hypochondriasis

While a biological etiology of Hypochondriasis has yet to be elucidated, there are three primary theories that account for the presentation. Psychoanalytic theories describe hypochondriac symptoms as the displacement of unconscious or repressed hostility toward other people onto the patient's own body. Behavioral theories suggest that Hypochondriasis results from an individual's selective attention to ordinary fluctuations in bodily functions that lead to misinterpretations of these variations as signs of serious disease. Finally, social theory states that Hypochondriasis symptoms are seen as a patient's way of adopting the sick role in order to recruit sympathy and/or help from others. One, some, all, or none of these theories may help to explain the actual etiology of a particular case and it is important to consider these and other causes when treating such patients.

Hypochondriasis Course and Treatment

The most common onset of Hypochondriasis is in middle or old age and follows a chronic course. The disorder is equally prevalent in women and men. The symptoms may last a few years, and even alternate with periods in which few symptoms present. These patients excessively utilize the health care system in seeking examinations, test, and treatments. When a physical illness is not found, patients tend to request further testing or second opinions rather than being reassured by the lack of findings. Accordingly, "doctor shopping" is common and physician-patient conflicts often occur when physicians fail to reassure their patients and reduce their distress. Therefore, treatment of Hypochondriasis presents a substantial challenge. The first step is

to exclude a true medical illness or other primary psychiatric disorder such as major depression. Once such maladies have been excluded, the treatment plan is similar to the treatment of Somatization Disorder. A physician should schedule regular doctor visits that reassure the patient that their symptoms and distress are taken seriously. Once a trusting relationship is established, mental help treatment should then be encouraged, not as a substitute for, but in addition to continued medical care. Fortunately, the prognosis is favorable, as approximately 50% of patients with Hypochondriasis improve over the course of their lives.

Pain Disorder

The characteristic feature of Pain Disorder is a painful condition that is sufficiently severe to demand clinical attention, but that defies anatomical patterns or pathological explanation, OR is grossly in excess of what can be physiologically explained. Instead of a physiologically-based explanation, psychological factors are found to account for the clinical presentation. Pain Disorder is divided into an acute (lasts less than 6 months) and chronic (more than 6 months) designation.

DSM-IV Diagnostic Criteria for Pain Disorder

*The diagnosis of Pain Disorder is a difficult diagnosis to make, as a large number of etiologies must be ruled out.

A. Pain in one or more anatomical sites is the predominant focus of the clinical presentation and is of sufficient severity to warrant clinical attention.
B. The pain causes significant distress or impairment in social, occupational, or other important areas of functioning.
C. Psychological factors are judged to have an important role in the onset, severity, exacerbation, or maintenance of the pain.
D. The symptom or deficit is not produced or malingered.
E. The pain is not better accounted for by a Mood, Anxiety, or Psychotic disorder, and does not meet criteria for Dyspareunia. (Dsypareunia is defined as the occurrence of pain during sexual intercourse).

Etiology of Pain Disorder

Like all somatoform disorders, the exact etiology of Pain Disorder is uncertain. Nevertheless, there are two predominant theories that attempt to describe the etiology. Psychoanalytic theory interprets Pain Disorder as the expression of conflict between a strict superego requiring achievement and independence with unmet childhood dependency needs. Social-behavioral theory sees the pain symptoms as attention-seeking behavior usually learned during childhood and continuing as an inappropriate way of seeking attention.

Course and Treatment of Pain Disorder

Unfortunately, little is known regarding the course of Pain Disorder, as relevant research is lacking. The onset is usually in the 30's or 40's and the symptoms can be disabling, especially if the physiological component is significant. The treatment of Pain Disorder can be quite difficult, and again rests on establishing a firm and trusting therapeutic relationship with the patient. It is absolutely essential to rule out all possible organic factors. The focus of care is on rehabilitation and improving functional capacity rather than on medical diagnosis and cure. The most appropriate treatment setting involves management by a multidisciplinary team experienced in the treatment of pain. In addition to psychotherapeutic approaches, there are two primary psychopharmacological considerations in the treatment of Pain Disorder. First, because these patients are primarily afflicted by pain symptoms, they are frequently prescribed pain medication, and many patients may become addicted to these drugs. Second, there is substantial evidence that anti-depressants, especially TCAs, are useful in treating Pain Disorder.

Body Dysmorphic Disorder

The central feature of Body Dysmorphic Disorder is a gross preoccupation with an imagined or minor defect in physical appearance. The defect is usually, but not always in the face or head. These patients are intensely distressed and frequently seek unnecessary surgery to repair the defect. The patient is never satisfied, even after surgical corrections. This disorder first appeared in the DSM-III-R, which introduced the important distinction between *non-delusional* Body Dysmorphic Disorder and

Delusional Disorder, somatic type, which is a psychotic disorder. This distinction still holds, and it is important to distinguish between the delusional and non-delusional disorders.

DSM-IV Diagnostic Criteria for Body Dysmorphic Disorder

1. Preoccupation with an imagined defect in appearance. If a slight physical anomaly is present, the person's concern is markedly excessive.
2. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
3. The preoccupation is not better accounted for by another mental disorder (Anorexia Nervosa, Delusional Disorder, psychotic type)

Course and Treatment of Body Dysmorphic Disorder

Body Dysmorphic Disorder is usually a chronic illness, with symptoms persisting and sometimes worsening over time. The usual age of onset is in the late teens. This disorder is characterized by a high degree of suffering and impairment, although their level of concern and distress can vary over time. The preoccupation with the defect can take up much of the day and cause intense distress, avoidant behavior, suicidal ideation, and unnecessary plastic surgery. Unfortunately, when these patients do undergo surgery, they are often unsatisfied with the results, and commonly the scars become the new focus of attention or preoccupation. Therefore, plastic surgery must be cautiously used. The three major treatment modalities include medical therapy (antidepressants), behavioral therapy (systemic desensitization and exposure therapy), and psychotherapy *prior* to plastic surgery. However, there is no

clear consensus regarding optimal treatment, although everyone agrees that treatment should be psychiatric and not surgical.

Malingering

Malingering is the conscious simulation or exaggeration of physical or mental illness for financial or other obvious gain (e.g., avoiding work or incarceration). The malingering patient avoids treatment, and the symptoms improve as soon as the desired gain is obtained. Unlike somatoform disorders, this is a *conscious* disorder, in which the patient actively induces their symptoms.

DSM-IV Diagnostic Criteria for Malingering

1. The intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives (e.g., avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, obtaining drugs), etcetera.
2. Under limited circumstances malingering may represent adaptive behavior (e.g., feigning illness while captive of the enemy during wartime).

Factitious Disorder

Patients with Factitious Disorder know they are pretending to have a mental/physical illness or are actually inducing physical illness to achieve the role of the patient and obtain medical attention. Note that unlike somatoform

disorders, this is a conscious disorder in which the patient actively induces their symptoms.

DMS-IV Diagnostic Criteria for Factitious Disorder

1. Intentional production or feigning of physical or psychological signs or symptoms.
2. The motivation for the behavior is to assume the sick role.
3. External incentives for the behavior (e.g., economic gain, avoiding legal responsibility) are absent.

* Note that this last criterion distinguishes Factitious Disorder from Malingering.

A seldom seen, but important subset of Factitious Disorders is Factitious by Proxy (a.k.a. Munchausen Syndrome By Proxy), in which an adult, usually a parent, feigns or induces illness in a child to obtain medical attention. This is a form of child abuse and must be reported!

Etiology of Factitious Disorder and Factitious by Proxy

Factitious patients often have a history of childhood illness that resulted in protected medical care. In the course of this illness, the patients felt cared for and protected. Upon resolution of their initial illness, the loss of the sick role and the accompanying attention drives patients to feign symptoms to regain attention. In addition, many patients have a history of childhood abuse or neglect.

Course and Diagnosis of Factitious Disorders

The most commonly feigned symptoms are abdominal pain, fever, blood in urine (hematuria), tachycardia, skin lesions, and seizures. Their preoccupation with illness and medical care frequently causes work, school, and social relationships to suffer. In the course of diagnosis and treatment, a factitious patient may undergo expensive and unnecessary medical procedures or receive unnecessary medication, both of which are clearly detrimental to the patient and the resources of the medical community as a whole.

Unfortunately, the diagnosis of Factitious Disorder/Malingering is inherently difficult as a physician is essentially refuting a patient's claims. There are really only three definitive and foolproof ways to diagnose Factitious or Malingering Disorder. The first is that patient is caught in the act of injuring himself or herself. The second is when the patient confesses to causing self-harm. The final way to diagnose such a patient is if there is simply a preponderance of scientific evidence against their claims.

A Comparison of Clinical Features of Somatoform, Factitious, and Malingering Disorders

Disorder	Clinical Presentation	Motivation for Symptoms	Demographics And Epidemiology
Somatization Disorder	Polysymptomatic, recurrent and chronic symptoms, "sickly" by history	Unconscious	Female predominance 20:1; younger age; 5-10% incidence in primary care setting
Conversion Disorder	Monosymptomatic, Mostly acute, simulates disease	Unconscious	Female predominance, Rural/Lower socioeconomic class??
Pain Disorder	Pain syndrome simulated or magnified by physiological factors	Unconscious	Female predominance 2:1; familial pattern; 4^{th}-5^{th} decade onset
Hypochondriasis	Disease concern or preoccupation	Unconscious	Previous physical disease; middle or older age; no gender predominance
Body Dismorphic Disorder	Subjective feelings of ugliness or concern with body defect	Unconscious	Adolescence or young adult; Female predominance??
Factitious Disorder	Feigned or simulated physical symptoms, signs, or disease	Conscious effort to assume patient status	Female; younger; socially conforming; employed in medical field
Malingering	Feigned or simulated with physical or psychological symptoms	Conscious response or external incentives	Male predominance?? Psychosocial stress or failure present

* The diagnostic subtypes of somatization disorder, conversion disorder, pain disorder, hypochondriasis, and body dismorphic disorder all fall under the umbrella of "Somatoform Disorders". Factitious and malingering disorders are each separate and unique disorders.

Return to the Case

The patient appears to have a somatoform disorder given the lack of an apparent physical basis for a variety of complaints. The diagnosis of somatoform disorder should only be considered a diagnosis of exclusion. It is possible that the patient has an organic problem that has not fully manifested or can't be detected by the available technology.

That being said, the most likely diagnosis would be a somatization disorder. The diagnosis is supported by a history of shifting physical complaints of many years duration with an unclear physical basis. This disorder would be distinguished from a factitious disorder in that the symptoms appear to be an unconscious manifestation of stress. There is no evidence presented here that the she is intentionally producing the symptoms for a specific secondary gain such as disability benefits (malingering) or simply because she enjoys being the center of medical attention (Munchausen's Syndrome).

The major medical risks for this lady are the iatrogenic risks that multiple medical procedures carry. There may not be much wrong with her heart or GI tract now but each cardiac catheterization or exploratory surgery carries the risk of new, very real, medical problems. Another medical risk is the risk that her physicians may be more likely to discount any new symptoms because of the extensive history of working up physical symptoms to no avail in the past. One of those new symptoms may represent a serious condition that is not worked up aggressively because of this history.

There is no ideal treatment for somatization disorder. Among the better treatments, if possible, is to have frequent, regularly scheduled visits with a physician she trusts whether not the patient is having symptoms. These frequent visits circumvent the need for a patient for a patient to unconsciously produce a symptom to seek help. During these sessions, each complaint should be respectfully heard out and pursued within reason. In addition, the opportunity to talk about what's going on the patient's life can be very helpful. Referral to an empathic therapist can be helpful in the rare instances when a patient will accept the referral. Most somatization disorder patients are convinced of the medical nature of their symptoms and thus may be insulted at the suggestion of an emotional basis for the disorder.

Dissociative Disorders

Dissociative Disorders

Jose R. Maldonado, M.D.

Introduction

Dissociative disorders can be understood as the pathological separation of aspects of mental functioning, including perception, memory, identity, and consciousness, which would normally be processed together.
The several types of dissociative disorder are listed in the following table.

Dissociative Disorders

Syndrome	Problem (lack of integration in)
Dissociative Amnesia	Memory
Dissociative Fugue & Dissociative Identity Disorder	Identity and consciousness
Depersonalization	Perception

All individuals experience some degrees of dissociative alterations of consciousness, information processing and memory storage and retrieval. Examples include "spacing out" or daydreaming. Other phenomena in which dissociation plays a role include hypnosis and meditation. When dissociation becomes extreme, it can cause significant impairment of function.

Pierre Janet is the first to associate dissociation with a pathological process. According to him, it is a condition in which traumatic memories could not be

integrated into preexisting cognitive structures and instead were split off to form "subconscious fixed ideas" that existed outside of an individual's usual conscious experience and continued to exert an effect on behavior. Clinical manifestations vary from transient depersonalization to dramatic episodes of amnesia and fugue to lifelong patterns of disturbance in patients with dissociative identity disorder.

Dissociation usually occurs in the face of overwhelmingly traumatic experiences and memories. Trauma has been linked to depersonalization disorder, dissociative amnesia, dissociative fugue, dissociative identity disorder, acute stress disorder, Post-Traumatic Stress Disorder, borderline personality disorder and bulimia. The traumatic experience forces the victim to reorganize mental and psycho-physiological processes in order to buffer the immediate impact of the trauma (Maldonado & Spiegel 1994). Dissociative defenses can initially be adaptive and directed at maintaining control at times of overwhelming stress. However, some trauma victims develop persistent dissociative, amnesic and anxiety-like symptoms. "The ultimate sequelae of trauma may be the development of Acute Stress Disorder, Post-Traumatic Stress Disorder or a Dissociative Disorder" (American Psychiatric Association 1994).

Not everyone exposed to trauma will develop dissociative symptoms: less than 25% of soldiers exposed to combat go on to develop dissociation or ASD/PTSD (Keane and Fairbank 1983). This suggests that other factors must be involved in the production of dissociative symptoms.

Trauma and hypnotizability

Many trauma victims describe experiencing detachment at the time of the trauma. More commonly seen in victims of repeated trauma. These could represent episodes of spontaneously induced self-hypnosis. Exposure to stressful events may be one of the paths that naturally lead toward the development of high hypnotizability. Several authors have reported a positive correlation between severity of punishment undergone during childhood and hypnotizability levels. (Chu & Dill 1990; Hilgard 1970, 1984; Nash & Lynn 1986; Nash, Lynn & Givens 1984; Putnam 1993; Spiegel 1988, 1990; Spiegel & Cardena 1991; Spiegel et al., 1988).

There is an extraordinarily high incidence of dissociative-like defenses and other pathological symptoms in victims of early childhood abuse. There is evidence to suggest that victims of intense trauma use their dissociative defenses to guard themselves from the full impact of the traumatic experience. Later in life, many of these victims will again use their already mastered hypnotic-like capacities in the face of further traumatization.

Dissociative Amnesia (Psychogenic Amnesia)

Hallmark: inability to recall important personal information beyond what could be explained by ordinary forgetfulness. This is the most common of all dissociative disorders. Amnesia usually involves difficulties with explicit or episodic memory. Memory deficits are usually reversible as the amnesia causes difficulties in retrieval rather than encoding or storage. There are two clinical presentations: acute and generally severe amnesia; chronic and insidious amnesia. It is a disorder and a symptom found in a number of other

dissociative and anxiety disorders (ASD, PTSD, somatization disorder, dissociative fugue, and dissociative identity disorder). An episode of dissociative amnesia is usually precipitated by a particularly intense psychological trauma. There is a direct relationship between the *severity* of the exposure to trauma and the *incidence* of amnesia. Amnesic patients are usually *aware* of their memory loss, *capable* of learning new information, and have *intact* cognition.

Epidemiology: unknown. It is most common in adolescent and young adult females and is most rare in elderly.

Etiology: One hypothesis suggests that amnesia is post-traumatic and generally occurs within the context of severe psychosocial stress.

Duration: A few days to a few years. Spontaneous resolution of the symptoms is not uncommon. Gradual recovery is more common.

Differential Diagnosis

Any of several neurological etiologies of amnesia should be ruled out by careful medical-neurological evaluation. Here is the list of diseases to keep in mind:

Epilepsy, brain malignancies, head trauma, Rx-side effects, drug abuse, acute intoxication, cardiovascular/metabolic abnormalities, other dissociative disorders, organic brain syndrome, factitious disorder, malingering.

Dissociative Fugue (Psychogenic Fugue)

Hallmark: "Sudden, unexpected travel away from home or one's customary place of daily activities, with inability to recall some or all of one's past." Accompanied by a sense of confusion about personal identity or assumption of a new identity. No "alters"- patients appear 'normal'. Patients typically present for help after the fugue wanting to recall what happened.

Predisposing factors: The pathological cause of fugue is not understood. There is a prevalence of 0.2%. The incidence & sex ratio of amnesia is not known. This phenomenon appears to be precipitated by extreme psychosocial stress, personal &/or financial pressures or losses, heavy alcohol use or overwhelming stress (i.e., assault, rape). A history of head trauma is not uncommon.

Dissociative Identity Disorder (Multiple Personality Disorder)

Dissociative identity disorder is characterized by the presence of 2 or more distinct identities or personality states that recurrently take control of the subject's behavior. Additionally, there must be some degree of dissociative amnesia present to make the diagnosis. As diagnostic evaluation and treatment proceed, various personality parts may recurrently manifest. On average there are 2 to 4 personalities at initial presentation, but 13 to 15 personalities may be discovered at the time of treatment. The underlying problem of dissociative identity disorder might be a failure to integrate various aspects of identity, memory and consciousness which in turn results in memory gaps in personal history with asymmetric & selective amnesia.

Typical symptoms of dissociative identity disorder include memory deficits, moodiness, erratic and unpredictable behavior, depression, self-mutilation, suicidal ideation or attempts, or the overt manifestation of an alternate personality. Transition is usually sudden and is commonly triggered by environmental factors.

"Alter" identities may have different names, sexes, ages and personal characteristics. Different personalities may reflect various attempts to cope with difficult issues and problems. Personalities can either have a name or named after their function/description. Physical and sexual abuse during childhood is the most commonly found etiological factor in these patients.
It is uncommon to find a DID patient who has not been exposed to intense trauma, usually physical (or sexual) abuse, to the point of also fulfilling Criterion A for the diagnosis of PTSD.

Factors associated with the development of DID

Almost all diagnosed patients report histories of exposure to overwhelming childhood trauma: sexual abuse (70-97%), physical abuse, non-sexual (75%), neglect, confinement, severe intimidation with physical harm, witnessing physical or sexual abuse of a sibling, witnessing the violent death of a relative or close friend, traumatic physical illness on self, near death experiences, etc. Other associating factors include victim's age, relationship of offender to the victim (one's relative versus a stranger), victim's natural hypnotic capacity, effects of state dependent memory, victim's developmental maturity. Affected individuals seem to have an increased tendency to enter dissociative states using hypnosis.

Epidemiology

DID more commonly presents during adolescence and young adulthood. Average age at diagnosis is 29 to 35 years. Limited data indicates that usually the appearance of the first alter personality occurs by age 12. The youngest case described in the literature was 3 y/o when diagnosed. It is more common in women than men (3-9:1). Females are reported to present more personalities (average of 15) than men (average of 8). Total average of 13 to 15 personalities. There is a high incidence among first-degree relatives. Estimated prevalence: 0.01%

Differential Diagnosis

Patients usually present with other associated psychiatric and medical syndromes: depression (85-88%), insomnia, suicide attempts or gestures, self-destructive behaviors, phobias, anxiety, panic attacks, substance abuse, auditory and visual hallucinations, somatization, "psychotic" behavior. There is a high incidence of dissociative symptoms: Amnesia: 85 to 98%, Fugue episodes: 55%, Depersonalization: 53%, Derealization: 54%. DID has been identified across all major racial groups, socio-economic classes, and cultures.

Depersonalization Disorder

Depersonalization is an alteration of experience in which a person feels detached from his or her body or mental process. Hallmark: persistent or recurrent episodes of feelings of detachment or estrangement from oneself. Reality testing is intact. Incidence and prevalence is unknown. People with no psychiatric condition have also transiently experienced it.

Disorders associated with depersonalization

The symptom of depersonalization occurs in a number of psychiatric conditions including agoraphobia and panic disorder, acute and post-traumatic stress disorder, schizophrenia, other dissociative disorders, personality disorders and psychotic mood disorder. There is also a number of Neurological disorders associated with depersonalization: epilepsy, Meniere's disease, sensory deprivation, sleep deprivation, hyperventilation, migraine headaches, and acute drug intoxication & withdrawal. (*N.B. Meniere's disease is an affection characterized clinically by vertigo, nausea, vomiting, tinnitus, and progressive hearing loss due to hydrops of the endolymphatic duct.)

The *symptom* of depersonalization has been described as being the third most common psychiatric symptom. Under severe stress up to 50% of all adults have experienced at least one single brief episode of depersonalization. The sex distribution is unknown. Although the incidence ratio of women to men is ~2-4:1. There is no known familial pattern.

Etiology: ranges from the completely physiological to the purely psychological, to combinations of both. Exposure to traumatic experiences seems to be the common etiological factor. Course: usually chronic.

Treatment of Dissociative Disorders

Dissociative Amnesia

No single treatment modality has been systematically studied nor is there established pharmacological treatment except for the use of drug-assisted interviews. Therapy involves two general steps: The initial step in the treatment is to provide a safe environment. Simply removing the person from the threatening situation has allowed for the spontaneous recovery of memory. The second step is to explore his or her distress through treatments. This includes hypnosis, barbiturate or benzodiazepine interview, caffeine (McCall 1992), methylphenidate (Hurwitz 1988). No study has addressed the efficacy of hypnosis.

Hypnosis

Hypnosis can be very effective in recovering and working through traumatic memories. Hypnosis can potentially reverse the amnesia, restructuring the events and defining the factors that led to the amnesia, establish appropriate defenses and mechanisms to prevent further dissociation. Advantage: allows for a controlled recovery of traumatic experience, at a pace the patient can tolerate. Extensive abreaction is not needed, nor recommended. Hypnosis allows the patient to access the memories and to reframe the experience. This work is similar to the working through of memories associated with PTSD. (*N.B. "Abreaction" comes from Freudian psychoanalysis and is an episode

of emotional release or catharsis associated with the bringing into conscious recollection previously repressed unpleasant experiences.)

Dissociative Fugue

There are no controlled studies. Information is based on case reports on limited numbers of patients. It was urged that treatment be undertaken as quickly as possible while the repressed material was more readily accessible, before the memories had consolidated into a nucleus, thereby increasing the possibility of future flight episodes. Treatment: provision of rest and assurances of safety, development of therapeutic relationship, recovery of personal identity, review of triggers or factors, reprocessing of traumatic material, reintegration of traumatic memories, return to his/her previous life.

Hypnosis and drug-facilitated interviews have commonly been used during the stages of recovery of personal identity and memories associated with the onset of the fugue. No pharmacotherapeutic agents have been systematically studied.

Patients may also experience spontaneous memory recovery upon removal from the stressful situation, when exposed to cues from their past, or when they feel psychologically safe. Psychodynamic psychotherapy may help to address the conflicts that precipitated the amnesia or fugue, thereby reducing subsequent dissociation under stress.

Dissociative Identity Disorder

Treatment of DID involves:

1) Development of a therapeutic relationship based on safety and trust
2) Negotiation with the patient about cooperation with treatment
3) Development of a contract against harm to self or others
4) History taking and understanding personality structure
5) Abreaction and working through of traumatic experiences and frequently, repressed or dissociated material
6) Negotiating and modulating 'conflicts' among aspects of identity and personality states
7) Development of mature and more appropriate, non-dissociative defenses
8) Working toward integration of alters

Hypnosis can facilitate control over dissociative episodes and integration of traumatic memories. Special efforts such as development of a social network and support system are often required to prevent further traumatization. Protection must be provided from abusive family members. After integration, further work focuses on working with residual or renewed dissociative responses to external stress or internal conflicts and to further integration with society. Integration is usually desirable, but sometimes a reasonable degree of conflict-free collaboration among the personalities is all that can be achieved.

The most common treatment modality used is individual psychotherapy facilitated by hypnosis. The average DID patient is seen twice a week for a period of about 4 years. Kluft (1985a) has established that there is no

spontaneous remission if left untreated. The treatment of the many symptoms and associated diagnoses do not help in the resolution of the problem unless the dissociation is addressed directly. There has been no comprehensive systematic research into the treatment of DID. Kluft emphasizes the need for continuity of treatment beyond initial fusion.

Clinical Research on Individual Modalities

The scientific literature on the treatment of DID is largely descriptive and prescriptive. There has been no systematic research support for the following which is based on case reports and the reflections of individual therapists experienced in working with DID. The virtue of these reports is that they represent the conclusions drawn by those practitioners who have led the field in the treatment of DID. Their limitation is that they are based on limited samples, without proper controlled scientific scrutiny and comparison, and they may be subject to the expectations and biases of their reporters.

Individual Psychotherapy is widely considered the treatment of choice for DID. Homogenous Group Therapy: some patients may benefit from group therapy as a supplemental or adjunctive intervention. Group therapy presents an opportunity for the DID patient to participate with other humans in a social context. The group presents a potential to learn interaction for DID patients who are already in individual therapy. Family Treatments: Benjamin suggests the use of family-based approach since the disorder is precisely about the failure of a healthy family process. Some have proposed that family interventions should be an integral part of an overall treatment plan. It is essential that the abuse that precipitated dissociation in the DID child has

ceased; the abuser be identified and be willing to admit to the abuse and to change; and that this can be verified.

Hypnosis as a treatment tool

Traumatic memories may be elicited during psychotherapy without techniques for memory enhancement. Nevertheless, hypnosis can facilitate access to repressed memories, which have not yet emerged. Many trauma victims respond to the traumatic event by using dissociative-like defenses. Most patients suffering from dissociative disorders are highly hypnotizable. If hypnotic-like defenses are used during traumatic experiences, it makes sense to use it to retrieve memories and affects associated with the original trauma: theory of state-dependent memory. Hypnosis is used both as a diagnostic tool and treatment technique. Hypnosis allows for the recovery and reprocessing of recovered memories at a pace the patient can tolerate. There are no systematic studies regarding its efficacy. Properly done, hypnosis facilitates symbolic restructuring of the traumatic experience.

Psychopharmacology of DID

Little is known about the rational approach to the use of psychoactive substances in the treatment of DID. There is no good evidence that medication of any type has a direct therapeutic effect on the dissociative process manifested by DID patients. Most dissociative symptoms seem relatively impervious to pharmacological intervention. There has been no double-blind controlled study of psychopharmacological agents in DID patients. The little data available is limited to case reports and small uncontrolled samples. Treatment has been limited to control of signs and symptoms afflicting DID patients. Several researchers have suggested that there is variability in

response to medication related to the predominance of different personality states. Antidepressant medications should be used only when the host and a large number of alter personalities experience symptoms of MDD (Major Depressive Disorder). There is however poor consistent therapeutic results across alters. MAOI's are strongly discouraged (high lethality). Newer SSRI's are effective at reducing co-morbid depressive symptoms and have the advantage of far less lethality in overdose.

Antipsychotics are associated with an extremely high incidence of adverse side effects. Complications can include creation of new alters in response to medication. They are rarely useful for the control of extreme agitation or disorganization; they are not helpful for maintenance. In DID patients misdiagnosed as schizophrenic, antipsychotics complicate the situation by flattening affective response.

Benzodiazepines should be limited to cases where high levels of anxiety are experienced across all Alters or where anxiety interferes with therapeutic work. Loewenstein et al. (1988) described the only systematic study of pharmacotherapy of DID. In his study, he used clonazepam successfully to control PTSD-like symptoms in a small sample (n = 5) of DID patients. After treatment patients exhibited improvement in sleep continuity, and decreased flashbacks and nightmares.

Anticonvulsants have limited use in DID patients. Only single case reports are available. Carbamazepine has been used in patients with concomitant bipolar affective disorder or epilepsy. No controlled studies have been done and no follow-up of reported cases has been published. Unless there are clear EEG

changes, anticonvulsants play no role and should be avoided due to serious potential side effects.

Autonomic system agents like clonidine and high-dose propranolol have been used for the treatment of hyperarousal, anxiety, poor impulse control, disorganized thinking, and rapid or uncontrolled switching in DID patients. Open trials suggest "good outcomes" with these agents. However, *no specific data* about the patients or the drug trials were reported such as the number of patients given either drug, actual success rate, co-morbid diagnoses in the sample, or prevalence of adverse drug reactions.

Electro-convulsive therapy (ECT): There are no controlled studies. Small, non-controlled samples. Bowman and Coons (1992) reported that, in a small (n=3) prospective study, there was a 50% drop in depression scores and marked clinical improvement with gains being maintained for at least 4 months. ECT did not affect the dissociative condition and should only be used when depression is experienced by most of the active alters.

Eye-Movement Desensitization and Reprocessing (EMDR): EMDR was initially described by Shapiro (1995). It is mostly applied to the treatment of traumatic memories and PTSD. No systematized or controlled studies on have been performed on DID. Reports confirming its usefulness are anecdotal. Studies present equivocal findings and do not confirm any specific effects.

Depersonalization Disorder

Treatment maybe used for accompanying symptoms such as anxiety, depression, or obsessions but the value of medication for depersonalization itself has not been carefully studied. Treatment modalities employed include: paradoxical intention, record keeping and positive reward, flooding, psychodynamic psychotherapy, psychoeducation, psycho-stimulants, antidepressants, antipsychotics, anticonvulsants, benzodiazepines, electroconvulsive therapy (ECT), and hypnosis. Hypnosis has been described in multiple case reports; suggestibility has been implicated in the etiology of the disorder and is recommend as a treatment modality. All studies were inadequately designed, had small samples, no double blind, and no controls.

Hypnosis in the Treatment of Dissociative Disorders

RATIONALE: Trauma leads to dissociation. The dissociation is intended to buffer the emotional difficulty of dealing with the trauma. Some victims develop chronic anxiety, psychosomatic and/or dissociative symptoms. Patients suffering from disorders characterized by dissociative phenomena are highly hypnotizable. Many trauma victims may unknowingly be using their hypnotic capacities in order to remain unaware of the content of their traumatic memories while creating different types of psychopathology. If hypnotic-like states are spontaneously elicited during traumatic experiences, it is possible that the very entry into this same state will lead to the retrieval of memories and affects associated with the original trauma as predicted by the theory of state-dependent memory. If an uncontrolled use of their hypnotic

capacity is the source of their symptoms (e.g., flashbacks, alter personalities), appropriate use of hypnosis in the context of therapy can become a tool to access previously dissociated material.

Psychotherapy using hypnosis: The therapist's task is to provide a structured and safe environment, help patients organize painful memories, express them in ways that do not foster self destructive behaviors, encourage the development of mature defenses, restructure the ways patients think about themselves, and help patients develop an improved self-esteem and self-image. There is a risk of further re-traumatization in the continuous reliving of traumatic experience without adequate restructuring before new defenses are in place. Victims of traumatic events experience their symptoms as occurring unexpectedly and beyond their control. Hypnosis can be used to provide controlled access to the dissociated or repressed memories and help patients restructure their perceptions. When hypnosis is used correctly, it allows patients to discover the amount of control they have over these states of mind, which they experience as automatic, uncontrollable and unpredictable.

Hypnosis applied: Hypnotic retrieval involves the use of techniques that promote physical levels of relaxation and a sense of mental and emotional control. Recovery of traumatic memories should proceed at a pace patients can tolerate. There are a number of hypnotic techniques that can facilitate the process of recovery of traumatic memories while still allowing patients to feel in control. Hypnotic techniques include: relaxation techniques, projective techniques (e.g., split-screen technique), age regression, and the affect bridge.

The two major treatment goals are to bring into conscious awareness previously repressed traumatic memories and to develop a sense of congruence between the traumatic memories and the self. Treatment stages are **(6 C's)**: Confrontation, Condensation, Confession, Consolation, Concentration and Control.

Successful "working through" helps patients: understand the why and how of what happened, accept the past with a better sense of perspective, restructure their ideas of self and their current existence. Merely uncovering memories is not enough. Abreactive techniques, with or without the use of hypnosis are not enough. Exploring the trauma for its own sake has no therapeutic benefits.

Hypnosis and the law: When using hypnosis with physical-sexual trauma victims, consideration must be paid to the patients' ability to testify in court, the possibility of contamination, the phenomena of concreting and confabulation and the way the mind stores and processes memory. What patients remember, in the context of therapy, may seem true to the patient. That does not mean that it is true. But, the mere fact that hypnosis was used does not mean that the results are the product of suggestion or confabulation. Because it is almost impossible not to add some degree of contamination to any memory enhancement procedure, the practitioner should take the following precautions if hypnosis is used:

1. Obtain the patient's permission to consult with their attorney
2. Obtain an informed consent regarding hypnotic retrieval; explain to the subject and his/her attorney about the nature of hypnotically retrieved memories

3. Document any and all pre-hypnosis memories
4. Record ALL interactions electronically (videotape/audiotape)
5. Use an expert as a hypnosis consultant
6. Conduct the interview in a neutral tone; avoid leading or suggestive questions.
7. Carefully debrief the subject at the end of each session.

If memories of possible abuse are recovered, remind the patient about the nature of memory processing, fantasy and difficulties differentiating among them. Encourage "working through" the "memories" as you would do with any other therapy material, and do not encourage confrontation with the alleged perpetrator or to pursue legal action.

Eating Disorders

Eating Disorders

Kara Fitzpatrick, M.D.

Katherine Williams, M.D.

Case

Mrs. J. is a 40-year-old woman in her 16th week of pregnancy. Ever since marrying five years ago, Mrs. J and her husband had been trying to conceive. At first, physicians suspected that her husband's borderline sperm count was causing the infertility; however, as time went on, it became clearer to the physicians that Mrs. J. was intermittently anovulatory.

Mrs. J's physicians counseled her that her extremely demanding job as a CFO of a biotech firm may be contributing to functional amenorrhea and referred her to a relaxation class. When Mrs. J. became pregnant she was initially overjoyed; however she found the prospect of weight gain abhorrent, since she had always been very slender and considered herself already "too fat". Prior to pregnancy, she weighed 98 lbs., at five feet two inches in height. She exercised several hours per day in order to minimize gaining weight and spent what seemed, to her concerned husband, to be hours in front of the mirror examining her body.

At the first OB/GYN appointment when her physician noted that she had lost 5 pounds, he asked if she had suffered the common problem of first trimester nausea and vomiting and Mrs. J denied any problems. Her husband was stunned by her secretiveness, as he was certain he had heard her vomiting each day, up to several times per day.

Past psychiatric history: Major Depression – treated with an SSRI for 6 months, two years ago.

Medical History: Anemia

Medications: none

Habits: No EtOH or recreational drugs

Family History: Major Depression (maternal aunt and mother)

Physical Exam: BP: 100/70 sitting; 90/60 standing. P: 60 sitting; 83 standing.

Mental Status Exam: Neatly dressed, thin woman appears older than her stated age. Fair eye contact.

Mood: "Really good".

Affect: constricted and not congruent with mood. She denies vegetative symptoms of depression.

Speech: Normal rate and tone without pressure.

Thought Content: No delusions or hallucinations. She demonstrates preoccupation with weight and the amount that she will gain during pregnancy and how she will lose it afterwards. No suicidality or homicidality.

Thought Process: No looseness of associations, tangentiality or circumstantiality.

Insight and Judgment: Poor. She appears unconvinced that her weight loss, if it continues, is potentially harmful to the fetus and she is resistant to recommendations, including dietary, medical or psychiatric evaluations.

Questions:

What is the most likely diagnosis?

What are the personality variables that are important to clarify in eating disorder patients?

What is the significance of Mrs. J's history of Major Depression?

What are the acute and long-term medical complications involved in this disorder?

What would you recommend as treatment options to Mrs. J, her husband and her physician?

Introduction

Eating disorders include Anorexia Nervosa (AN), Bulimia Nervosa (BN), and Eating Disorder Not Otherwise Specified (NOS). Binge Eating is categorized under Eating Disorder NOS.

Eating disorders are becoming more prevalent amongst all cultures. To date, in this country, Caucasian women have been at greater risk for the development of eating disorders than Asian, Hispanic and African American women, but studies reveal that dieting and body weight concerns are infiltrating into all socioeconomic and racial groups.

Anorexia Nervosa

Diagnosis

Individuals with Anorexia Nervosa have a very rigid image of being overweight; no matter how thin they have become they believe they need to lose more weight, and they suffer from an intense fear of gaining weight. Diagnostic criteria require that the individual suffers from this distorted body image and that they have lost weight to $\leq$ 85% of their expected weight for height and body size (100 lbs. for 5 feet and 5 lbs. per inch after 5 feet is one commonly used body weight guideline). Amenorrhea must be present for at least 3 cycles to meet diagnostic criteria.

Diagnostic Criteria for Anorexia Nervosa
1. Refusal to maintain body weight or above a minimally normal weight for age and height.
2. Intense fear of gaining weight even though underweight.
3. Denial of the seriousness of the current low body weight, undue influence of body weight on self-evaluation.
4. Amenorrhea (at least three consecutive menstrual cycles).

There are two types of Anorexia Nervosa. The Binge Eating/Purging Type involves self-induced vomiting, and misuse of laxatives, diuretics or enemas (not to be mistaken with Bulimia Nervosa). The Restricting Type involves no binge-eating or purging behavior. It appears that there are differences in personality variables between these two types. Individuals with Binge Eating/Purging Type Anorexia tend to be more extroverted and may have trouble with impulse control. They may engage in kleptomania or sexual promiscuity. Restricting type anorexics lose weight by pure calorie restriction. They may be more rigid, perfectionistic, meticulous and socially withdrawn. They often are prone to obsessional thinking. It is important to ask about such personality variables when interviewing a patient you suspect has Anorexia Nervosa because these personality variables have prognostic and treatment significance.

It is important to note that anorexia nervosa is not synonymous with loss of appetite but with *control* of appetite. Many patients are preoccupied with food and spend hours cooking for friends or hoarding food.

Epidemiology

Anorexia Nervosa has a prevalence of between 0.5 and 1.0% among females. Anorexia is rare in men, although rates appear to be increasing, especially among gay males. Anorexia nervosa appears to be more common in women of higher socioeconomic status and more frequent in Caucasians.

Etiology

Psychological Theories: The etiology of eating disorders appears to be multifactorial. An increase in societal pressure to be thin appears to be correlated with increased rates of eating disorders in this country during the 20th century. For example, exposure to popular girls' magazines in adolescence has been correlated with higher rates of eating disorders. However, Michael Strober has argued that individual psychological factors/predispositions are critical in the development of anorexia and bulimia, since all women do not develop these disorders despite their exposure to a culture fixated on thinness. Strober hypothesizes that psychological risk factors for the development of eating disorders include problems with autonomous functioning, low self-esteem and interpersonal skills.

Regina Casper's studies of personality factors in eating disorders reveal that the traits of risk avoidance, compliance and emotional and cognitive restraint may lead to increased risk of developing an eating disorder. It is hypothesized that when faced with the conflicts of independence, assertiveness and sexual development, some individuals focus upon controlling food and their bodies in a misguided effort to control their environment and their own developmental tasks. Research suggests that early relationships which provide secure attachment, encourage independence and are not overly protective decrease the risk of developing an eating disorder, whereas sexual and

physical abuse and disrupted relationships increase the risk of developing an eating disorder.

Biological Theories: Anorexia nervosa appears to be genetically transmitted since studies show an increased risk of eating disorders in monozygotic twins compared to dizygotic twins. First-degree relatives of eating disorder patients appear to have an increased risk for both eating disorders and mood disorders. Recent studies have studies have identified a genetic linkage at chromosome 1 in anorexic patients. An increasing number of investigators regard anorexia as primarily a "biological" disorder with psychological sequelae rather than a " psychological" disorder with biological consequences.

Early theories of anorexia nervosa defined the illness as the result of hypothalamic-pituitary dysfunction. However, recent studies reveal that the endocrine abnormalities seen in anorexia (see below) are the *result* of starvation and they are not unique to eating disorders. Correction of weight corrects these abnormalities.

Course and Prognosis

The mean age of onset of anorexia nervosa is 17 years. Onset is rare after the age of 40. The course of the disease is quite variable. Thirty to 40% of individuals with anorexia nervosa recover fully in the first 10 years. However, there is a 10-15% mortality rate at 10 years and a 20% mortality rate at 20 years due to electrolyte imbalances, cardiac dysfunction and suicide.

How do symptoms become a disorder? An individual feels upset or out of control in some way. They choose to lose weight and then a cascade begins, where they begin to really invest in weight loss and refuse food. It is at that point where the body image becomes almost psychotic. Starvation appears to reinforce this obsessional, distorted thinking. The exact pathophysiology of this process is not yet known. Restoration of weight appears to improve these cognitive changes. Restless, excess energy accompanies

the starvation; this heightened activity level persists despite increasing emaciation and is one of the hallmarks of the disease.

Co-morbidity and Differential Diagnosis

50-75% of AN patients meet criteria for co-morbid major depression or dysthymia, and 10-25% will meet criteria for Obsessive Compulsive Disorder (OCD). The differential diagnosis for Anorexia Nervosa includes body dysmorphic disorder, obsessive compulsive disorder, delusional disorder, psychotic disorder and major depressive disorders. Body dysmorphic disorder is characterized by a real or imagined defect in appearance, such as a body part (i.e. large lips), rather than with weight in general. While medical illnesses such as cancer, HIV, ulcerative colitis and hyperthyroidism are all frequently associated with weight loss, these illnesses are differentiated from AN by a retention of a realistic sense of body weight and lack of desire to continue losing weight. Delusions regarding food may occur in psychotic disorders such as schizophrenia; however, these can be differentiated from AN by the associated presence of thought disorder (such as circumstantiality and tangentiality), hallucinations or associated non-food related delusions.

Physical Exam

The physical exam for an individual with anorexia will reveal emaciation, lanugo hair (very fine hair), peripheral edema and vital sign changes, including hypotension, bradycardia, and frequently hypothermia.

Medical Complications

Patients with anorexia nervosa have electrolyte imbalances, arrhythmias (including bradycardia and prolonged QT), leukopenia and mild normocytic normochromic anemia due to generalized bode marrow suppression, impaired renal function, dental problems, and osteoporosis and pathological fractures due to low estrogen, decreased calcium intake and increased cortisol secretion. It may be that the high level of energy expenditure is in some way helping to protect from even more bone

loss and fractures (related to evidence of weight bearing exercises preventing osteoporosis).

The hypothalamic-pituitary-gonadal axis is clearly affected in AN: LH, FSH and estradiol levels decrease and the individual enters a prepubertal hormonal state. Weight restoration leads to a "second puberty" in the individual and return to normal gonadotropin pulse secretion and gonadal function. Thyroid hormone changes include decreased T3 and low-normal T4. The hypothalamic-pituitary-adrenal axis is activated with hypercortisolemia, as seen in Major Depression.

Treatment

Refeeding and Behavioral Treatment Programs: The main treatment goal in AN is to correct nutritional problems and restore healthy eating patterns since the physical, cognitive and mood symptoms improve with weight restoration. This process is very difficult to implement because of the patient's persistent fear of gaining weight. Behavioral in-patient treatment programs are the treatment of choice for individuals <85% body weight with medical complications. Refeeding is accomplished by either parenteral nutrition or nutritional supplement diets such as Ensure. Weight gain must be gradual; the goal is approximately 1-3 lbs./week for in-patients. This can usually be achieved through a diet of 1000-1600kcal/day. Careful weights, intake and output and hemodynamic monitoring are done to prevent cardiac and renal damage due to fluid overload. Behaviorally oriented programs encourage patients to eat via incentives, such as visitors or passes off the ward.

Psychotherapy: Traditional *insight oriented* psychotherapy has not proved to be as efficacious in the treatment of AN as cognitive behavioral approaches. New treatments focus on evaluations of cognitions and fears around eating. Dysfunctional cognitions and problems with affect regulation are evaluated and treated. Family therapy appears to be clearly effective and one of the few empirically based treatments treatments for anorexia. However, family therapy has been studied primarily in adolescents with anorexia.

Psychopharmacologic treatment of AN has so far been relatively disappointing. Medications should never be used as monotherapy. Tricyclic antidepressants (TCAs) and SSRIs have been used for co-morbid depression. Cyproheptadine, a serotonin and histamine antagonist, has been shown to be useful for weight gain and increasing appetite in some nonbulimic AN patients. SSRIs have not been particularly effective in either the acute treatment or the prevention of relapse in anorexia but can help with co-morbid OCD or depression. Among the more promising groups of agents currently being evaluated are atypical antipsychotics such as olanzapine. Preliminary studies have suggested that olanzapine may help both weight gain and the agitation/anxiety associated with eating in anorexic patients.

Bulimia Nervosa

Diagnostic Criteria

A binge is defined as an all-consuming urge to eat large quantities of food -- in the range of 3000 - 6000 calories. There is a feeling of a complete lack of control and even dissociation, as individuals describe "being in a trance" and decreased awareness of what is going on around them. Another characteristics of a binge are that it occurs during a discrete period of time, usually less than 2 hours. Binge foods frequently include high fat & calorie foods such as ice creams and cakes.

Bulimia Nervosa involves recurrent episodes of binge eating with recurrent inappropriate compensatory behavior in order to prevent weight gain. The binge-eating and inappropriate compensatory behaviors must both occur, on average, at least twice a week for 3 months.

Diagnostic Criteria for Bulimia Nervosa
1. Recurrent episodes of binge eating. 2. Recurrent over-compensatory behavior in order to prevent weight gain, such as self-induced vomiting, misuse of laxatives, etc. 3. The binge eating and inappropriate compensatory behaviors both occur, on average at least twice at week for 3 months. 4. Self-evaluation is unduly influenced by body shape and weight. 5. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

As in AN, there are two subtypes of BN. Individuals with the purging subtype vomit or use enemas, laxatives, or other purgatives while non-purging subtypes use fasting or excessive exercise to compensate for the bingeing. Purging bulimics tend to demonstrate more self-injurious behaviors, including increased frequency of suicide attempts and substance abuse, have greater body image disturbance and more frequent histories of sexual abuse.

Epidemiology

Bulimia Nervosa is more common than AN, with a prevalence in females of 1 - 3%. Bulimic behavior may be as high as 20-40% on college campuses. BN tends to have its onset later in life than AN, usually in late adolescence or early adulthood.

Etiology

Psychological: The societal demands for thinness that are believed to contribute to etiology of AN, are considered important in the development of BN as well. Temperamental differences, such as the personality variables of restraint and impulse control, as well as the individual's innate natural appetite, may lead a patient to one form of eating disorder rather than another. Bulimic patients tend to be more extroverted,

impulsive and affectively labile than anorexic patients. Bulimic behavior is thought to be a means to regulate affect, and many patients report feelings of interpersonal and intrapsychic conflict escalate before a binge-purge cycle. Afterwards, they may have a sense of relief, calm and comfort, albeit mixed with lower self-esteem and guilt.

Biological: The efficacy of serotonin reuptake inhibitors in decreasing the frequency of bingeing and purging suggests that serotonin dysregulation plays a role in the etiology of the disease. The exact nature of this dysregulation remains to be understood.

Course and Prognosis

BN appears to be a chronic illness. Many patients relapse during periods of increased stress. Treatment, either psychopharmacologic or psychotherapeutic, is associated with significant rates of remission (>50% in most studies); however, many patients appear to relapse after treatment is withdrawn. Overall, BN has a better prognosis than AN.

Physical Exam

Patients with bulimia nervosa frequently show dental damage due to vomiting, enlarged salivary glands (particularly the parotid glands), calluses or scars on their hands. Electrolyte disturbances include hypokalemia, hypomagnesemia and hypercalcemia. Patients are at risk for cardiac arrhythmias because of the hypokalemia. Patients may suffer from hypotension and bradycardia and menstrual irregularities are not uncommon. Laxative abuse may be associated with bloody diarrhea.

Co-morbidity and Differential Diagnosis

BN can be differentiated from AN patients by their usually higher weight and lack of distortion regarding body image. Consumption of large amounts of food is common

in atypical depression, but these patients do not exhibit compensatory behaviors such as purging or excessive exercise.

75% patients with BN also meet criteria for major depression or dysthymia; anxiety disorders are also extremely common (30-40%) as is substance abuse. Personality disorders are frequently found in BN patients (50-75% in some studies); borderline personality disorder is exceedingly common.

Treatment

Psychological: Cognitive-behavioral therapy (CBT) is the psychotherapy treatment of choice for bulimia nervosa. CBT involves self monitoring of both diet and feelings and conflicts prior to binge-purge cycles and the episodes themselves in order to clarify eating patterns and affective triggers to the behavior. Dysfunctional cognitions regarding food and self-esteem are carefully evaluated and restructured. Several studies also support the role of interpersonal psychotherapy (IPT) for BN, suggesting that the behavior is a maladaptive means of resolving conflicts such as frustration over roles, anger, and communication problems. With CBT or IPT alone, between 50 and 90% of individuals experience an average of 70% reduction in bingeing frequency.

Pharmacotherapy: Fluoxetine in high doses (60 mg/day) has been shown to be useful in decreasing the frequency of bingeing and purging by 50-60% (vs. 10% control group); it now has FDA approval for use in BN. The efficacy of Fluoxetine in decreasing bingeing and purging is independent of the medications' effects on mood. Tricyclic antidepressants have also been shown to decrease binge-purge frequencies; however, they are associated with the risk of QT prolongation and patients with BN are already at risk for arrhythmias due to electrolyte imbalances associated with purging. Recent studies suggest that CBT and medication in combination may be more effective than either treatment alone.

When to hospitalize?

Most patients with BN can be treated on an outpatient basis. If someone is bingeing and purging several times a day, having electrolyte changes and syncope, and feeling very out of control subjectively, that is when you hospitalize.

Return to the Case

The clinical presentation is strongly suggestive for anorexia nervosa, which is characterized by an excessive fear of gaining weight that leads to weight loss to less than 85% of ideal body weight. Since Mrs. J is 5 feet 2 inches, her "expected" pre-pregnancy body weight would be 110 pounds (by one common standard suggesting that for women, ideal weight is 100 pounds at 5 feet and 5 pounds each additional inch). While low at 98 pounds, she did not meet the anorexia criteria for weight loss until she reached 93.5 pounds. She did already suffer from the amenorrhea, which is one diagnostic criterion; along with the belief that she was "too fat" although she was clearly very thin even before her weight loss. It is not clear from the history given whether Mrs. J had maintained her weight through restriction alone or through purging, and this information is needed in order to be sure of the diagnosis.

Review Questions

1. What is the difference between Anorexia Nervosa and Bulimia? Which eating disorder do you think Mrs. N is suffering from? How would you proceed in making the diagnosis?

2. What are the current treatments for Anorexia Nervosa and Bulimia? How successful are these treatments?

3. What are the medical complications of eating disorders?

4. What are the psychological risk factors for the development of eating disorders?

Anxiety Disorders

Anxiety Disorders

Elias Aboujaoude, M.D.

Christer Derksen

~~Katherine~~ E. Williams. ~~M.D.~~

Case

Mrs. N, a 28 year-old new mother was referred by her OB/GYN for "postpartum adjustment problems." At the first interview, Mrs. N. described an easy, uneventful pregnancy until the ninth month when she suffered from preeclampsia. She underwent an emergency C-section and delivered a healthy baby boy. Ever since the surgery, Mrs. N. had been "feeling anxious." While holding her child she felt an overwhelming sense of dread and "almost dropped him on the floor." Her heart raced, she felt like she might suffocate or pass out, her hands tingled and she decided at that moment she "must be going crazy." At the second interview, Mrs. N. admitted that she had also been having some "unusual thoughts." After much coaxing, she admitted that she had been having intrusive, brief thoughts of stabbing her baby. She was horrified by these thoughts, "didn't know where they came from" because she adored her new son. Terrified, she hid all the knives in her house. These thoughts seemed to be coming more frequently over the past few days and she had not admitted them to even her husband.

Introduction

Most likely, you have all experienced anxiety at some point in the past. It is a very common emotion that actually has many positive, adaptive aspects. Anxiety can warn us of danger or help us prepare in advance for things. Anxiety disorders are defined as anxiety that has taken on proportions beyond that of the general worries of the general population, interfere in daily functioning, and cause marked distress. In other words, the sense of fear in these disorders is quite different from "normal" fear in that it causes severe distress and disability and the source of the anxiety is frequently unknown, not recognized, or inadequate to account for the symptoms.

Anxiety disorders are common in the general population, and all physicians, no matter what specialty, can expect to see many patients with anxiety disorders which may impact their medical treatment. In fact, anxiety disorders often present with symptoms that mimic medical conditions, including substance abuse, excess caffeine intake, Vitamin B12 deficiency, hypothyroidism, hypoglycemia, and cardiac arrhythmias. Consequently, it is critical that all physicians be able to recognize and understand the etiology and treatment of these conditions.

The predominant physiologic manifestations of Anxiety Disorders include shakiness, palpitations, loss of perioral sensation, sweating, dizziness, dilated pupils, syncope, tingling extremities, GI disturbances, and increased urinary frequency.

The DSM IV lists the following anxiety disorders:

Adjustment Disorder with Anxiety

Panic Disorder with or without agoraphobia

Obsessive Compulsive Disorder (OCD)

Social Phobia

Specific Phobia

Posttraumatic Stress Disorder (PTSD)

Generalized Anxiety Disorder (GAD)

Acute Stress Disorder

In this class we will discuss in detail Panic Disorder, Obsessive-Compulsive Disorder (OCD), Post Traumatic Stress Disorder (PTSD) and Generalized Anxiety Disorder (GAD). Adjustment Disorder with Anxious Mood, Social and Specific Phobias are covered in the textbook.

Obsessive-Compulsive Disorder

Epidemiology: The Epidemiologic Catchment Area survey data suggest that the lifetime prevalence of OCD is between 2-3% in the general population; since so many people do not seek treatment for their symptoms because of embarrassment or lack of understanding, the prevalence may in fact be higher. The prevalence of OCD is similar in males and females.

Definition of Obsessions and Compulsions: An obsession is a recurrent, intrusive, ego-dystonic (i.e. the obsessions are both foreign and distressing) and distressing thought, image or impulse. Patients will report that the thought or image comes into their mind over and over and *seems odd to*

them. The thoughts are not confined to real-life worries or problems. Active efforts to suppress the thoughts are also key to the diagnosis. The most common obsession is one of contamination. Other common obsessions include pathologic doubt (i.e. "Did I shut the door? Did I shut off the water?), aggressive impulses, horrific impulses, bizarre sexual fixations, bodily functions, and a need for symmetry or order are also common. Compulsions are repetitive behaviors or mental acts that a person feels driven to perform in order to prevent or reduce the distress associated with the obsession. These may include stereotyped behaviors. Mental compulsions are an important new addition to the DSM-IV. They include special words, images or numbers recreated mentally to neutralize anxiety. Examples include special prayers repeated in a set manner, mental counting, mental list making and mental reviewing. In many cases, these are quiet acts that the patient will not reveal unless specifically asked. As with the obsession, these behaviors or acts are seen by the patient as strange or ego-dystonic. Checking, washing and counting are the most common compulsions. It is significant to note that these patients do have insight and view their thoughts and behaviors as being irrational.

DSM-IV Diagnostic Criteria for OCD

A. Either Obsessions or Compulsions (or both):

Obsessions are defined as

1. Recurrent and persistent thoughts, impulses or images that are experienced as intrusive and inappropriate, causing marked distress or anxiety.

2. The thoughts, impulses and images are not simply excessive worries about real-life problems.

3. The person attempts to ignore or suppress or neutralize (with other thoughts or actions) such thoughts, impulses or images

4. The person recognizes that the obsessional thoughts, impulses or images are a product of his/her own mind.

Compulsions are defined by:

1. Repetitive behaviors (washing, ordering, checking) or mental acts (praying, counting) that the person feels driven to perform in response to an obsession.

2. The behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event. However, these behaviors must not be connected in a realistic way with what they are designed to neutralize or are excessive.

B. At some point, the person has recognized that the obsessions or complusions are excessive or unreasonable.

C. The obsessions or compulsions cause marked distress or impairment.

D. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g. preoccupation with food in an Eating Disorder).

E. The disturbance is not due to a substance or general medical condition.

Diagnostic Criteria for OCD and Differential Diagnosis: As stated above the criteria for OCD include experiencing obsessions, compulsions, or both. Obsessions or compulsions are recognized as excessive and unreasonable by the patient (ego-dystonic). Obsessions are time consuming (occupy more than 1hr/day) and interfere with functioning. If there is a comorbid Axis I disorder diagnosed, the obsessions cannot be confined to that disorder and if they are, then that Axis I disorder takes precedence. For instance, ruminative

thinking about one's faults may be a predominant symptom during a major depressive episode or obsessionality regarding food a prominent part of an eating disorder.

The fact that these obsessions are experienced as *ego-dystonic* is a distinguishing diagnostic criteria from psychosis. In contrast to similar thoughts in psychosis, obsessions are considered to be part of the patient's own mind, one's own thought, and NOT controlled by forces outside one's own mind. Command hallucinations are not experienced and reality testing is normal. While the content of OCD patients' thoughts may at times appear psychotic, they should not demonstrate evidence of a thought disorder, such as circumstantiality, tangentiality or looseness of associations.

Eating disorder patients frequently feel that their food and body obsessions are appropriate and they deny distress about their low body weight and restricted diet; in contrast, OCD patients recognize that their obsessions are "intrusive and inappropriate" and efforts are made to suppress them.

It is important to distinguish obsessive-compulsive disorder from obsessive-compulsive personality disorder (OCPD) or traits. It is likely that many of the individuals who succeed in pre-medical and medical training show obsessive-compulsive traits, but these do not warrant a diagnosis of OCD. Personality disorders are diagnosed when traits are very inflexible, very durable over time and lead to disability in one's life. OCPD individuals tend to be very aloof, not necessarily engaging socially, excessively devoted to work and very scrupulous. For most of them, there is a real rigidity

around perfectionism. These individuals do not find their symptoms to be ego-dystonic and they do not try to suppress the obsessionality. On the contrary, they are often quite proud of their obsessions or compulsions.

In order to help identify the diagnosis of OCD, you should ask questions concerning the most common obsessions and compulsions (e.g. Do you wash/clean a lot? Do you check things excessively? Are you concerned with order? Do you have any significant doubts or fears? Do you have any thoughts that seem bizarre or troubling to you?). Remember, these patients have insight, and they see these behaviors as being strange or distressing, so they are apt to respond to the right questions.

Etiology: The etiology of OCD is currently unknown, but it has been found to be more frequent in disorders associated with lesions in the basal ganglia, specifically the caudate nucleus. Consequently, OCD is frequently seen in other diseases affecting the basal ganglia such as Parkinson's disease, Huntington's disease, Sydenham's chorea. Functional imaging studies, such as PET, reveal increased metabolism and blood flow in the caudate nuclei, orbitofrontal cortex and anterior cingulate cortex of OCD patients compared to controls. Therefore, it is likely that OCD involves lesions in the anterior brain (especially orbitofrontal cortex) and the basal ganglia.

While it has been consistently shown that medications that increase serotonin are associated with improvement in OCD symptoms, it is not yet clear whether serotonergic dysregulation is a cause or consequence of OCD. Multiple studies focusing upon whether OCD is the result of decreased

serotonin or abnormal serotonergic receptor response have failed to show consistent abnormalities.

Dopamine also appears to be involved in the pathophysiology of OCD in some patients. Animal and human studies show that dopaminergic medications such as bromocriptine and amphetamine are associated with stereotypic movements that resemble OCD behaviors. OCD is also highly correlated with Tourette's Disease, a neurological condition associated with multiple vocal and motor tics which are thought to result from dopaminergic dysfunction in the basal ganglia. (It is estimated that between 45-90% of Tourette's Syndrome patients also suffer from OCD).

Genetic studies of OCD suggest that it is a genetically heterogeneous disorder (no specific gene has yet been found); the precise mode of transmission is not yet known. Concordance rates in monozygotic twins are greater than in dizygotic twins (53%-86% vs. 22%-47%) and family members of probands are at increased risk of having the illness (metanalysis suggest that first degree relatives have a 5 times greater chance of having OCD than relatives of non-OCD patients).

***Course and Prognosis*:** OCD tends to occur early in life, typically around the age of 20. Males appear to have an earlier onset (age 6- 15 years) than females (20-29 years). In many OCD cases, onset appears to follow a stressful event. However, it has been difficult to pinpoint the instigating event in most cases. One-third of patients have onset in childhood.

Treatment for OCD is frequently delayed until 5 to 10 years after the onset of symptoms. This delay often occurs, because people are reluctant to report symptoms. Treatment for OCD is typically quite effective in reducing symptoms, although complete remission of the disorder is not as common (<20%) as in major depression or other anxiety disorders.

Treatment: It is now recognized that OCD is primarily a biologically mediated illness rather than one related to psychic conflict. Insight-oriented therapy is rarely efficacious. The most effective treatment is a combination of behavior therapy (i.e., exposure/response prevention in which patients are exposed to the anxiety causing situations in a controlled setting) and medications with potent serotonergic properties, such as the tricyclic antidepressant clomipramine and SSRIs. Paroxetine, fluoxetine, fluvoxamine and sertraline have all been approved by the FDA for treatment of OCD. Clomipramine, while efficacious in OCD, has the disadvantage of anticholinergic and antiadrenergic side effects and toxicity in overdose.

It appears that patients with OCD need higher doses of SSRIs than those usually used in the treatment of Major Depression and many patients require the maximum SSRI dose for improvement in their symptoms. It may take as long as 4-8 weeks for patients to respond to medication and maximal response may take even longer, from 12-20 weeks. Unfortunately, in most studies, only a small percentage of patients achieve complete remission (<20-30%); but it must be emphasized that treatment usually does lead to significant improvement in symptoms, and consequently significantly improved quality of life. Since OCD is usually a waxing and waning chronic illness, relapses are common if medication or therapy are withdrawn. For

refractory OCD, sometimes surgical approaches, e.g. cingulotomy, may be successful.

Co-morbidity: There is a high co-morbidity of Major Depression in individuals with OCD, around 30%. Fortunately, many of the same medications are effective for both OCD and Major Depression (SSRIs and clomipramine).

Co-morbidities with other anxiety disorders are common. Panic disorder, Simple Phobia, Social Phobia, and Eating Disorders commonly co-exist with OCD. It is important to also evaluate substance abuse in these individuals, since many patients may self-medicate their anxiety symptoms.

Panic Disorder

DSM-IV Diagnostic Criteria for Panic Attack

A panic attack is a *discrete period of intense fear or discomfort* during which one feels symptoms of hyperarousal. Diagnostic criteria require the presence of *4 or more of the following symptoms, developing abruptly* and *reaching a peak within 10 minutes:*

- palpitations
- sweating
- trembling
- sensations of shortness of breath, smothering or choking
- chest pain or discomfort
- nausea or abdominal distress
- feeling dizzy or faint
- derealization (i.e., things just don't seem real to you) or depersonalization (i.e., when you feel like you are not part of your own body).
- fear of losing control/going crazy
- fear of dying
- paresthesia (i.e., tingling in arms or legs)
- chills or hot flashes

Note that these are the symptoms of a panic attack, not a panic disorder, which is defined as recurrent, unexpected panic attacks followed by at least a month of additional attack or worry.

In addition, people experiencing panic attacks can have many of these symptoms at the same time, making their presentation in the Emergency

Department often confusing. Panic attacks are common in other psychiatric disorders, most frequently in major depressive episodes.

There are three distinct types of panic attack. Situationally bound panic attacks occur in response to a cue or trigger. Situationally predisposed panic attacks are those which are likely to occur in response to a cue or trigger. Unexpected panic attacks are those which come on immediately with no cue or trigger.

Diagnostic Criteria for Panic Disorder

Panic disorder is defined as recurrent, unexpected attacks, at least one of which is followed by one month or more of persistent concerns about having additional attacks, worry about the implications of the attack or its consequences, and significant changes in behavior related to the attacks (such as stop driving a car). Panic disorder can occur with or without agoraphobia. (Agoraphobia is when the sensation of panic is triggered by an open space where escape would be difficult or help unavailable) For diagnosis, panic attacks cannot be due to the effects of a substance, to a general medical condition, or to another mental disorder. There is a potential for these symptoms to develop into a phobia.

DSM-IV Diagnostic Criteria for Panic Disorder

A. Both (1) and (2)

1. Recurrent unexpected panic attacks

2. At least one of the attacks has been followed by 1 month or more of one or more of the following:

a. persistent concern/worries about having additional attacks

b. worry about the implications or consequences of the attack

B. The panic attacks are not due to a substance or general medical condition.

C. The panic attacks are not better accounted for by another mental disorder.

Epidemiology: Epidemiologic studies indicate that the lifetime prevalence of panic disorder is between 1-2%. Panic Disorders are more prevalent in females than males and with studies suggesting a 2:1 ratio.

Differential Diagnosis: Medical illnesses must always be considered in the differential diagnosis of panic disorder, and they must be ruled out before a psychiatric diagnosis can be made. Common metabolic and endocrine disorders that may present with panic symptoms include hypoglycemia, hyperthyroidism, pheochromocytomas, cardiac disease, and vestibular disorders (e.g., Meniere's disease). Many patients will think that they have a cardiac disorder and will present to the ER certain they are experiencing a heart attack. A 24-hour holster monitor can be used to evaluate whether the patient is experiencing any arrythmias, such as premature atrial tachycardia (PAT) or atrial fibrillation. Neurologic conditions which may present with panic attacks include cerebral vascular disease (TIAs) seizure disorders, especially partial complex seizures, multiple sclerosis, atypical migraines and vestibular disorders such as Meniere's Disease. A strong clinical rule of thumb is to suspect panic attacks when a patient presents with symptoms suggestive of cardiac, endocrine, or neurologic disorders and no discernible physical pathology.

Panic disorder patients can be distinguished from patients with generalized anxiety disorder (GAD) by their discrete episodes of anxiety,

usually lasting < 30 minutes. In contrast, GAD patients have a constant sense of anxiety but not in the paroxysmal manner of a panic attack. Panic disorder patients with agoraphobia may be difficult to distinguish from social phobia patients. Since panic attacks do occur in both conditions, the history is crucial in the differential diagnosis. The development of agoraphobia occurs after *unexpected* panic attacks. The initial fear is of a panic attack. In social phobia, the patient has a fear/discomfort with social situations and dreads their response to this contact.

Comorbidity: Individuals with affective disorders (e.g., Major Depression) may have panic disorder as well. It is very important to ask individuals with Major Depression whether they are having panic attacks, because comorbid panic disorder is associated with an increased risk of suicide attempts. Panic disorder is frequently comorbid with substance abuse, especially alcohol abuse since patients may try to use substances to calm down. Other frequently comorbid disorders include Somatization Disorder and personality disorders.

Etiology: The cause of panic disorders is currently unknown. Clinical experience and studies suggest that Panic Disorder patients may have a hypersensitive noradrenergic system which leads to excess norepinephrine and eventual downregulation of postsynaptic adrenergic receptors. Yohimbine is an alpha-2 adrenergic antagonist which increases the availability of norepinephrine. Individuals with Panic Disorder appear to be extremely sensitive to this increase in norepinephrine, and Yohimbine can precipitate panic attacks in vulnerable patients.

High levels of caffeine, sodium lactate injections and inhalation of carbon dioxide can also stimulate panic attacks, which have led to the theory that panic attacks are caused by CO2 hypersensitivity.

A corollary theory is that panic attacks represent a "false suffocation alarm;" anatomical areas that may be involved in this hypervigilance and response to somatic symptoms include the locus ceruleus and the limbic system.

Treatments

Medications

Effective pharmacologic treatments include tricyclic antidepressants, serotonin reuptake inhibitors, MAOIs and benzodiazepines.

In general, SSRI's are the first line pharmacotherapy. Benzodiazepines are not utilized as first line monotherapy since they are extremely addictive. However, they may be helpful in the first few weeks of treatment when combined with an antidepressant as a means of improving symptoms and function while the antidepressant begins to be effective. After approximately 2-6 weeks, the benzodiazepine is tapered and discontinued.

Sertraline and paroxetine have received FDA approval for the treatment of panic disorder; other SSRIs such as fluoxetine, fluvoxamine and citalopram have been found to be effective and may be used. When initiating SSRI treatment, small doses are used (i.e. 5 mg. Paxil) because

starting with higher doses may lead to increased frequency and intensity of panic attacks. Patients are titrated up to usual therapeutic doses over several weeks. Venlafaxine and Nefazodone offer promise as well, but larger studies are needed. In addition, anticonvulsants such as Gabapentin are being studied as treatments for Panic Disorder.

Panic disorder frequently recurs and treatment guidelines are the following: if a patient has had ≤ 2 episodes, then they should be treated for a minimum of one year, then tapered off the medication over the next several months. If panic attacks recur, or if the patient has had ≥ 2 episodes, then maintenance medication is indicated.

*Anxiety can be induced by the introduction of drugs. Many people will have significant anxiety, especially starting their course. In addition, because both the SSRIs and the Benzodiazepenes have their doses altered over the course of the treatment, many patients can develop anxiety reactions triggered by dosage changes. The best way to treat this symptom is to anticipate it and have an open and thorough dialogue with the patient.

MAOIs are rarely used due to the large number of dangerous side-effects. Buproprion and Trazodone have not been found effective in the treatment of panic disorder.

Psychotherapy

Cognitive-behavioral therapy is extremely effective in the treatment of panic disorder and appears to be as effective as medication. The goal of CBT is to help patients identify their catastrophic attributions to a non-

catastrophic stimulus and to teach them other cognitive appraisals as well as relaxation techniques. Attempts are made to identify the association between stimuli and physical manifestations and then reattribute their anxiety. In this manner, we strive to desensitize them to the anxiety-inducing stimuli.

To summarize, the treatment of Panic Disorder involves SSRIs, tapered Benzodiazepines, CBT and relaxation therapy.

Post-traumatic Stress Disorder (PTSD)

Diagnosis

PTSD is the only diagnosis in psychiatry that must have a *stressful event as a precipitating factor*. The event must be of catastrophic proportions, such as an actual or threatened death, assault, rape, serious injury, or watching another person undergo assault. Individuals with PTSD then continue to reexperience and avoid any stimuli associated with the event. Over time, psychic numbing develops and the individual becomes aloof and disengaged from the world. A state of persistent increased arousal is seen and significant impairment must be present. The duration of symptoms must be more than one month. If similar symptoms are seen for less than one month, then a diagnosis of Acute Stress Disorder may be made.

Reexperiencing the trauma can include having distressing dreams, intrusive recollections, dissociation and flashbacks, and intense distress when exposed to trauma-related stimuli. Many patients report that they dread going to sleep, because they will remember and dream about the trauma. Dissociation and flashbacks can be particularly traumatic for individuals, and they may lead to avoidance of activities or thoughts that remind them of the traumatic event. This "psychic numbing" is manifested as a decreased interest in activities, feelings of detachment from others, dissociation, social withdrawal, and a restricted range of affect. Many individuals experience a sense of foreshortened future, where they can not make meaningful plans for the future.

Increased arousal can be a very difficult symptom to cope with, both for the individual with PTSD and those who interact with him/her. Irritability is common. Many individuals are also hypervigilant and have an exaggerated startle response. Sleep disturbances and difficulty concentrating are also commonly seen in individuals with increased arousal.

In summary, the diagnosis of PTSD requires the exposure to a traumatic event and the above three symptom types: persistent reexperiencing of the event, persistent avoidance of stimuli related to the event and hyperarousal. These intrusive symptoms must last for more than one month for diagnosis (this time course distinguishes PTSD from Acute Stress Disorder, in which symptoms last 2 days to 4 weeks).

DSM-IV Diagnostic Criteria for PTSD

A. The person has been exposed to a traumatic event in which both of the following were present:

1. The person experienced, witnessed or was confronted with an event that involved actual or threatened death or serious injury to himself or others.

2. The person's response involved intense fear, helplessness or horror.

B. The traumatic event is persistently reexperienced in one or more of the following ways:

1. Recurrent and intrusive recollections of the event (images, thoughts, perceptions)

2. Recurrent or distressing dreams of the event.

3. Acting or feeling as if the traumatic event were recurring (sense of reliving, illusions, hallucinations, and dissociative flashbacks)

4. Psychological distress on exposure to internal or eternal cues that symbolize or resemble aspects of the event.

5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble aspects of the event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness as indicated by three or more of the following:

Efforts to avoid thoughts, feelings or conversations associated with the trauma

Efforts to avoid activities, places or people that arouse recollections of the trauma

Inability to recall important aspects of the trauma.

Markedly diminished interest or participation in activities

Feeling of detachment or estrangement from others

Restricted range of affect

Sense of foreshortened future

D. Persistent symptoms of increased arousal as indicated by two or more of the following: difficulty falling or staying asleep, irritability or outbursts of anger, difficulty concentrating, hypervigilance, exaggerated startle response.

E. Duration of disturbance is more than 1 month.

F. Clinically significant distress or impairment in social, occupational or other important areas of functioning.

Epidemiology: PTSD is more common in women than in men; lifetime prevalence rates for women are 12.5% vs. 6.2% for men.

Etiology and Risk Factors for Development of PTSD: The etiology of PTSD is currently unknown. Emerging evidence from basic animal studies

suggest that serotonergic pathways are involved in avoidance behavior; SSRI treatment studies further support the role of serotonin dysregulation in PTSD. Neuroanatomical areas involved in PTSD include the amygdala, which appears to be important in fear conditioning and the hippocampus, which is important for learning and memory. The anterior cingulate cortex appears to be important for integrating cognitive, affective and sensory function, and pathology in this area may lead to exaggerated response to potential threats. In summary, from functional and neuroimaging research, a model of PTSD emerges in which the amygdala is hyperresponsive, the hippocampus and anterior cingulate cortex which would normally modulate the fear response are hypofunctional.

The state of the individual at the time of the trauma affects the risk of developing PTSD. Individuals with preexisting anxiety or depression or with a family history of anxiety disorder or antisocial behavior are more likely to develop PTSD. It has been suggested that early childhood separation from parents may predispose a person to the development of PTSD. The nature and extent of the trauma appear to influence the likelihood of PTSD development, although it is not necessarily causative. The availability of social support after the trauma may also affect the risk of developing PTSD; consequently, many disaster relief services now offer acute counseling to victims.

The cortisol response of individuals with PTSD appears to be altered. In normal individuals during times of acute stress, cortisol is elevated. However, in individuals with PTSD, cortisol levels do not rise during stress. It is almost as if they are adapted to this level of stress by the ability to be

very responsive to small levels of cortisol. This low cortisol level in response to stress may be useful as a biologic marker of a predisposition to PTSD.

Differential Diagnosis and Comorbidity: The differential diagnosis for PTSD should include other anxiety disorders, such as Panic Disorder, OCD and Adjustment Disorder, with anxious mood. PTSD can be distinguished from schizophrenia by preservation of reality testing and lack of formal thought disorder. It is important to distinguish the symptoms of intrusive recollections seen in PTSD, from thought blocking seen in states of psychosis. Thought blocking occurs in psychotic individuals when they experience intrusive voices or hallucinations. They may be in the middle of a conversation with you and just stop talking and appear as if they are attending to something else. The intrusive recollections of PTSD may give a similar appearance. PTSD may be difficult to differentiate from dissociative disorders, but dissociative disorder patients usually do not have the hyperarousal and avoidance of stimuli. Substance abuse should be considered in the differential diagnosis; frequently patients are comorbid for substance abuse. There is a high co-morbidity of PTSD and affective disorders and somatization disorder. In fact, the prognosis of PTSD is based on the presence of comorbid disorders such as anxiety disorder or major depression.

Treatment

Treatment is typically symptom specific and includes psychotherapy and medication. SSRIs are usually first line treatment for PTSD since they

have been found to be helpful in decreasing all three symptom clusters of PTSD--hyperarousal, persistent reexperiencing of the event/intrusion and persistent avoidance of associated stimuli/numbing. Sertraline has recently received FDA approval for treatment of PTSD.

If SSRIs are ineffective, then switching to nefazodone or venlafaxine is indicated.

TCAs and MAOIs have been evaluated in the treatment of PTSD in a limited number of double blind placebo controlled trials, and both have been found to be superior to placebo in decreasing symptoms of hyperarousal. For individuals with hyperarousal and anxiety, clonidine (an alpha-blocker) or propranolol (a beta-blocker) are sometimes prescribed.

If sleep disturbance is prominent, then trazodone or tricyclic antidepressants are commonly prescribed. The use of benzodiazepine is avoided due to the high risk of abuse in this population. If psychotic symptoms are prominent, then antipsychotics are indicated. Mood stabilizers should be used for individuals with extreme anger or irritability.

For individuals with hyperarousal and anxiety, clonidine (an alpha-blocker) or propranolol (a beta-blocker) are commonly prescribed. TCAs and MAOIs have been evaluated in the treatment of PTSD in a limited number of double blind placebo controlled trials, and both have been found to be superior to placebo in decreasing symptoms of hyperarousal.

Effective psychotherapy for PTSD includes cognitive behavior therapy, exposure therapy and anxiety management. The choice of these therapies depends upon the predominant PTSD symptoms. For instance, intrusive thoughts and flashbacks are best treated by exposure therapy, while hyperarousal may be improved by anxiety management, including relaxation training and breathing retraining. Psychosocial support is critical component of care.

To summarize, the treatment for PTSD involves primarily SSRIs and symptom-specific psychotherapy. Approximately 50% of PTSD patients recover in 3 months, while 10% remain the same or worsen.

Generalized Anxiety Disorder (GAD)

Diagnosis: What Makes Worries Pathological?

When anxiety occurs more days than not, lasts for more than six months, and causes marked distress, the diagnosis of generalized anxiety disorder should be considered. The diagnosis of GAD can be made if the above criteria are met and the person finds it very difficult to control the worry and at least three of the following symptoms are present: irritability, sleep disturbance, restlessness, difficulty concentrating, fatigue, muscle tension. GAD is a common psychiatric disorder; 1 year prevalence rates in community samples are approximately 3% and lifetime prevalence 5%; however, since many patients do not seek treatment, the prevalence of the disorder may be higher.

DSM-IV Diagnostic Criteria for Generalized Anxiety Disorder

A. Excessive anxiety and worry (apprehensive expectation) occurring more days than not for at least 6 months, about a number of events or activities.
B. Difficulty in controlling the worry.
C. The anxiety and worry are associated with three of the following symptoms: restlessness (feeling keyed up or on edge), being easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, sleep disturbance.
D. The focus and worry is not confined to features of an Axis I disorder.
E. Clinically significant distress or impairment in social, occupational or other areas of function.

F. The disturbance is not due to the effects of a subsatnce or a general medical disorder and does not occur exclusively during a Mood Disorder, a Psychotic Disorder, or a Pervasive Developmental Disorder.

Etiology and Treatment

The cause of GAD is unknown. Psychoanalytic and psychodynamic theories conceptualize generalized anxiety as a signal from the unconscious that conflicts exist related to drives and impulses. Anxiety is believed to be caused by the ego's repression of these conflicts and psychodynamic psychotherapy attempts to clarify and work through these unconscious conflicts. Such therapy may improve anxiety in some patients, but large, controlled outcome studies are lacking. Behavioral treatment, specifically relaxation training appears to be helpful in GAD.

Biological theories of GAD focus upon the serotonin, norepinephrine and y-aminobutyric acid (GABA) neurotransmitter systems. First line psychopharmacologic treatments for GAD include buspirone and venlafaxine. Preliminary studies suggest that the nefazodone and mirtazapine are also effective in GAD. Benzodiazepines are effective in the disorder, but they have the potential for tolerance and abuse.

The best treatment for GAD is therefore a combination of drugs such as SSRIs and psychosocial intervention (psychotherapy, CBT, etc.)

A Brief Summary of Anxiety Disorders

Anxiety Disorder	Characteristic Features	Treatment
OCD	Obsessions (thoughts) and Compulsions (behavior) that are: -Ego-dystonic -Time Consuming -Interfere with functioning	-Cognitive Behavior Therapy -Clomipramine (TCA) -SSRIs
Panic Disorder	Recurrent, unexpected, discrete "attacks" of intense fear or discomfort	-SSRIs -Tapered benzodiazepines -CBT -Relaxation Therapy
PTSD	-Flashbacks -Avoidance of stimuli related to the event -Hyperarousal	-SSRIs -Symptom-specific psychotherapy
GAD	-Difficult to control worry -Occurs more days than not	-SSRIs -CBT -Psychotherapy

Review Questions

1. What is your psychiatric differential diagnosis for Mrs. N's symptoms. What medical conditions must be considered?
2. What are the pharmacologic treatments for the disorders listed above?
3. What are the three "symptom clusters" in PTSD?
4. How can PTSD be differentiated from GAD? Psychosis? Substance Abuse?

Alcohol and Substance Abuse Disorders

Substance Related Disorders

David L. Smith, M.D.

Alcohol Use Disorders

Definitions

Approximately 90% of American adults use at least some alcohol; when does use become misuse? There are several different words used to describe misuse of alcohol. **Alcoholism** is a general term (not a DSM-IV diagnosis) that refers to the use of alcohol in a detrimental way.

Alcohol abuse is a formal diagnosis that indicates serious *negative consequences* from inappropriate use of alcohol. These consequences can be in impaired social, family, or occupational functioning, in legal trouble stemming from alcohol use, and in the use of alcohol in dangerous situations (such as when driving).

Alcohol dependence is also a formal diagnosis and refers to the *physiologic state* of being "hooked" on alcohol and the *negative behaviors* that result. Characteristic symptoms are tolerance, withdrawal, escalating amounts of use, unsuccessful efforts to cut down use, a great deal of time spent on obtaining alcohol, a reduction in other important activities in favor of increased time spent drinking, and continued use despite medical consequences (like liver cirrhosis). If a patient meets the criteria for both dependence and abuse, then only dependence is diagnosed.

Note that it is not the actual number of drinks that is important; rather, it is the development of impaired functioning and negative consequences that defines abuse and dependence.

Epidemiology

Alcoholism causes significant medical and economic costs. An estimated 85 billion dollars a year is lost in health care costs and reduced productivity. Alcohol is thought to play a role in 20-50% of all hospital admissions, and even more so in the ER and on surgical services because of alcohol's association with motor vehicle accidents and violent trauma.

There is a 5-7% incidence and a 13% lifetime prevalence of abuse or dependence for Americans. The disease affects males 2 to 3 times as often as females, and has a high comorbidity with other psychiatric illnesses (like depression). Active alcohol use is a significant risk factor for suicide in at-risk populations and is involved in over 50% of attempted suicides.

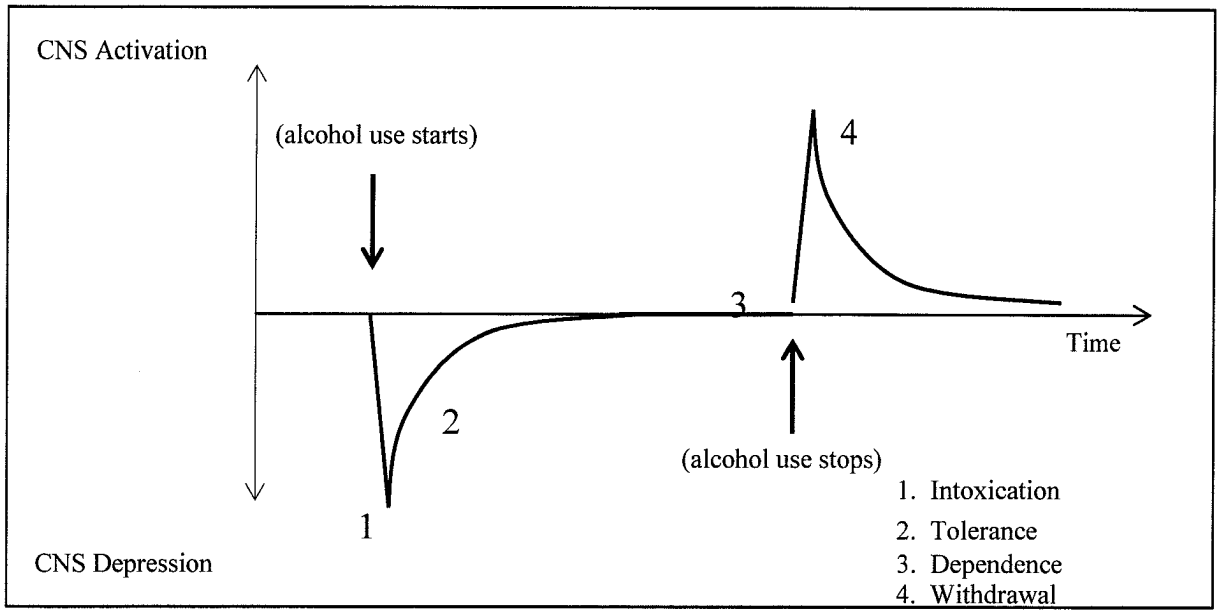
Etiology

Alcoholism likely has a multi-factorial basis, with both genetic and environmental factors playing roles. The evidence for a genetic component is most clearly established for male-to-male transmission of alcoholism (i.e., from father to son).

Acute homeostatic effects of alcohol

The brain is a homeostatic organ; it invokes compensatory mechanisms when faced with consistent external pressure in order to return to its natural "set-point." Alcohol is a CNS depressant – probably by enhancing the activity of GABA (an *inhibitory* neurotransmitter) in the brain – and leads to predictable homeostatic changes in brain functioning as detailed below.

Alcohol and CNS Homeostasis



Acute ingestion of excessive alcohol (in someone not already tolerant to its effects) leads to a syndrome called **alcohol intoxication**. The symptoms of intoxication – behavioral disinhibition (inappropriate sexual or aggressive behavior, mood lability, impaired judgment) and neurological dysfunction (slurred speech, incoordination, unsteady gait, nystagmus,

stupor or even coma) – are due to short-term CNS depressant effects of alcohol. Alcohol intoxication is rarely life threatening in and of itself because most people are incapable of continuing to drink to the point of seriously depressing their respiratory drive. However, intoxication can prove fatal if other people force the drinker to continue when already stuporous (such as during hazing rituals), if alcohol is combined with other CNS depressants, or if an intoxicated person engages in activities that require attention and coordination (like driving).

Tolerance results when a person continues to drink over time and can manifest in two ways. If the same amount of alcohol is ingested every day, the acute effects of alcohol gradually diminish (the CNS depressant effects are less and less pronounced as time goes on). The other way tolerance is manifest is when a person consistently escalates the amount of alcohol consumed each day in order to achieve the same effect.

Physiologic dependence refers to the condition of having developed enough tolerance to alcohol to have withdrawal symptoms upon discontinuation of use. At this point the brain's compensatory mechanisms have reached their full potency; the brain is "pushing" hard enough against the depressant effects of alcohol to bring its level of arousal back to baseline. If the alcohol is suddenly withdrawn, then these compensatory mechanisms raise the level of CNS activity to abnormally high levels.

Alcohol withdrawal is the syndrome that results from this CNS hyperarousal; the symptoms can include autonomic hyperactivity (tachycardia, hypertension, sweating), tremor, insomnia, nausea, transient

hallucinations or illusions, agitation, anxiety, and seizures. Note that these symptoms are essentially the opposite of the symptoms of intoxication. An uncommon, but very dangerous, type of alcohol withdrawal is called *delirium tremens*, which can occur 3 to 10 days after the cessation of drinking. It is characterized by hyperarousal and by autonomic instability, which are manifestations of severe dysregulation in the brain. Delirium tremens is associated with a high mortality rate even if treated; the instability of vascular tone can lead to organ damage, including kidney failure. As future physicians, it will be important for you to remember that the hospital is a common setting for alcoholic withdrawal. An alcoholic patient may be admitted to the hospital without health care professionals becoming aware of the addiction until the onset of withdrawal symptoms such as the aforementioned delirium tremons, which can be fatal! This scenario it especially common on surgical floors. Social histories *are* important!

Treatment of alcohol withdrawal

The goal in treating alcohol withdrawal is to prevent the acute peak of CNS activation that occurs with cessation of use. The treatment of choice is to prescribe *benzodiazepines*, which are CNS depressants and enhance the response of the chloride channel to GABA. Benzodiazepines help with withdrawal by replacing the inhibitory effects of alcohol; they are then tapered over time, which allows the brain to downregulate its compensatory mechanisms in a gradual and much less dangerous way.

Nutritional problems associated with alcoholism

Alcoholics tend to replace food with alcohol for their daily calories and thus can become deficient in several vitamins. *Thiamine* is an important factor in neuronal metabolism, particularly in limbic structures like the mammilary bodies. **Wernicke's encephalopathy** is a reversible condition that results from thiamine deficiency-induced injury to these structures; the classic symptom triad is confusion, ataxia, and ophthalmoplegia (gaze paralysis). If the sensitive limbic structure are severely damaged or die then Wernicke's progresses to a condition called **Korsakoff's syndrome**, which is a state of profound amnesia and is usually irreversible. Patients with Korsakoff's syndrome characteristically exhibit *confabulation*; they invent or make up information that they cannot remember.

Thiamine metabolism is dependent on the availability of glucose in the brain; giving a bolus of glucose or dextrose to a patient with Wernicke's can precipitate Korsakoff's syndrome acutely. *Always give thiamine to all alcoholic patients in the hospital, and always give thiamine before glucose or dextrose to any stuporous or comatose patient.*

Other common nutritional abnormalities in chronic alcoholism are folate and vitamin B12 deficiencies, which can lead to hematological and neurological dysfunction.

Other consequences of chronic alcoholism

Chronic alcohol use can affect almost every organ system, including the liver, pancreas, GI tract, cardiovascular system, brain, and bone marrow.

Outpatient treatment

Alcoholism is best treated with a multi-pronged approach. The mainstay of treatment is *psychosocial* intervention, such as involvement in *Alcoholics Anonymous (AA).* AA is an independent patient-founded organization, whose agenda is to get alcoholics to stop drinking and to stay abstinent through a "twelve step" model (AA 12 steps can be found at the end of this chapter; there are also alternative self-help organizations without a religious basis). More formal alcohol treatment programs involve group and individual psychotherapy; the best-studied type of therapy for addiction is behavioral therapy.

Pharmacological interventions can be adjuncts to the psychosocial treatments. Disulfiram (Antabuse) interferes with the metabolism of alcohol metabolites and causes nausea, headaches; it is thus creates immediate negative consequences to drinking. Patients must be willing to take disulfiram for it to be effective! The other relatively new agent that is useful for alcoholism is naltrexone (Revia), which is an opiate antagonist. Opiate receptors are involved with the reward system in the brain; an opiate antagonist can diminish the pleasurable effects of alcohol and also appears capable of reducing cravings.

Assessing for alcoholism

My three rules for the assessment of alcoholism are "Ask, Assume, and Overestimate." **Ask** everybody! Make screening questions for alcoholism part of your routine medical evaluation. If you only ask some people, you will miss a lot of people with alcohol problems. **Assume** that every patient drinks, unless they actively deny it. Instead of asking, "Do you drink alcohol?" ask, "How much alcohol do you drink in an average week?" Lastly, **overestimate** patients' use so that they must actively contradict you if you overstate their use. For example, if a patient tells me that he drinks a six pack a day, I often pretend not to hear accurately and say "So, you drink two six packs a day?" If he says, "No, just one six pack a day," then I find that information more believable.

A useful screening tool for alcoholism is to ask the four "CAGE" questions:

> **C**: Have you ever felt the need to **C**ut down on drinking?
>
> **A**: Have you ever felt **A**nnoyed by criticisms of your drinking?
>
> **G**: Have you ever had **G**uilty feelings about drinking?
>
> **E**: Have you ever taken a morning **E**ye-opener?

Other Drugs of Abuse

We will cover the other drugs of abuse in brief. The criteria for abuse and dependence are the same for all drugs of abuse.

Drugs of abuse can be organized based on the receptor systems that they affect. This information helps in knowing what to expect from each agent's intoxication and withdrawal syndromes; drugs within a class tend to be very similar to each other.

Brief list of Drugs of Abuse and their profiles…

Class of Agent	**Examples**	**Receptor**	**Intoxicating Effects**	**Withdrawal Symptoms**
CNS Depressants	Alcohol Benzodiazepines Barbiturates	GABA (Chloride Channel)	CNS depression, disinhibition, anxiolysis	Autonomic hyperactivity, tremor, insomnia…
Stimulants	Cocaine Amphetamine	Dopamine	Euphoria; elevated CNS activity	Seizures and strokes, but intoxication is more dangerous
Opiods	Heroine Morphine	Opiod receptors	Analgesia Euphoria	Insomnia, pain Not life threatening
Hallucinogens	PCP LSD	NMDA (PCP) 5-HT (LSD)	Transient psychosis Perceptual changes	Minor

CNS Depressants include alcohol, benzodiazepines, and barbiturates. They all depress neuronal activity by increasing the entrance of chloride ions through the chloride channel (the first two by increasing GABA activity). All CNS depressants have the same intoxication and withdrawal syndromes

(see alcohol section above for details). Barbiturates are the most dangerous agents in the class because the way they affect the chloride channel is not dependent on the availability of GABA; they are lethal in overdose and cause particularly severe withdrawal states.

Stimulants include amphetamines and cocaine. They both affect the dopamine system in the brain. Stimulant intoxication is characterized by euphoria and elevated CNS activity (agitation, tremor, tachycardia, and insomnia). Stimulant intoxication is much more dangerous than withdrawal; seizures and hypertension-induced strokes can occur. Stimulant withdrawal is not life-threatening and is characterized by dysphoria and hypersomnia.

Both endogenous and synthetic **Opiates**, naturally enough, affect opiate receptors. Opiate intoxication involves euphoria, analgesia (absence of pain), constipation (due to activation of opiate receptors in the gut), and respiratory suppression. Opiates are rarely lethal in overdose, except for overdoses of IV heroin. Opiate withdrawal is characterized by insomnia, pain, diarrhea, and autonomic hyperarousal, and generally is not life threatening.

Hallucinogens include LSD (which interacts with the serotonin (5-HT) system), and PCP (which is an NMDA receptor antagonist). Intoxication with hallucinogens leads to temporary psychotic symptoms, including perceptual abnormalities. Hallucinogens do not cause physiologic dependency and as a consequence there is little in the way of a withdrawal syndrome.

The AA 12 Steps:

1. We admitted we were powerless over alcohol—that our lives had become unmanageable.

2. Came to believe that a Power greater than ourselves could restore us to sanity.

3. Made a decision to turn our will and our lives over to the care of God as we understood Him.

4. Made a searching and fearless moral inventory of ourselves.

5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.

6. Were entirely ready to have God remove all these defects of character.

7. Humbly asked Him to remove our shortcomings.

8. Made a list of all persons we had harmed, and became willing to make amends to them all.

9. Made direct amends to such people wherever possible, except when to do so would injure them or others.

10. Continued to take personal inventory and when we were wrong promptly admitted it.

11. Sought through prayer and meditation to improve our conscious contact with God, as we understood Him, praying only for knowledge of His will for us and the power to carry that out.

12. Having had a spiritual awakening as the result of these Steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs.

Study Guides for POM Psychiatry

By Bernard Chang, former psychiatry TA

Schizophrenia

Overview

- A disorder marked by severe disturbances in cognition and sensory processing.
- Failure in reality testing with possible deficits in perception/emotion/communication

Epidemiology:

- Prevalence is approximately 1% worldwide; no gender differences
- Onset usually occurs in late adolescence; onset of schizophrenia is later in women.

Signs/Symptoms:

- Positive symptoms: paranoid delusions (ideas of reference, persecution, and grandeur), sensory hallucinations (mostly auditory, then visual and somatosensory)
- Negative symptoms: anhedonia, social withdrawal, alogia, avolition.
- Thought disorder symptoms: loosening of associations, thought alienation, poverty of thought.

Pathophysiology:

- Genetics: Genetic risk: 50% concordance rate among MZ twins; 10% risk among first-degree relatives; biological children of schizophrenics who are adopted into non-schizophrenic households still have an elevated risk of developing schizophrenia.
- Other risk factors: 2nd trimester influenza, born in winter months.
- Neuroimaging/Gross: increased ventricular size, decreased cortical volume, hypoactive dorsolateral prefrontal cortex.
- Neuropath/Histo: widespread gliosis, synaptic pruning (loss of dendrites but not cell bodies)
- Etiological Models/Factors
- (1) Dopamine hypothesis: schizophrenia associated with overactive dopamine activity in the frontal lobe leading to disturbances in the dorsolateral prefrontal cortex.
- (2) Glutamate hypothesis: schizophrenia associated with hypoactive glutamate activity
- (3) Expressed emotion: intense criticism, hostility and intrusiveness overexpressed in a family system; associated with increased relapse

Treatment/Clinical Course:

- Atypical (ie olanzapine) and typical (ie haloperidol) antipsychotics; supplemented with supportive psychotherapy, family therapy, and education.
- About 20-30% of schizophrenics termed "high-functioning" (i.e., live independently and have jobs and families).
- Over 50% of patients have courses marked by repeated hospitalizations, exacerbations of symptoms, and episodes of suicidality.

HY factoids

- 20-70% of persons with schizophrenia have comorbid substance abuse problem.
- 10% risk of suicide
- Early onset of illness, FH of schizophrenia, and prominent negative symptoms suggestive of poor prognosis

Unipolar Major Depression

Overview

• Mood disorder marked by decreased positive mood along with numerous psychomotor and somatic symptoms (SIGECAPS).

Epidemiology:

• Lifetime prevalence of depression in the U.S. is 17%; 26% for women, and 12% for men.
• 2x more common in women
• The mean age for a first episode is in the thirties.
• Risk factors: lower socio-economic status, recent immigration status, and being married (for women only; for men marriage is a protective factors)

Signs/Symptoms:

• Characterized by at least two weeks of either depressed mood (most of the day, nearly every day) or anhedonia, a markedly diminished interest or pleasure in daily activities, plus at least four additional symptoms such as significant weight loss/gain, fatigue, impaired concentration or feelings of worthlessness or guilt (See DSM-IV).

Pathophysiology:

• Genetics: 4x higher risk for individuals with 1st degree relative with depression; concordance for MZ twins is about 50%
• Neuroimaging/Gross: hypoactive dorsolateral prefrontal cortex,
• Sleep Abnormalities: shorter/decreased REM latency
• Etiological Models/Factors :
• (1) Biogenic Amine Hypothesis: depression associated with deficiency of amines, like serotonin and norepinephrine (site of action for many anti-depressants)
• (2) Hypothalamic Pituitary Axis abnormalities: elevation in cortisol levels often found in psychotic depression.
• See elevated rates of depression in Cushing's Disease

Treatment/Clinical Course:

• SSRIs (Setraline, Fluoxetine), TCA, Heterocyclics, Cognitive Behavioral Therapy

HY factoids

• Some disorders often associated with depression: hypothyroidism, SLE, corticosteroid use, fibromyalgia.

Bipolar-Affective Disorder

Overview

• Mood disorder marked by alternating periods of major depression and mania.

Epidemiology:

• Lifetime prevalence in United States is 1-1.6%; 0.8% having bipolar affective I, 0.5% having bipolar affective II.
• Average age of onset is adolescence/ young adulthood

Signs/Symptoms:

• There is a spectrum of bipolar affective related disorders including: bipolar I (BPI), bipolar II (BPII), cyclothymia (oscillating high and low moods), and major depression. BPI also is referred to as classic manic-depression, characterized by distinct episodes of major depression contrasting with episodes of mania
• BPII is a milder disorder consisting of depression alternating with periods of hypomania. Hypomania may be thought of as a less severe form of mania that does not include psychotic symptoms or lead to major impairment of social or occupational function.

Pathophysiology:

• Genetics: 1st degree relatives of bipolar affective patients have 7x increase risk of developing bipolar disorder compared to general population
• Etiological Models/Factors
• (1) Assumed to be interaction between biological and environmental. Imaging work has found differences in hippocampus and subgenual prefrontal cortex
• (2) "kindling" theory: people who are genetically predisposed toward bipolar disorder experience a series of stressful events, each of which lowers the threshold at which mood changes occur. Eventually, the mood episode itself is sufficient to trigger recurring difficulties

Treatment/Clinical Course:

• Lithium, Atypical antipsychotics, lamotrigine, carbamazepine. Antidepressants may be used in combination with mood stabilizers during the depressed phase. However, antidepressant can precipitate mania or more rapid cycling.

HY factoids

• People with bipolar disorder are generally more in the depressed phase of their illness than the manic portion.

Obsessive Compulsive Disorder

Overview

• Anxiety disorder marked by **obsessions** (recurrent, intrusive, ego-dystonic (i.e. the obsessions are both foreign and distressing) and distressing thought, image or impulse.) or **compulsions** (repetitive behaviors or mental acts that a person feels driven to perform in order to prevent or reduce the distress associated with the obsession)

Epidemiology:

• Prevalence is approximately 2-3%.

Signs/Symptoms:

• Presence of either obsessions or compulsions leading to marked distress/impairment.

Pathophysiology:

• Genetics: Concordance rates among MZ twins: 53%-86%
• Increased risk of OCD in biological offspring of individuals with OCD although seems to be elevated non-specific risk to increased mental illness
• Neuroimaging/Gross: dysfunctions in basal ganglia (caudate), cingulate gyrus, and orbitofrontal cortex
• Etiological Models/Factors
• (1) Basal Ganglia dysfunction: see OCD symptoms often in diseases such as Parkinson's, Huntington's, and Sydenham's chorea;
• (2) Dopamine dysfunction also associated with OCD; OCD correlated with Tourette's; bromocriptine and amantadine have noted OCD side effects.

Treatment/Clinical Course:

• Exposure and Response therapy
• SSRIs, Clomipramine

HY factoids

• IQ of OCD patients as a group is higher compared to the normal population.

Panic Disorder

Overview

• Anxiety disorder characterized by recurrent, unexpected attacks and at least one panic attack followed by 1 month or more of persistent concerns about having additional attacks, worry about the implications of the attack or its consequences, and significant changes in behavior related to the attacks
• Panic disorder can occur with or without agoraphobia

Epidemiology:

• Lifetime prevalence is between 1-2%.
• More prevalent in females than males and with studies suggesting a 2:1 ratio.

Signs/Symptoms:

• A panic attack is defined as a discrete period of intense fear or discomfort during which one feels symptoms of hyperarousal. Possible associated signs/symptoms include, palpitations, sweating, trembling, sensations of shortness of breath, smothering or choking, chest pain or discomfort nausea or abdominal distress

Pathophysiology:

• Etiological Models/Factors
• (1) Hyperactive noradrenergic hypothesis: PD patients may have a hypersensitive noradrenergic system which leads to excess norepinephrine and eventual downregulation of postsynaptic adrenergic receptors.
• Evidence: Yohimbine (an alpha-2 adrenergic antagonist), increases the availability of norepinephrine; Yohimbine can precipitate panic attacks in vulnerable patients.
• (2) CO2 hypersensitivty: PD patients may have an increased sensitivity to CO2; mild/moderate levels of CO2 may "convince" body that the CO2 levels are too high, precipitating hyperventilation and associated symptoms.
• Evidence: High levels of caffeine, sodium lactate injections and inhalation of carbon dioxide can also stimulate panic attacks
• A related theory is that panic attacks represent a "false suffocation alarm" in which the body falsely believes that it is hypoxic and induces hyperventilation; anatomical areas that may be involved in this hypervigilance and response include the locus ceruleus and the limbic system.

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Treatment/Clinical Course:

• SSRIs (setraline, paroxteine),Venlafaxine, TCAs, CBT

HY factoids

• Panic disorder patients can be distinguished from patients with generalized anxiety disorder (GAD) by their discrete episodes of acute anxiety, usually lasting less than 30 minutes

PTSD

Overview

• Anxiety disorder marked by the onset of physiological and psychological symptoms following a traumatic event. Individuals with PTSD then continue to reexperience and avoid any stimuli associated with the event. Over time, psychic numbing develops and the individual becomes aloof and disengaged from the world.

Epidemiology:

• Lifetime prevalence rates for women are 12.5% vs. 6.2% for men.

Signs/Symptoms:

• The diagnosis of PTSD requires the exposure to a traumatic event and the above three symptom types: persistent reexperiencing of the event, persistent avoidance of stimuli related to the event and hyperarousal. These intrusive symptoms must last for more than one month for diagnosis (this time course distinguishes PTSD from Acute Stress Disorder, in which symptoms last 2 days to 4 weeks). .

• The event must be of catastrophic proportions, such as an actual or threatened death, assault, rape, serious injury, or watching another person undergo assault.

• A state of persistent increased arousal is seen and significant impairment must be present. The duration of symptoms must be more than 1 month. If similar symptoms are seen for less than 1 month, then a diagnosis of Acute Stress Disorder may be made.

Pathophysiology:

• Genetics: Individuals with preexisting anxiety or depression or with a family history of anxiety disorder or antisocial behavior are more likely to develop PTSD

• Neuroimaging/Gross: dysfunctions in basal ganglia (caudate), cingulate gyrus, and orbitofrontal cortex

• Etiological Models/Factors

• (1) Findings suggest that amygdala is hyperresponsive, the hippocampus and anterior cingulate cortex which would normally modulate the fear response are hypofunctional.

• (2) Dysregulation of cortisol response: The cortisol response of individuals with PTSD appears to be altered (fail to see a dexamethsone suppression in PTSD patients); low cortisol levels in response to stress may be a biological marker for predisposition to development of PTSD

Treatment/Clinical Course:

• SSRI, clonidine (for hyperarousal/anxiety), TCAs, divalproex (anger outbursts/mood instability), atypical antipsychotics (anger, mood instability)

• Psychotherapy, exposure therapy (flashbacks), relaxation training and breathing retraining (for hyperarousal), psychosocial support

HY factoids

PTSD is the only diagnosis in psychiatry that must have a stressful event as a precipitating factor.

Generalized Anxiety Disorder

Overview

• Diffuse, often non-specific anxiety disorder marked by a feeling of anxiety that occurs more days than not, lasts for more than six months, and causes marked distress.

Epidemiology:

• Lifetime Prevalence is approximately 5-6%

Signs/Symptoms:

• Excessive anxiety and worry (apprehensive expectation) occurring more days than not for at least 6 months concurrent with other symptoms such as restlessness (feeling keyed up or on edge), being easily fatigued, difficulty concentrating, irritability, muscle tension, or sleep disturbance.
• GAD is a diagnosis of exclusion; need to rule out the presence of anxiety only during a mood disorder, psychotic disorder or other condition

Pathophysiology:

• Genetics: Runs in families; often comorbid with another axis I disorder such as unipolar depression
• Etiological Models/Factors
• (1) Biological theories of GAD focus dysregulation of serotonin, norepinephrine and GABA neurotransmitter systems;
• (2) Psychoanalytic and psychodynamic theories: conceptualize generalized anxiety as a signal from the unconscious that conflicts exist related to drives and impulses. Anxiety is believed to be caused by the ego's repression of these conflicts

Treatment/Clinical Course:

• Psychotherapy, CBT
•SSRIs,Venlafaxine,Buspirone, Venlafaxine, Mirtazapine, Benzodiazepines

HY factoids

• Patients often unable to recall the onset of their anxiety

Anorexia Nervosa

Overview
• Eating disorder marked by a relentless pursuit of thinness in which the person fails to maintain normal body weight for age and height,

Epidemiology:
• Lifetime prevalence is 1% with onset most often occurring between ages 14 and 18.
• More prevalent in females than males and in industrialized countries

Signs/Symptoms:
• Refusal to maintain body weight above a minimally normal weight for age and height.
• Intense fear of gaining weight even though underweight.
• Denial of the seriousness of the current low body weight with undue influence of body weight on self-evaluation.
• Amenorrhea (at least three consecutive menstrual cycles).
• Two types of Anorexia Nervosa. The Binge Eating/Purging Type involves self-induced vomiting, and misuse of laxatives, diuretics or enemas (not to be mistaken with Bulimia Nervosa). The Restricting Type involves no binge-eating or purging behavior.

Pathophysiology:
• Genetics: increased concordance of AN in MZ twins compared to DZ twins
• Etiological Models/Factors
• (1) Psychological: An increase in societal pressure to be thin appears to be correlated with increased rates of eating disorders in this country during the 20th century.
• Psychological risk factors for the development of eating disorders include problems with autonomous functioning, low self-esteem and interpersonal skills.
• Personality models: traits of risk avoidance, compliance and emotional and cognitive restraint may lead to increased risk of developing an eating disorder.
• (2) Biological Theories: Possible hypothalamic-pituitary dysfunction, though recent studies reveal that the endocrine abnormalities seen in anorexia are the *result* of starvation and they are not unique to eating disorders. Correction of weight corrects these abnormalities

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Treatment/Clinical Course:
• Refeeding, hospitalization, psychotherapy, pharmacotherapy should not be used as monotherapy (TCA, SSRIs, cyproheptadine).

HY factoids
• Hispanic and Caucasian women have been at greater risk for the development of eating disorders than Asian and African American women, but studies reveal that dieting and body weight concerns are infiltrating into all socioeconomic and racial groups.

Bulimia Nervosa

Overview

• An eating disorder in which people eat large amounts of food in a sitting (binging), and then vomit (purging). The vomiting is triggered by a fear of weight gain, from stomach pain, or from the guilt of overeating. People with bulimia also use laxatives, diuretics, and vigorous exercise to lose weight.

Epidemiology:

• Lifetime prevalence is between 1-3%; may be as high as 20-40% on college campuses.
• Tends to have its onset later in life than anorexia nervosa, usually in late adolescence or early adulthood.

Signs/Symptoms:

• A binge is defined as an all-consuming urge to eat large quantities of food -- in the range of 3000 - 6000 calories.
• Accompanied with a feeling of lack of control and decreased awareness of what is going on around them.
• Binge foods frequently include high fat & calorie foods such as ice creams and cakes.
• The binge-eating and inappropriate compensatory behaviors must both occur, on average, at least twice a week for 3 months to meet DSM criteria for BN.

Pathophysiology:

• Genetics: increased concordance of BN in MZ twins compared to DZ twins
• Etiological Models/Factors
• (1) Psychological Theories: The societal demands for thinness considered important in the development of BN as well.
• (2) Personality: Temperamental differences in restraint and impulse control, as well as the individual's innate natural appetite, may lead a patient to one form BN.
• Bulimic patients tend to be more extroverted, impulsive and affectively labile than anorexic patients.
• (3) Biological Theories: The efficacy of serotonin reuptake inhibitors in decreasing the frequency of bingeing and purging suggests that serotonin dysregulation may play a role in the etiology of the disease.

Treatment/Clinical Course:

• Chronic illness; Many patients relapse during periods of increased stress.
• Treatment with psychopharmacologic (SSRI) and psychotherapy
• Many patients appear to relapse after treatment is withdrawn.

HY factoids

• 75% patients with BN also meet criteria for major depression or dysthymia; anxiety disorders are also extremely common (30-40%) as is substance abuse.

Attention-Deficit and Hyperactivity Disorder (ADHD)

Overview

• Behavioral disorder characterized by poor concentration, distractibility, hyperactivity, and impulsiveness that are inappropriate for the child's age (or functionally impairing for an adult).

Epidemiology:

• Lifetime prevalence in United States is 3-10% of the school-age population.
• Rates may be 4 times higher in boys than in girls.
• Rates appear to drop as children age, with approximately 4% of middle school children and less than 1% of high school students receiving stimulant medication for the treatment of ADHD.

Signs/Symptoms:

• Children and adults with ADHD are often easily distracted by sights and sounds in their environment, cannot concentrate for long periods of time, are restless and impulsive, and slow to complete tasks.
• Differential: adjustment disorder, learning disability

Pathophysiology:

• Genetics: First-degree relatives of children with ADHD have a higher incidence of ADHD, and children whose parents have ADHD may be twice as likely to have ADHD themselves
• Etiological Models/Factors
• (1) Biological: Possible dysregulation in norepinephrine and dopamine circuits, especially in the prefrontal cortex.
• Differences in brain structure volumes (caudate lobe), compared to controls.
• EEG differences have also been reported.
• Biological insults during pregnancy (e.g., maternal alcohol use, long labor) or infancy (e.g., lead poisoning, malnutrition) have been implicated in some cases of ADHD.

Treatment/Clinical Course:

• Psychotherapy (behavior modification, family/group therapy, CBT), psychostimulants (methylpheniate, d-amphetamine)

HY factoids

• Two-thirds of adults who were diagnosed with ADHD during their youth continue to show some symptoms and limitation in functioning.

Borderline Personality Disorder

Overview

• One of the most common personality disorders, marked by an instability in self image, mood states, interpersonal relationships, and impulse control

Epidemiology:

• Lifetime prevalence in United States is 1-3% in the general population but 11% of psychiatric outpatients and 19% of psychiatric inpatients.
• Women outnumber men 3:1.

Signs/Symptoms:

• Borderline personality disorder patients demonstrate a pervasive pattern of impulsivity and instability in personal relationships, self-image and affect. There is a tremendous fear of abandonment and use of primitive defenses such as splitting and projection
• Patients are prone to view others as all good or all bad. This phenomenon is called "splitting." Maintaining a rigid classification system often leads the patient to shift between extreme points of view and selectively attend to information in a way that confirms his or her current opinion.

Pathophysiology:

• Genetics: show some familial pattern; often see male family members with ASPD (perhaps a ASPD and BPD are different manifestations of a common personality/neurobiological lesion)
• Etiological Models/Factors
• (1) Psychosocial: BPD associated with sexual/physical trauma in childhood; BPD symptoms may be related to negative cognitive patterns stemming from severe emotional neglect.
• (2) Personality: Many borderline patients have parents with impulsive or depressive personality traits.
• (2) Biological: may have decreased serotonin, associated with increased impulsiveness

Treatment/Clinical Course:

• SSRI, psychotherapy, behavioral skills training

HY factoids

• 10 to 15 percent of these individuals will die by suicide