Table of Contents

Recommended Syllabus Statement ................................................................. 4
Course Description ...................................................................................... 5
Textbooks ........................................................................................................ 5
Course Objectives and Expectations ............................................................. 6
   Overall Goal ............................................................................................ 6
Course Organization ..................................................................................... 7
   Patient Care Rounds (30-50% time or 14 to 20 hours per week) ....... 8
   Case Presentations (10% time or two to four hours per week) ......... 8
   Lectures (5-10% time) ......................................................................... 9
   Teaching Conferences (2-10% time or one to four hours per week) or Grand Rounds 9
   Evening/Night Call (0-1% time) ......................................................... 9
Overview of Course Organization, Teaching Formats and Learning Activities 10
   Instruction and Supervision of the Student ............................................. 10
   Feedback on Student Performance ..................................................... 10
   Evaluation by the Student .................................................................... 10
Grading and Shelf Exam Information .......................................................... 11
Course Faculty Contact Information ......................................................... 114
Patient Encounters (PxDx) ......................................................................... 17
PxDx Module Instructions ........................................................................... 18
Shelf Exam Content and Sample Questions .............................................. 19
   Shelf Exam Content ........................................................................... 19
Psychiatric Case History Outline ............................................................... 21
   Interviewing Skills ............................................................................. 21
   Identifying Data ................................................................................ 21
   Chief Complaint .............................................................................. 21
   Present Illness .................................................................................. 21
   Past Medical History ....................................................................... 22
   System Review .............................................................................. 23
   Family History .............................................................................. 23
   Personal and Social History .............................................................. 23
   Physical Examination ..................................................................... 24
   Mental Status Examination ............................................................ 24
   Diagnosis ....................................................................................... 27
   Discussion ....................................................................................... 27
   Recommendations .......................................................................... 27
Suggestions for Presenting Patients ............................................................. 28
2013-2014 Harold Lawn Award Winner ...................................................... 30
Mini Mental State ....................................................................................... 39
Psychopharmacology .................................................................................. 41
Drugs For Mood Disorders ......................................................................... 52
   Antidepressants: Tricyclics ............................................................. 52
   Antidepressants: SSRIs ................................................................. 53
   Antidepressants: MAOIs ............................................................... 54
   Antidepressants: Others ............................................................... 55
   Mood Stabilizers .......................................................................... 56
Hypnotics ................................................................................................. 57
Benzodiazepines ................................................................. 57
Antihistamines ................................................................. 57
Omega-1 Receptor Agonists (non-benzodiazepine) ................. 58
Other ............................................................................. 58
Anxiolytics ........................................................................ 59
Benzodiazepines ............................................................. 59
Others ............................................................................. 60
Antipsychotics ................................................................. 61
Typical—Dopamine receptor antagonists ............................ 61
Atypical .......................................................................... 62
Psychological Assessment .................................................. 63
I. Objectives ..................................................................... 63
II. Neuropsychological Techniques - Cognitive Functioning .... 63
III. Projective Techniques ................................................ 64
IV. The Mmpi ................................................................. 64
Sample Psychiatric Screens .................................................. 65
Introduction ..................................................................... 65
Psychiatric Work-Up ....................................................... 65
Alcohol And Drug Abuse Screen ....................................... 65
Anxiety Disorders Screen ............................................... 66
Depression Screen ........................................................ 66
Hypomania Screen ......................................................... 67
Eating Disorders Screen .................................................. 68
Family Violence Screens ................................................. 68
Psychosis Screen ........................................................... 69
Sexual Screen ................................................................. 70
Sleep Disorders Screen ................................................... 71
Suicide And Violence Screen ............................................ 71
Trauma Screen ............................................................... 71
Substance Use Disorders .................................................. 72
Definition ....................................................................... 72
Etiology And Pathogenesis ............................................... 73
Individual Factors ........................................................ 73
Epidemiology ................................................................... 76
Pathology ....................................................................... 77
Clinical Evaluation ......................................................... 82
Clinical Course .................................................................. 83
Differential Diagnosis ..................................................... 84
Treatment ....................................................................... 84
Disability Services recommends that all University faculty use the following statement on their course syllabi to inform students of the faculty member’s willingness to provide reasonable accommodations:

The University of Minnesota is committed to providing all students equal access to learning opportunities. Disability Services is the campus office that works with students who have disabilities to provide and/or arrange reasonable accommodations. Students registered with Disability Services, who have a letter requesting accommodations, are encouraged to contact the instructor early in the semester. Students who have, or think they may have, a disability (e.g. mental health, attentional, learning, vision, hearing, physical, or systemic), are invited to contact Disability Services for a confidential discussion at 612-626-1333 (V/TTY) or at ds@umn.edu. Additional information is available at the DS website http://ds.umn.edu.

Recommended by the Senate Committee on Educational Policy, June 2009

The Provost’s Committee on Student Mental Health recommends the use of the following syllabus statement to inform students of campus resources:

As a student you may experience a range of issues that can cause barriers to learning, such as strained relationships, increased anxiety, alcohol/drug problems, feeling down, difficulty concentrating and/or lack of motivation. These mental health concerns or stressful events may lead to diminished academic performance or reduce a student’s ability to participate in daily activities. University of Minnesota services are available to assist you with addressing these and other concerns you may be experiencing. You can learn more about the broad range of confidential mental health services available on campus via www.mentalhealth.umn.edu

Developed and endorsed by the Provost Committee on Student Mental Health, June 2006
Recommended by the Senate Committee on Educational Policy, June 2009
ADPY 7500 is a requirement all medical students will complete in their 3rd or 4th year. The primary goal of this course is to prepare medical students to recognize, diagnose, and care for patients with psychiatric disorders encountered in most medical practices. Students will be assigned to work with interdisciplinary teams who will aid the student in meeting course objectives. Students will be assigned patients and will follow these patients in the hospital and, in some cases, as outpatients. Students will attend teaching rounds/lectures and a variety of teaching conferences (e.g., Grand Rounds). Students will attend a series of lectures/discussions at their individual sites. Each student will be required to write a brief paper concerning a patient-related problem due at the end of the 5th week.

Textbooks

All students should have available to them the Psyche Primer (by Dr. Thomas Mackenzie) which is used for the Year 2 course. The psychopathology information should be reviewed and it will be expanded upon during the clerkship by the lectures, which will also address treatment information.

Each student will have to have access to Quick Reference to Diagnostic Criteria for DSM-IV-TR (Mini-D) and DSM-5 (which is available for 2-day checkout in the Psychiatry Education Office, F256 West, Riverside Campus), and is also available online at:

(Click to link) Diagnostic and Statistical Manual of Mental Disorders: DSM-5

( http://dsm.psychiatryonline.org/content.aspx?bookid=556&sectionid=41101782#CHDCCGHF)

There is no one particular text for the psychiatric externship. The following are some options:


To help prepare students for the shelf exam:

- The Medical Basis of Psychiatry (in med student room)
Course Objectives and Expectations

**Overall Goal**

To prepare the medical student to recognize, diagnose, and care for patients with psychiatric disorders encountered in most medical practices.

**Specific Objectives**

1. Using appropriate interview techniques, the student will be able to elicit a complete psychiatric history from psychiatric and medical patients and will be able to amplify or confirm the patient’s history by information from relatives and/or social agencies.

2. The student will be able to perform a physical examination emphasizing aspects pertinent to the psychiatric evaluation and a mental status examination sufficiently comprehensive to detect, at a minimum, disorders of orientation, thinking, mood, and cognition.

3. The student will be able to select and apply major diagnostic tests and procedures including laboratory tests, neuroimaging tests, psychometrics, and electroencephalography in psychiatric practice.

4. The student will be able to identify psychiatric emergencies (e.g., suicidal, violent, or delirious patients; withdrawal symptoms) and be familiar with their management. In particular, the student will develop a repertoire of questions and interpretive skills sufficient to permit estimation of the likelihood of suicide and methods of safeguarding against it.

5. The student will recognize and apply principles of giving and receiving consultation from other physicians and to cooperate with social service agencies.

6. The student will recognize basic processes of judicial commitment in Minnesota and other basic forensic issues.

7. The student will effectively utilize the processes of patient education, reassurance, and support. The student will learn indications for, and gain some familiarity with, other psychological interventions.

8. The student will be able to describe the clinical presentations, course, and prognosis of the following disorders with special emphasis on findings discriminating among them:
   a. Depressive Disorders (e.g., Major Depression)
   b. Neurocognitive Disorders (e.g. Delirium, Dementia)
   c. Schizophrenia Spectrum and Other Psychotic Disorders
   d. Bipolar Disorders
   e. Anxiety Disorders (e.g., Panic Disorder, GAD, Social Anxiety)
   f. Addictive Disorders
   g. Child or Adolescent Psychiatric Problems (e.g., ADHD, Conduct Disorder)
   h. Personality Disorder (e.g., Borderline, Anti-Social, Paranoid)
i. Trauma-Related Disorders (e.g., PTSD)

j. Eating Disorders

9. The student will identify and describe somatic treatments:
   a. Common pharmacologic treatments, including indications, contraindications, and side-effects of antianxiety agents, antidepressants, antipsychotics, and sedative-hypnotics.
   b. Electroconvulsive (ECT) treatment indications and effects.

10. The student will be able to detect common psychiatric disorders in the aged.

11. The student will be able to identify common psychiatric disorders first diagnosed in infancy, childhood, or adolescence.

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**Course Organization**

**Level of Responsibility of Students**

In general, the student will function in a “sub-intern” capacity. The extent of the specific responsibilities may vary from site to site.

All sites except Anoka Metro Regional Treatment Center (AMRTC) will be staffed by residents as well as attending staff. If residents are available, the first patient contacts by the student will be with the resident or staff (i.e., the student may observe the resident or staff interview the patient and then discuss the case and the appropriate evaluation and treatment plan with the resident). Thereafter, the student may continue to function directly under the supervision of the resident, as on other clinical services. However, the student should also be assigned his or her own cases so that they may take primary responsibility for total management of several patients. He or she should write management therapy, drug therapy, and administrative orders under supervision. If residents are not available at teaching sites, the attending staff will assume all such supervision.

Any mechanical procedure to be done on patients will be done under direct supervision of the resident or attending staff. The student will only be allowed to do procedures unsupervised when staff feels the student is ready.

In order to give students exposure to a diversity of patients, they may be placed on different services (usually only two services, e.g. two 3-week blocks) during their clerkship.

**Student Learning Activities**

**Patient Care (50% time or 20 hours per week)**

Each site will have available a variety of inpatients and/or outpatients. All students *should participate in the evaluation and management of patients with the following diagnoses:*
1. Mood Disorders (depressive disorders, bipolar).
2. Neurocognitive Disorders (dementia, delirium).
3. Psychosis (schizophrenia, bipolar, psychotic depression, organic psychosis)
4. Anxiety disorder (obsessive-compulsive, panic disorder, GAD, PTSD or phobic disorder).
5. Substance use disorder.

In addition, it is SUGGESTED that the student also see patients with the following disorders:

1. Geriatric psychiatric problem
2. Child or adolescent psych problem
3. Cross-cultural psych problem
4. Eating disorder.
5. Antisocial or borderline personality disorder.
7. Mental retardation.
8. ADD/ADHD

Students will complete some psychiatric patient work ups during the six week clerkship. These will include a complete history, relevant physical exam, and mental status exam as well as a working diagnosis and treatment plan. These reports will be written or dictated, and when reviewed and countersigned by supervisory staff, will become a part of the patient’s medical record.

Each student will observe at least one ECT procedure.

**Students are required to maintain a Patient Encounter Form** (see page 12), which is a record of the number of patients with various required (and some suggested) diagnoses. This form with the student’s patient encounters is to be reviewed at mid-term and again at the end of the rotation in discussions with their attendings. Students should work with their attendings to make sure they see patients with all the required diagnoses. Students should turn in the completed form to the site coordinator at the end of their rotation. If there are certain categories for which the student has not seen or observed patients, the student could list case or topic discussion to fulfill this requirement.

**Patient Care Rounds (30-50% time or 14 to 20 hours per week)**

Patient care rounds will vary according to the teaching site. In general, these will be held with various ancillary team members (psychologist, social worker, nurse, occupational therapist) as well as the supervising staff (resident, attending staff).

**Case Presentations (10% time or two to four hours per week)**

The student will present cases to attending staff, or, at the discretion of each site, to clinical staff asked to come to the various sites for the purpose of helping with medical student teaching.
Lectures (5-10% time)

To help provide uniformity in student learning, a series of pertinent lectures which deal with teaching the course objectives will be taught at the individual teaching sites. The following topics will be covered at each site:

- Psychiatric History and Mental Status
- Assessment of Violence and Suicide (Forensic Psychiatry)
- Principles of Psychopharmacology
- Psychological Assessment
- Psychiatric Manifestations of Medical Illness
- Neurocognitive Disorders
- Affective Disorders
- Psychotic Disorders
- Anxiety Disorders
- Substance Use Disorders
- Somatoform Disorders
- Personality Disorders
- ECT
- Child / Adolescent Disorders (if offered)

The student is expected to attend all lectures.

In addition, other lectures, determined by the individual site directors, may be offered at the various sites. The student is expected to attend these lectures.

Please note that lectures will not cover all material evaluated on the Shelf Exam. Extra reading and study to augment student knowledge is needed.

Teaching Conferences (2-10% time or one to four hours per week) or Grand Rounds

Each teaching site will usually have at least one major teaching conference per week which is attended by the various psychiatric staff at each site. The format should be similar to a grand rounds presentation and should cover various topics or case presentations. If a case presentation is involved, the student assigned to the case should do the case presentation.

Evening/Night Call (0-1% time)

The student will be expected to take evening/night call depending on the teaching site. If evening/night call is expected, the student will be supervised.
Overview of Course Organization, Teaching Formats and Learning Activities

**Instruction and Supervision of the Student**

Attending staff will take primary responsibility in supervising the student either directly or through the resident. One attending staff will supervise between one and three students at a time. Where residents are not available, the attending staff will provide the necessary day-to-day supervision, feedback, and role-modeling. Faculty/residents will hear case presentations, co-sign chart notes and patient orders, read and provide feedback to the student about work-ups, and meet regularly with the student to discuss case management. Students may also give lectures. The attending staff will be responsible for assigning patients to the student for work-up and case management.

Psychiatric residents will be available to help supervise the medical student at teaching sites (except at AMRTC). The resident will serve as a role model as the student watches the resident work with patients. Although no formal supervising time is normally arranged between resident and student, the resident will provide supervision with feedback to the student on a day-to-day basis for the patients assigned to the student. The resident can countersign student-written orders.

The student will have exposure to, and supervision by, other health professionals (including social workers, psychologists, and nurses) on an informal and individualized basis, usually only in conjunction with the needs of specific patients. These personnel will usually be present during patient rounds. Psychologists may lecture on psychological assessments.

Psychiatrists other than the attending staff may provide formal supervision or instruction to the student at the various sites. They may supervise the weekly case presentations or provide pertinent lectures.

**Feedback on Student Performance**

Students will receive day-to-day feedback from supervising staff.

Each student should be observed doing at least one patient interview (preferably live, but possibly on videotape by the attending staff and should be provided with performance feedback).

Midterm evaluations will be conducted in an effort to support student growth and opportunities for improvement.

**Evaluation by the Student**

Each student will be asked to provide feedback about the teaching and general experiences during the clerkship to the attending staff (and residents) by completing an on-line evaluation through the web based E*Value system.
Grading and Shelf Exam Information

Although a grade for the clerkship is based primarily on the subjective evaluation by instructors, it is important that some amount of standardization be achieved in order to indicate to students how they performed in relation to their peers, to compare outcomes at the various teaching sites, and to provide general feedback to the Department of Psychiatry on the effectiveness of its teaching and accomplishing the course goals and objectives. The final grade for each student for the clerkship will therefore be derived from a composite score based on the following:

1. **Clinical Performance Rating.** Attending staff will complete an on-line evaluation of the student’s clinical performance through the web-based E*Value system. The attending staff will consult with residents and/or other staff involved with teaching the student during the duration of the clerkship when completing this form. The clinical performance is the most important determinant in the overall clerkship grade. **The evaluation score is worth 60 points and 60% of the student’s grade.**

2. **Written Paper.** The student will need to write a paper to complete the course. The paper should focus on one of the patients under the student’s care and should be between five and ten pages long (or 1250-2500 words, double spaced) with an additional bibliography of at least six major references. This paper must be turned in to the attending staff by the end of the fifth week of the clerkship. A staff member, usually the attending, will read and evaluate the paper. Late papers may result in a grade reduction. Five categories are considered when grading the paper: Relevance (5 points); Analysis (7 points); Clarity (5 points); and Sources (3 points). **The paper is worth 20 points and 20% of the student’s grade.**

   There is a $500.00 cash prize given each year (the Harold Lawn Award) for the best paper submitted by a medical student.

3. **Psychiatry Shelf Exam.** Students will take psychiatry Shelf Examination toward the end of their rotation. This exam is a requirement and cannot be rescheduled except in the case of an emergency. **The exam is worth 20 points and 20% of the student’s grade.**

   You will need to pass the Shelf Exam in order to pass the course. The passing score is 62. If a student fails the exam, they will take the exam at the end of the next 6 week rotation without having to repeat the clerkship. If a student fails the exam twice, they will need to repeat the entire clerkship.

   A content outline of the Shelf Exam in psychiatry and some sample questions are in this syllabus.
**Grading System**

A grade of Honors (H), Excellent (E), Satisfactory (S), No Credit (N) or Incomplete (I) will be determined by the following rubric:

- Clinical performance (E*Value evaluation) = 60%
- Paper Grade = 20%
- Shelf Exam = 20%

Grades are assigned according to the following score table:

- Honors = 92-100
- Excellent = 83-91
- Satisfactory = 62-82

Grade information will be posted on the ADPY 7500 Moodle site. The Shelf Exam scores are posted within three weeks after the exam and will have been adjusted to represent the 20% of the grade. The mean score for the period can be obtained by contacting the Psychiatry Clerkship course coordinator.

The clinical scores and paper scores may be entered into the Moodle site any time after the end of the rotation up until four weeks after the rotation after all evaluations are completed by the student.

Failure to submit a paper or to take the exams will result in a failing grade regardless of the number of points accumulated from the student’s clinical performance. A failing grade can only be changed by repeating the course and completing all the requirements. Submitting a written paper late, without permission, may result in a grade reduction. It is **NOT** an option to reschedule the Shelf Exam except under unusual/emergency circumstances and only with the written permission by the Psychiatry Clerkship course coordinator and/or the Psychiatry Clerkship director.

Students can view their official evaluations for the rotation. (This evaluation form, as well as the definition and anchors, can be found on the Psychiatry Clerkship Moodle website). The definition and anchors are also posted on the E*Value evaluation as well. At the beginning of the fifth week of the rotation, attendings and students will receive an e-mail telling them that they have a pending evaluation to complete before the end of the rotation. The E*Value system automatically sends out reminders every few days to those people who have pending evaluations. If you do not have a login for the E*Value system or if you have questions about the procedure then please contact the Course Coordinator.

Students must complete all pending evaluations for the Psychiatry rotation, as well as patient encounter forms in order to access grades or look at evaluations of performance. Also, all evaluations must be completed prior to graduation.
Evaluation Procedures

On the Psychiatry rotation, some students will have one attending for the first three weeks and a second attending for the remaining three weeks. The mid-rotation evaluation and the end of rotation evaluation will both be on E*Value for UMMC, Regions, and Veteran Affairs Medical Center, but not HCMC or AMRTC, where only the end rotation evaluation will be on E*Value.

Midterm feedback is very important and students should meet with their attending at mid-term (or at the end of a 2 or 3 week rotation). A mid-term evaluation form is in this syllabus, which can be used for this purpose, as well as for the final evaluation. Each student should take this special mid-term evaluation form or the E*Value form to their attending and ask the attending to complete it (and ideally the attending should then meet with the student and discuss student performance). Students should then make a copy of the completed feedback and give it to the next attending. Students should give the other copy of the completed form to the site coordinator, who then will keep these forms for a later reference. Sites will determine which attendings will complete evaluations based on the level of involvement with the student.

The mid-term and final evaluation forms can be found on Psychiatry Clerkship Moodle website.
## Course Faculty Contact Information

### Anoka Metro Regional Treatment Center

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<thead>
<tr>
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<th>Name</th>
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**University of Minnesota Medical Center, Fairview (Riverside Campus)**

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Psychiatric Clerkship—Procedure for Monitoring Patient Encounters

1. Students will carry the patient encounter form with them and will keep count on the form of their patient encounters.
2. Students will use the form to **discuss** with their attendings at **mid-term** whether any adjustments need to be made to assure their seeing at least one patient in each required category.
3. Students will use the form to **discuss** with their attending at the **end of the rotation**.
4. Students will **turn in the competed** form to the site coordinator at the end of their rotation.
5. The site director should look at these forms and attempt to make corrections so students could have all the necessary patient encounters.
6. Site coordinators will maintain these forms and send the copies to the Psychiatry Medical Student Coordinator, who will then tabulate the results.
7. Depending on outcome, adjustments will be made in the curriculum.

### Patient Encounter

<table>
<thead>
<tr>
<th>Patient Encounter</th>
<th>Number of 1:1 Interviews Performed</th>
<th>Number of Evaluations Observed</th>
<th>Number of Cases Reviewed/Discussed</th>
<th>Clinical Setting</th>
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<tbody>
<tr>
<td>1. Mood Disorder (depression, bipolar)</td>
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<td>2. Neurocognitive Disorder (dementia, delirium)</td>
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<td>3. Psychosis (Either Schizophrenia, Bipolar, Psychotic Depression, or Organic Psychosis)</td>
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<td>4. Anxiety Disorder (Either OCD, Panic Disorder, GAD, PTSD, or Phobic Disorder)</td>
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<td>5. Substance Use Disorder</td>
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<td>6. Geriatric Psychiatric Problem</td>
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<td>7. Child or Adolescent Psych Problem</td>
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<td>8. Cross-Cultural Psych Problem</td>
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<td>9. Personality Disorder (Borderline, Anti-Social, Paranoid, Etc.)</td>
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<tr>
<td>10. Eating Disorders</td>
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<td>11. Trauma-Related Disorders</td>
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Note: At least one patient in each category 1-5 is required.

Patient with multiple diagnoses can count for more than one patient encounter.
Dear M3 & M4 Students:

Welcome to the Psychiatry Clerkship! U of M students electronically log clerkship cases and procedures via an E*Value software module called PxDx. This allows for electronic tracking of experiences by student and by clerkship to determine levels of experience and areas for additional exposure. You will not need to complete the patient encounter form that is on Moodle. I will continue to leave this document for you in case you want to use it for your tracking purposes.

The process is pretty straightforward and is outlined below:

1. Log onto [www.e-value.net](http://www.e-value.net):
   - Navigate to the USER Menu → Select the PxDx program – Do NOT select the psychiatry clerkship
   - Click Add New PxDx Records

2. The first entry screen is MAIN → Three required elements need to be added:
   - Enter date of procedure/case
   - Enter clerkship (from drop down list—use may use filter box)
   - Enter clinical site (from drop down list—you may use filter box)
   - Hit Next button

3. The next screen will be Diagnosis … IF YOU ARE ENTERING a PROCEDURE Click on the PROCEDURE TAB and skip to #4.
   - Select the case/diagnosis you saw
   - Under the role item, select: Actual patient, simulation, standardized patient, web/paper/video case.
   - Hit ADD
   - Hit SAVE—You are done and the screen will return to MAIN for another entry.

4. To enter a Procedure
   - To enter a procedure select the Psychiatry clerkship and the required procedure list will appear
   - Select the procedure/exam you did
   - Under the role item, select: assisted, observed or performed.
   - Hit ADD
   - Hit SAVE—You are done and the screen will return to MAIN for another entry

At the end of your clerkship you will need to print out a report of your cases to give the clerkship coordinator/director on the final day.

A few notes:
- The “Forgot Password” link should work and will send you a link to change your password
- If it does not, email me and I will send your password to you (nesbi029@umn.edu)

Please do not hesitate to contact me if you have any questions or problems.

Brooke Nesbitt, MS
(612) 626-5387  nesbi029@umn.edu
Shelf Exam Content

General Principles 5%-10%
Organ Systems
Mental Disorders
Establishing a Diagnosis
Mental disorders usually first diagnosed in infancy, childhood, or adolescence 5%-10%
Substance-related disorders 5%-10%
Schizophrenia and other psychotic disorders 5%-10%
Mood disorders 5%-10%
Anxiety disorders 5%-10%
Somatoform disorders 1%-5%
Other disorders/conditions 5%-10%
Applying Principles of Management 20%-25%
Diseases of the Nervous System and Special Senses 10%-15%

Note: In addition to the above information about content, the exam covers much more neurology and child-adolescent psych diagnosis and treatment—questions on normal development, developmental disabilities, metabolic abnormalities, mental retardation, Down syndrome, Asperger’s disorder; substance abuse (including Wernicke-Korsakoff encephalopathy); some brain/structure/function questions; consult-liaison questions of medical/psych issues, dementia, delirium; cardiac toxicity of overdoses, MAOI/other drug toxic reactions.

Sample Questions

1. A previously healthy 17-year-old girl is brought to the physician for evaluation because of the loss of appetite, sleeplessness, and extreme irritability for 3 weeks. After missing many practices, she quit the softball team that she previously enjoyed. She often feels tired and has difficulty sitting still and concentrating on schoolwork. Her menses occur at regular intervals. She weighs 50 kg (110 lb) and is 168 cm (66 in) tall. Her blood pressure is 110/70 mm Hg, pulse is 74/min, and respirations are 16/min. Which of the following is the most likely diagnosis?

(A) Adjustment disorder with mixed disturbances of emotions and conduct
(B) Anorexia nervosa
(C) Attention-deficit/hyperactivity disorder
(D) Dysthymic disorder
(E) Major depressive disorder

2. A 45-year-old man is brought to the physician by his spouse. He has been drinking heavily since he was passed over for a job promotion 3 days ago. He stayed in bed over the weekend. He has no personal history of psychiatric disorders and no personal or family history of alcohol abuse. He is crying and states, “I can’t believe it” when addressed. When asked what he will do, he states, “I don’t know, but if I don’t go back to work tomorrow, I’ll likely lose my job.” Which of the following is the most likely diagnosis?
3. A 52-year-old woman whose husband died 2 months ago consults a physician because of headaches and feelings of uncertainty. She describes the headaches as a band around her head; they occur unpredictably and are not accompanied by any other symptoms. She has no history of psychiatric illness. While talking with the physician, the patient begins to cry and talk about her deceased husband; she feels her life is empty now and worries about her future. Which of the following is most appropriate at this point?

(A) Allow her to express herself
(B) Prescribe an antianxiety drug
(C) Prescribe an antidepressant drug
(D) Refer her for psychological testing
(E) Obtain a psychiatric consultation

4. A 10-year-old boy is brought to the physician because of increasing behavior problems in school since starting 5th grade 3 months ago. His teacher states that he is unable to sit quietly through a classroom period and frequently disrupts the class and interrupts other children while they are talking. His parents report that he has always been an active child and are concerned because he is inattentive when he runs or walks. During the examination, he fidgets with his hands and feet and is easily distracted from completing a task. Which of the following is the most appropriate pharmacotherapy?

(A) Amitriptyline
(B) Fluoxetine
(C) Haloperidol
(D) Imipramine
(E) Methylphenidate

5. A 32-year-old woman is brought to the emergency department because of fever, hallucinations, agitation, and confusion for 8 hours. She has a history of alcohol, cocaine, and benzodiazepine abuse. Her temperature is 37.8°C (100°F), blood pressure is 150/90 mm Hg, pulse is 110/min, and respirations are 16/min. She is tremulous. The lungs are clear to auscultation. She has a holosystolic murmur; the abdomen is tender, and the liver edge is palpable 3 cm below the costal margin. Rectal examination shows no abnormalities. She has telangiectasia. A complete blood count and liver function tests show no abnormalities. Her serum alkaline phosphatase activity is 200 U/L, serum alanine aminotransferase (ALT, GPT) activity is 60 U/L, and serum aspartate aminotransferase (AST, GOT) activity is 90 U/L. Which of the following is the most likely cause of this condition?

(A) Acute cocaine toxicity
(B) Alcohol withdrawal
(C) Benzodiazepine withdrawal
(D) Panic disorder
(E) Schizophreniform disorder
Psychiatric Case History Outline

**Interviewing Skills**

An earnest attempt must be made to include all the diagnostic possibilities and to avoid prejudice by presenting data which refers to only one of the illnesses which require differential consideration.

1. Begin the interview with open-ended questions (e.g., Could you tell me what brings you to the hospital? What sort of problems have you been experiencing?).

2. Allow patients to answer questions in their own words; avoid questions which might shape the patient’s answer, at least initially.

3. Avoid questions that might suggest the appropriateness of an answer (e.g., You haven’t been hearing voices, have you?).

4. Ask more specific questions as the interview progresses.

5. If the interview is not progressing, try obtaining a chronological life history. Ask about childhood, early school experiences, etc.

6. If questions are upsetting to the patient, especially if the potential for violence is escalating, switch to questions which are effectively neutral. Return to your original line of questioning when the patient has calmed down.

7. Always inquire about suicidality. Mentioning suicidality does not precipitate suicidal ideation in patients.

8. It is improper to employ flippant language. The hospital record is a formal document which may be subject to inspection by courts of law.

**Identifying Data**

This is the nth psychiatric admission for this (age) year old, (marital status), (race), (sex). He/she was admitted to the hospital by (relative, police, self).

**Chief Complaint**

Should always be a direct patient quotation of the patient’s own view of the problem.

**Present Illness**

The Present Illness is the most important part of the history. Most of the data which will aid, directly or indirectly, in the diagnosis and treatment of the patient’s illness should be included in the Present Illness. Although certain phases or manifestations of an illness may have existed for years, they are reported in the Present Illness. For example, in the case of a patient admitted to
the hospital with affective symptoms, an affective episode twenty years ago is described in the Present Illness.

When relevancy of certain data of the more remote history is indeterminate, such data should be included in the past medical or social history as is appropriate.

In most cases, the data of the Present Illness are presented chronologically. Occasionally, the complexity of a Present Illness will require separate consideration of part of the history or separate consideration of one informant’s report.

The following specific considerations should be observed when writing a Present Illness:

1. What was the mode of onset? Was it insidious or abrupt? Was it first apparent to the patient or to others?

2. How did the evolving illness affect the patient’s usual life functions? Was his marriage, occupation, or avocations disrupted? Did his relationships with people change? Were there alterations in habits such as those involving taking meals or sleep patterns? If alterations have occurred, when did they occur, and how extensively?

3. What are the specific symptoms which have appeared during the time of the present illness? A psychiatric case history, like histories elsewhere in medicine, is based on patterns of symptoms.

Other events during the time of a Present Illness are important, but the central issue is that of the symptoms. A diagnosis becomes possible when it is found that a patient has experienced a pattern of symptoms similar in content and chronology with the natural history of a known illness.

Do not forget to include pertinent negative findings as well as positive findings.

Obviously, a great many questions could be asked of each patient, but certain symptoms have proven to be particularly important in psychiatric disorders. They include symptoms of change in physiologic functions (eating, sleeping, elimination, menses, potency), loss or gain in weight, changes in mood, changes in memory or judgment, changes in behavior suggesting hallucinations or delusions, ideas of sin, guilt, persecution, jealousy, or infidelity. This list is not complete, but representative.

**Past Medical History**

Pertinent childhood illnesses or facts concerning growth and development.

In chronological order: operations, other hospitalizations, significant injuries, and significant illnesses not resulting in hospitalization.

Specific inquiry should be made concerning head injury and neurological illness.
Ask about medicines and drugs, both those prescribed and those obtained without prescription. Ask about amount and kind of alcohol intake.

**System Review**

The chief function of the System Review in a psychiatric case history is to provide a systematic investigation of symptoms of non-psychiatric illnesses. The System Review does not serve to extend the Present Illness, but fills gaps which may have been left in the Present Illness.

Report positive findings here not usually seen in psychiatric illness (hemoptysis, melena, orthopnea, etc.). It should be noted that when the patient’s psychiatric diagnosis is hysteria, the special symptom review for that illness becomes part of the Present Illness.

**Family History**

Note the presence or absence of psychiatric or neurologic illness among first degree relatives (parents, siblings, or children). Inquire specifically about “nervous breakdown”, depression, schizophrenia, alcoholism, mental deficiency, delinquency, legal difficulties, suicide, suicide attempts, neuroses, epilepsy, syphilis, hospital care, and psychotherapy. When any positive material emerges, age of onset, the course of the illness, specific symptoms, and treatment are all important.

Similar history concerning second degree relatives (especially aunts, uncles, and grandparents) is also important.

Finally, questions should be asked concerning family history of the more important and common non-psychiatric illnesses.

**Personal and Social History**

Upbringing including family constellation, socioeconomic status, and religion. Schooling facts including grade completed and age when stopped, reason given, ability, performance, and behavior in school. Types of work and job history if pertinent. Military service, record, and type of discharge. Sexual and marital history including details not only of sexual experience, but also family dynamics and the patient’s role may be of importance. Premorbid personality refers to the personality of the patient before the onset of an acute psychiatric illness. Although it is often delineated with difficulty, it is worth assessing a patient’s personality in order to appreciate the changes subsequent to illness. Describe briefly his premorbid activities, interests, general mood, and social patterns.

**Physical Examination**

Includes complete neurological examination.
Mental Status Examination

The Mental Status Examination is an amplification of the examination of highest neurological function. As amplified, it is rendered separately and placed after the Physical Examination. Ordinarily, the Mental Status Examination is divided into six areas.

1. **General Appearance and Behavior.** Does the patient appear his stated age? Describe his facial expression as well as his condition and dress. If his motor activity is unusual in any way, describe it (it may be overactive, underactive, or give evidence of neurologic disturbance). Is the patient responsive and alert? Is he cooperative?

2. **Form of Thought.** This area of the Mental Status Examination is the most difficult for students. It involves observations about verbal patterns which one does not ordinarily make. Students should remember that this area of the Mental Status Examination is the least precise. If a student is confused as to whether an abnormality does or does not exist, he should describe what the patient says with a few brief quotations.

Does the patient give evidence of peculiarities of rate and rhythm of speech? Is there evidence of a flight of ideas (rapid digression from one idea to another)? Remember that flight of ideas is often accompanied by push of speech, a jocular mode, puns, or clang associations. Is tangential speech present? In tangential speech the connection between two ideas is not understandable. Such speech is impossible to follow logically. Is circumstantial speech present? Circumstantial speech involves over inclusion of many trivial details. The connection between ideas is easily understood. Circumstantial speech is not necessarily pathological.

Other terms sometimes used to describe patterns of speech are as follows:

- **Blocking:** Sudden stoppage of speech or interruption of a train of thought without apparent reason.

- **Mute:** No speech.

- **Echolalia:** Repeating what is said by other people in an echoing fashion.

- **Loosening of Associations:** More or less equivalent to tangential speech.

- **Incoherent Speech:** More or less equivalent to tangential speech.

- **Perseveration:** Repeating the same word, phrase, or idea over and over again.

- **Word Salad:** An extreme type of tangential speech in which the individual words occur in an entirely disconnected fashion.
Neologism:  Words invented by connecting syllables to produce a new word.

3. Thought Content. Phobias, obsessions, and compulsions are included here if the patient speaks of these phenomena as occurring at the present time (they are otherwise described in the Present Illness). A phobia is an intense, unreasonable fear associated with some situation or object, (i.e., fear of heights, closed places, etc.). An obsession is a recurrent or persistent idea or thought which is recognized as foreign or alien to the individual and which is accompanied by the desire to resist it. A compulsion is a recurrent act recognized as foreign or alien to the individual and which is accompanied by the desire to resist it.

Also included in abnormalities of mental content are self-deprecatory and self-accusatory ideas, suicidal or homicidal ideas, and unusual preoccupations or ruminations if they occur at present.

Hallucinations, delusions, ideas of reference, feelings of derealization, and depersonalization are reported in this section of the mental status examination. They are certainly reported here if they occur at present. They are sometimes reported even if they were a former part of the Present Illness.

Hallucinations are false sensory perceptions. Sometimes an attempt is made to distinguish between illusions (the misinterpretation of real sensory stimuli) and hallucinations which are said to occur in the absence of real external sensory stimuli. For practical purposes, one can distinguish between illusions and hallucinations. It is likely that most patients with hallucinations are experiencing both true hallucinations and illusions. Hallucinations can occur in any of the five sensory modalities. Auditory hallucinations are the most common. Tactile hallucinations are sometimes called hepatic hallucinations and should not be confused with hypnagogic hallucinations which occur in the state between wakefulness and sleep. Olfactory and gustatory hallucinations may sometimes occur.

A delusion is a fixed false belief. It is often said that a delusion must be in keeping with a patient’s subculture. This qualification is of interest to psychiatrists in special situations, but usually it is not a practical consideration. Persecutory delusions are obviously those of persecution. Delusions of megalomania are delusions of greatness. One kind of delusion which has its own name because it occurs so frequently in schizophrenia and in mania, is the delusion of passivity. This is the belief that one’s thoughts or motor behavior is under the control of an outside agent. The outside agent may be either animate or inanimate. It may be close at hand or at a distance. The patient may believe that his mind is being controlled, that thoughts are being put in his mind, taken out of his mind, or somehow molded. He may believe that his body is being controlled marionette-like. This experience of passivity is often accompanied by a complex array of other delusions and hallucinations so that it may be difficult to determine at what point one pathological phenomenon ends and another begins.

The term “idea of reference” is often confusing to students because it covers a wide variety of experiences. Normal people have ideas of reference in embarrassing social situations. Patients who are psychotic may experience ideas of reference in a bizarre and
pronounced fashion. An idea of reference is the unwarranted idea the people are talking about you, watching you, or noticing you.

Derealization is the feeling that the world has changed, usually in some alien way. The patient may or may not know that this feeling is abnormal. Depersonalization is a similar feeling, however, it applies to the patient’s own body. He feels that his body is somehow changed or that his identity has somehow changed or become lost. The patient may or may not believe the feeling is abnormal.

4. Affect and Mood. Affect is the patient’s current emotional state. It is the state the interviewer can observe. There are three basic questions which can be asked about affect.

   A. What is the type of affect? Is the patient’s affect depressed, normal, or elevated?

   B. Is the patient’s affect labile? Does his affect remain stable or does it change noticeably as you speak with him?

   C. Is the patient’s affect appropriate? This is the vaguest aspect of description of affect. A patient’s affect may be judged to be inappropriate for any of a number of reasons. If the student decides that his patient should be described as having inappropriate affect, it is the student’s responsibility to explain how the affect is inappropriate. For example, the affect may be inconsistent with the patient’s expressed mood or thought content.

Mood refers to the pervading and prevailing emotional tone. Mood usually refers to what the patient says about his emotional state. Remember, a person may be in a depressed mood while he is at the moment responding affectively with a smile or joke. Mood may also be labile, meaning there is a rapid fluctuation between feeling happy, sad, angry, etc.

5) Sensorium and Intellectual Resources:

a) Sensorium:
   Orientation - time (day of month, month, year, day of week, season), place, person. If not oriented give patient’s answers and correct information.
   Retention and immediate recall - give a stress address, a person’s name and a color and test in five minutes.
   Recent memory - date of admission, brought to the hospital by whom.
   Remote memory - when and where born, date of marriage, names and ages of children.

(Section b of Sensorium and Intellectual Resources is required only if there is impairment of section a, or if the history suggests a brain syndrome.)

b) Intellectual functioning:
   In evaluating the following tests of intellectual functioning, factors such as the patient’s educational level, ability to concentrate, anxiety, and willingness to cooperate should be considered Calculations - subtract 7 from 100 and 7 from the answer and each succeeding answer. (Average adult has less than four errors and finishes within 60 seconds.) Multiply 7 x 8, 3 x 4, etc. Count from 20 backwards to 1.
General information - name five large cities and the last five presidents.

Meaning of proverbs - (1) Don’t cry over spilt milk. (2) All that glitters is not gold. (3) A bird in the hand is worth two in the bush. (4) A rolling stone gathers no moss. Is the patient able to identify the abstractions involved in the proverbs?

6. Insight and Judgment. Insight signifies that the patient realizes that he is ill and he understands something of the nature of his illness. It does not refer to etiology or psychodynamic aspects of the illness.) Insight may be assessed by evaluating the patient’s response to the following questions:

   A. Are you sick in any way?
   B. What sort of sickness?
   C. Do you need help?
   D. What sort of sickness do people have here?

Judgment may be assessed by evaluating the patient’s responses to the following questions:

   A. What would you like to do next?
   B. What do you plan to do when you leave?
   C. Why were you brought here?

**Diagnosis**

Diagnostic choice. Use Axes I through V of the DSM-IV diagnoses. Use a problem-oriented approach.

**Discussion**

Support your diagnostic choice. Length depends on complexity of case.

**Recommendations**

This section is brief and covers diagnostic and therapeutic counsel.
1. Do not read the history.

2. Do not exceed ten minutes (allowing for interruptions).

3. Start with the Identifying Data (name, age, race, marital status, employment status).

4. Avoid specific dates. Begin with “The patient was admitted to the University of Minnesota _____ days/weeks/months ago.” Do not refer to events occurring on specific days, instead use “At the age of 15, the patient...” Instead of saying “Between November and January of 1955 and 1956”, state “For a three month period when the patient was 20 years old...”

5. Begin with the Psychiatric History. List symptoms in order of severity. Explain how long the symptoms have persisted and what has happened as a result (i.e., hospitalization, medications, partial recoveries).

6. It is important to know whether the illness has been chronic, perhaps with fluctuations, or episodic with full remissions between episodes. If the patient has had more than one episode, describe subsequent episodes, briefly giving the same information that was given for the first episode. Symptoms and life events obviously are interrelated, but emphasize the symptoms rather than the life events unless the life events are presumed to be causally related to the symptoms (not subtly related), such as the death of a close relative, serious medical illness, etc.

7. A brief Family History should include whether a close blood relative of the patient had a serious psychiatric illness requiring treatment (and what the treatment was, if known), pertinent medical illnesses, suicides, and alcohol or drug problems.

8. Briefly, the Social History should include circumstances of upbringing (particularly whether the parents were divorced or separated and whether the patient was brought up by both parents), parental vocations, siblings, years of education and how well the patient did in school (grades and adjustment), military and job history, marital history, and number and ages of children.

9. Review the Past Medical History only as it is pertinent to psychiatric problems.
10. Give the Mental Status as it was obtained either on admission or at the first opportunity to fully examine the patient. The mental status findings should be presented in the following order:

a. Appearance and behavior.

b. Form and content of thought.

c. Affect and mood.

d. Memory and intellectual functioning.

e. Insight and judgment.

11. End the presentation with how the patient has been doing, whether he has improved, and what treatment he is receiving including medications.

With rare exceptions, all this can be presented in ten minutes. The trick is to keep in mind at all times the differential diagnosis and the points for and against each of the reasonably likely diagnoses. The reason for presenting the history and mental status according to the above sequence is to avoid leaving out important information and to make it easier for the listeners to follow the narrative.
The Role of Ambivalent Self-Structures in the Development and Maintenance of Obsessive-Compulsive Disorder: New Directions for Treatment

Megan Brandeland
ADPY 7500
Word Count: 3,040
Case Report: Sarah is a 16-yo female with a 5-year history of obsessive-compulsive disorder, depression and eating disorder NOS admitted for worsening OCD rituals and suicidal ideation. Sarah has been hospitalized 29 times for OCD-related symptoms, most recently 2 weeks ago. Since that time, her OCD behaviors have worsened significantly to the point where she felt that she “wanted to be dead due to being entrapped in OCD rituals.” Sarah’s OCD symptoms are variable and include repetitive movements, turning things on and off, and food obsessions. She says “I just have to do them” and often compels her mother and father to get involved. Prior to this hospitalization, Sarah’s parents had become so engrossed in her rituals that they were only able to sleep 2-4 hours a night. In addition to the OCD, Sarah displays regressive behavior when her mental health decompensates including poor hygiene and inability to use the bathroom without parental help. At the time of admission, Sarah showed marked ambivalence to her situation. It was not until she realized admission was inevitable that she began to cry and show distress.

Obsessive-compulsive disorder (OCD) is a debilitating anxiety disorder estimated to affect 2-3% of the population [1]. It is characterized by recurrent and intrusive thoughts, feelings or ideas (obsessions) and behaviors or mental rituals that serve to ease the distress associated with the intrusions (compulsions). Whereas some patients are driven to wash their hands compulsively out of a fear of contamination, others obsess over numbers and symmetry, and still others devise mental rituals to allay fears of inflicting harm on themselves or others. These repetitive thoughts and behaviors can have a significant impact on the sufferer’s quality of life and ability to function in society. In 2000, the World Health Organization (WHO) ranked OCD as one of the world’s leading causes of disability [2].

Although much progress has been made in understanding the neuroanatomic and neurochemical correlates of OCD in recent years, the precise etiology of this disorder remains unclear. For instance, although fMRI studies have demonstrated structural and functional abnormalities in the basal ganglia (especially caudate), cingulate cortex, and orbitofrontal cortex of patients with OCD, it is not entirely clear how these patterns are established and maintained [3, 4]. Likewise, although there appears to be a strong link between OCD and serotonin, the source of serotonin dysregulation in OCD patients is not fully known. At the present time, the gold-standard treatment for OCD consists of a combination of cognitive behavioral therapy (CBT) and selective-serotonin reuptake inhibitors (SSRIs) [1]. From a cognitive-behavioral point of view, the mechanism underlying OCD is misappraisal of otherwise innocuous thoughts secondary to dysfunctional core beliefs that over-emphasize personal responsibility, the importance of thoughts, and perfectionism [1]. When patients with OCD experience inappropriate thoughts, images or impulses, they attempt to neutralize them by employing fear-based
mechanisms that ultimately increase the frequency and salience of the thoughts over time [5]. With CBT, OCD sufferers learn to recognize the irrationality of their thoughts and to react appropriately to them.

Although cognitive-behavioral therapy and SSRIs have proven effective for many OCD patients, a substantial fraction of OCD sufferers remain refractive to these approaches. Research shows that CBT (specifically Exposure and Response Therapy) is successful in only 50-60% of cases and SSRIs in 40-60% [6]. This has led some researchers to argue that these therapies are not addressing the real root of the problem – that is, how the obsessions and compulsions emerge in the first place. According some authors, the origins of OCD lie deeper than simple misappraisal of innocuous thoughts. In their view, vulnerability to OCD originates in an individual’s earliest childhood experiences and is maintained through dysfunctional cognitive-affective models that are rooted in ambivalent mental schemas of self and others. When this “ambivalent self” is challenged by psychosocial stressors, the result is the clinical disorder known as obsessive-compulsive disorder.

**A Psychoanalytic Approach to OCD**

Freud was one of the first to distinguish OCD (“obsessional neurosis”) from other clinical entities. In his view, OCD was a manifestation of conflict between unacceptable, unconscious sexual or aggressive id impulses and the constraints of conscience and reality [7]. According to Freud, patients with OCD regressed to the anal-sadistic stage of psychosexual development in which the dominant mode of thinking was ambivalence. This led to excessive doubting, magical thinking and irrational ritualistic behavior. When patients attempted to neutralize unacceptable ideas and impulses with ego defenses like intellectualization, isolation, undoing and reaction formation, they were never entirely successful and OCD symptoms arose. Although Freud’s formulation on the origins of OCD has existed in the psychoanalytic literature for more than a century, interest a Freudian approach OCD treatment has dwindled in recent years with advances in neuroscience and psychopharmacology. Nevertheless, many modern psychiatrists argue that psychoanalytically-informed approaches have a great deal to offer patients with OCD.

Although the development and pathogenesis of OCD may in part be influenced by genetics, it has been known for some time that psychosocial factors play an important role
in the etiology of the disorder. Correlations between stressful life events and the onset and exacerbation of existing OCD symptoms are well documented in the psychiatric literature. A study by Buttolph and Holland (1990), for instance, showed that 69% of OCD patients could trace the onset or escalation of their symptoms to pregnancy, child-birth or child-rearing [8]. Likewise, Neziroglu, et al (1992) showed that among female patients suffering from OCD, the onset of symptoms was correlated with pregnancy more that any other life event [9]. For 39% of these patients, birth of their child marked the initial onset of symptoms. Loss of a loved one is another well-known trigger for OCD symptoms, as evidenced by the case report below:

Mr. B was a 38-year-old divorced man who had been who had been successful at a management position until he became unable to function effectively at work because of his obsessional thoughts and compulsive rituals... His obsessions and rituals had dramatically worsened at the time of his father’s death 2 years prior... The patient had been angry at his father the last time he saw him and felt extraordinarily guilty about the fact that their last meeting had been an angry one. The gradually worsening obsessions and rituals [focused mainly on his potential to hurt others] reflected his unconscious conviction that he had caused his father’s death because of the intensity of his anger.

He had initially tried cognitive-behavioral therapy that focused exclusively on his symptoms. He finally had to quit because he felt the cognitive-behavior therapist was not helping. He told me that he had repeatedly explained to this therapist that he was aware of how ridiculous the cognitive distortions were, but the awareness of their irrational nature did not help him change the thoughts [10].

The above examples support the view that OCD symptoms, regardless of how biologically influenced, nonetheless have meanings (conscious or unconscious) to the patient. The psychoanalyst might explain this in terms of OCD serving as a “vehicle” to express psychodynamically based conflicts [10].

Further support for this model is found in studies that explore the effects of culture and religion on OCD. In Egypt, for example, it has been shown that the great majority of compulsive rituals in OCD patients focus on cleanliness and ritual purity [11]. Researchers have suggested that this may be related to the emphasis Islam (practiced by 90% of the population) places on ritualistic prayer and cleansing processes. Likewise, sexual obsessions are among the most frequently reported in females, which is thought to be due to the fact that sexual matters remain an issue of prohibition, sin, uncleanliness and shame for women in Egyptian culture. When compared to studies performed in India, Jerusalem and Britain, significant differences can be found in the content of obsessions in each
country. Whereas patients from Egypt and Jerusalem (Muslim and Jewish, respectively) obsess primarily over issues of religion, cleanliness and dirt, their Indian and British counterparts (Hindu and Christian, respectively) obsess more often over orderliness and aggression or violence. This data supports the idea that OCD is far more complex than can be explained simply by biological model [11].

OCD and Attachment Theory

It is clear that both life events and sociocultural factors influence the course, nature and severity of OCD. But why is it that some individuals develop OCD following traumatic life events or challenging sociocultural conditions and others do not? Doron and Kyrios (2005) argue that early attachment and parenting are key factors in the development and maintenance of obsessive-compulsive disorder [12]. In their view, attachment theory serves as a “bridge” between the biological and the psychological by influencing the development of the child’s understanding of self, others and the world. Early in life, they argue, attachment to the primary caregiver influences the development of internal working models (IWMs) which in turn shape the individual’s subsequent expectations of others and perceptions of themselves. Children whose parents were emotionally available and supportive, for instance, develop a self-model that says “I am loveable and competent” whereas those with emotionally aloof, unreliable caregivers are more likely to consider themselves unlovable and unworthy.

Adult attachment research has shown that IWMs not only influence close, intimate relationships but also play a significant role in psychopathology. Insecure attachment has been linked to depression, anxiety, eating disorders and low self-esteem while secure attachment has been shown to offset the effects of life stressors [13]. What is the relationship between parental attachment and obsessive-compulsive disorder? According to Guido and Liotti (1983), the parenting of children with OCD is characterized by ambivalence. Children receive contradictory signals from their caregiver (e.g. both validation and rejection or love and hate) and in response develop a rigid self-image that tends towards certainty and perfection [14]. A classic example of this would be the parent who shows intense interest in the child’s intellectual and social development but fails to express emotional warmth. For these children, perfectionism and ritualistic behavior help them secure approval and
transform their conflicting self-perceptions into sense of being loveable and worthy. Several studies support this theory of the “ambivalent self” as the underlying vulnerability structure in OCD. Bhar (2001), for instance, showed that individuals with OCD score significantly higher on the Self Ambivalence Measure (SAM) than controls and that ambivalence in specific self-domains – especially morality – is strongly related to developing OCD [15].

The ambivalence of self-domains in OCD is in line with the psychoanalytic approach discussed earlier. From Freud’s point of view, OCD is a result of a constant conflict between feelings of love and hate. An early example of this emotional dissonance can be found in Freud’s famous interpretation of “Rat Man” (1909) in which he described an individual with obsessional neurosis as harboring feelings of both intense affection and powerful disgust towards his father. When Rat Man attempted to defend himself against these hostile impulses by suppressing or stopping the thoughts, he was unsuccessful and OCD ensued. In the context of attachment theory, Rat Man’s strong reaction to aggressive and immoral thoughts about his father would indicate the presence of an ambivalent self-model in which Rat Man is unsure of his worthiness and competence as a moral human being [16].

Although Freud’s analysis of Rat Man has not been without criticism in the hundred or so years since it was published, it is interesting to note that his analysis is nonetheless in line with a number of contemporary studies that make no reference to psychoanalytic or attachment theory. In 2008 Doron, et al investigated the sensitivity of self-beliefs in OCD and found that OCD patients are more likely than anxious controls to draw negative conclusions about themselves from having intrusive thoughts, which they concluded demonstrated the presence of a “feared self” that was bad, immoral or insane [17]. The presence of this “feared” self has important implications for treatment because many patients with OCD delay seeking treatment out of fear that they will be rejected socially if they reveal their obsessions and compulsions.

**Implications for Treatment: A Combined Approach**

From the above discussion, it is clear that both psychoanalysis and attachment theory have much to contribute to our understanding of obsessive-compulsive disorder. But what are the implications for treatment? Should cognitive-behavioral approaches be
abandoned in favor of a psychodynamic approach? The consensus seems to be that the most effective option would be to adopt a model that incorporates aspects of both treatment approaches. Because both psychoanalysis and cognitive-behavioral therapy view obsessive-compulsive disorder as dysfunctional mental representations of “self” and “others” the combined approach would start by bringing attention to these dysfunctional self-schemas and probing the reasons underlying them [18]. Therapists would then challenge the patient’s irrational thoughts and the excessive importance attributed to them and encourage patients to use cognitive-behavioral skills to change their responses. With this approach, ambivalence itself becomes the theme of therapy and progress is marked by integration of the patient’s “contradictory selves”.

The following case study shows how insight-oriented therapy can help patients better understand their experience of OCD and thereby create space between themselves and the disorder:

Dorothy is a 40-year old patient who has suffered from OCD since the age of 5 and has tried CBT numerous times without success…She related in much detail that she couldn’t accept that such impulses existed inside her. They were completely ego-dystonic, and she couldn’t understand why of all persons she, who had been brought up so strictly, and has such high moral standards, was suffering from such thoughts…I suggested to focus on her self-experience, and particularly the experience of “different selves” inside of her…I emphasized she shouldn’t be ashamed of such thoughts because everyone has such thoughts.

After this intervention, gradually she began to talk about her life, and in particular her childhood experiences, and how it felt to grow up with an emotionally cold mother and how she always had longed for the love of a mother which she never really did receive…As Dorothy told me one time in a session towards the end of treatment: “When I did something really well, that was only natural, she didn’t say anything about that, but when I did something wrong, it was as if all hell broke loose [18].”

Conclusion

Although SSRIs and cognitive-behavioral therapy have shown promise in the treatment of obsessive-compulsive disorder, there continues to be a significant fraction of patients who do not improve with these treatments. In most cases, psychiatrists look towards newer and more expensive techniques like electroconvulsive therapy (ECT) and deep brain stimulation (DBS) for these individuals [19]. However, these treatments are highly invasive and carry significant risks. Another option would be to take the approach
outlined above and look at OCD through the lens of attachment theory and psychoanalysis. Although the literature is sparse on the efficacy of psychodynamic approaches for OCD, the above investigation suggests that this would be a worthwhile area of research to pursue. By taking a combined psychodynamic / cognitive behavioral approach, patients will be better able to integrate their experience of OCD into a coherent life narrative and thereby gain more control over what can be a very controlling disease.

Owing to ignorance of the rope the rope appears to be a snake; owing to ignorance of the Self the transient state arises of the individualized, limited, phenomenal aspect of the Self.

– Guru Nanak Dev

References:


Mini Mental State

Patient __________________________
Examiner __________________________
Date __________________________

Score  Orientation
(    )  What is the (year) (season) (month) (date) (day)? (5 points)
(    )  Where are we? (state) (county) (town) (hospital) (floor) (5 points)

Registration
(    )  Name 3 objects: 1 second to say each. Then ask the patient to repeat all three after you have said them. 1 point for each correct. Then repeat them until learned. Count trials and record ___________________________. (3 points)

Attention and Calculation
(    )  Serial 7s. 1 point for each correct. Stop at 5 answers. Or spell “world” backwards. (Number correct equals letters before first mistake; i.e., d l o r w = 2 correct). (5 points)

Recall
(    )  Ask for the objects above. 1 point for each correct. (3 points)

Language Tests
(    )  name - pencil, watch (2 points)
(    )  repeat - no ifs, ands, or buts (1 point)
(    )  follow a 3 stage command: “Take the paper in your right hand, fold it in half, and put it on the floor.” (3 points)

Read and obey the following:
(    )  CLOSE YOUR EYES. (1 point)
(    )  Write a sentence spontaneously below. (1 point)
Copy design below. (1 point)

TOTAL 30 POINTS

The above test does not include abstraction. You may want to test for proverbs and similarities for your own information.
Chloral hydrate: 1832(1869), Bromide sleep therapy; 1980s
Barbiturates (Veronal-Bayer): 1903, then Phenobarbital (Luminal): 1912
Insulin coma therapy: 1933
Lithium: Lithium chloride; salt substitute in 1940’s
J. Cade (Australia) 1949; guinea pigs, then patients
Schou (Europe): 1950’s; US: approval in 1970
Chlorpromazine:
Based on work with antihistamines
4560 RP: 1st use in surgery 1950
Largactil® 1952; Thorazine® 1953
Imipramine; 1957
MAOI’s (iproniazid); early 50’s
Meprobamate (Miltown) 1955
chloridiazepoxide, diazepam; Late 50’s/early 60’s

Suggested reading: Edward Shorter *A History of Psychiatry: From the Era of the Asylum to the Age of Prozac* 1998

### 1. Antidepressants

<table>
<thead>
<tr>
<th></th>
<th>Therapeutic Dosage (mg)</th>
<th>t (hrs)</th>
<th>Sedation</th>
<th>Anticholinergic</th>
<th>α₁ Antag.</th>
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<tbody>
<tr>
<td>Older Agents</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td><em>Imipramine (Tofranil)</em></td>
<td>150-300</td>
<td>10-15</td>
<td>3+</td>
<td>3+</td>
<td>4+</td>
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<tr>
<td>Desipramine (Norpramine)</td>
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<td>20-25</td>
<td>2+</td>
<td>2+</td>
<td>2+</td>
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<tr>
<td><em>Amitriptyline (Elavil)</em></td>
<td>100-300</td>
<td>20-30</td>
<td>4+</td>
<td>4+</td>
<td>3+</td>
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<tr>
<td>Nortriptyline (Aventyl)</td>
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<td>35-45</td>
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<td>2+</td>
<td>1+</td>
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<td>Protriptyline (Vivactil)</td>
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<td>Amoxapine (Ascendin)</td>
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<td>30</td>
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<td><em>Doxepin (Sinequan)</em></td>
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<td>18-30</td>
<td>4+</td>
<td>3+</td>
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<tr>
<td><em>Clomipramine (Anafranil)</em></td>
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<td>20-24</td>
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<td>Nefazodone (Serzone)</td>
<td>300-600</td>
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<td>Trazodone (Desyrel)</td>
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<td>3+</td>
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<td>Newer Agents</td>
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<td>Fluoxetine (Prozac)</td>
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<tr>
<td>Sertraline (Zoloft)</td>
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<tr>
<td>Paroxetine (Paxil)</td>
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<td>15-22</td>
<td>+</td>
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<tr>
<td>Fluvoxamine (Luvox)</td>
<td>100-250</td>
<td>19-22</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Citalopram (Celexa)</td>
<td>20-60</td>
<td>35</td>
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<td>0</td>
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<tr>
<td>Escitalopram (Lexapro)</td>
<td>10-20</td>
<td>34</td>
<td>0</td>
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<td>0</td>
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<tr>
<td>Bupropion (Wellbutrin, Zyban)</td>
<td>100-300</td>
<td>12</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Keep in mind that cyclobenzaprine (Flexeril, Amrix) is very similar to amitriptyline
Side Effects/Adverse Reactions of Tricyclics:

1. Sedation and weight gain: due to anti-histamine effect (H1)
2. Postural hypotension: α1 block
3. Anti-cholinergic effects: dry mouth, blurred vision, constipation, urinary hesitancy, cognitive impairment
4. With overdose: agitation, confusion, memory disturbance, hallucinations, delirium, seizures, cardiac arrhythmia
5. Cardiac toxicity: slowing of conduction
6. Sexual dysfunction

Side Effects/Adverse Reactions of Non-Tricyclics:

1) Trazodone
   a) orthostatic hypotension
   b) sedation
   c) priapism (1 in 8,000)

2) SSRI’s
   a) nausea
   b) insomnia
   c) headache
   d) sexual dysfunction
   e) agitation/anxiety

3) Bupropion (Wellbutrin, Zyban)
   a) seizures: higher doses (maybe less with time release)
   b) agitation/anxiety
   c) insomnia; give in a.m. (XR) or a.m., early p.m.
   (SR)

4) Venlafaxine (Effexor), Duloxetine (Cymbalta)
   a) nausea & headache
   b) blood pressure (higher doses of Effexor)
   c) discontinuation syndrome (also with Paxil)

5) Nefazodone (Serzone)
   a) somnolence
   b) dry mouth
   c) dizziness
   d) rare cases of liver failure*

6) Mirtazepine (Remeron)
   a) Somnolence: strong antihistamine
   b) appetite, weight gain
   c) dizziness

7) Desvenlafaxine (Pristiq)
   a) metabolite of Effexor
   b) maybe less discontinuation problems
   c) cleaner metabolism (not dependent on CYP.2D6 activity)

8) Vilazodone (Viibryd)
   a) partial agonist at 5-HT1A
   b) titrate weekly 10 mg, to 20 mg
   c) take with food (50% difference)

9) Vortioxetine (Brintellix)
   a) Use ½ dose with cyp 206 inhibitors
   b) It is a SSRI along with binding to multiple 5-HT receptors
   c) Nausea is most frequent adverse effect

10) Levomilnacipran (Fetzima)
    a) Dosing 20-120mg
    b) More effect on NE receptors than 5-HT
    c) Nausea is common when started

Other Uses of SSRIs and TCAs: The anxiety disorders (OCD, Panic, GAD, SAD), Bulimia, PMDD, PTSD

2. Stimulants
   1) Increased alertness, mild to moderate euphoria
   2) Used occasionally for:
       a) medically ill depressed
       b) augmentation of other antidepressants

Do not use in patients with cardiac risks (structural abnormalities)
3. M.A.O. Inhibitors:

<table>
<thead>
<tr>
<th>Hydrazine</th>
<th>Dosage</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenelzine (Nardil)</td>
<td>15 to 90 mg/d</td>
<td>All affect MAO A&amp;B; and are irreversible</td>
</tr>
<tr>
<td>Transdermal Selegiline (Emsam)</td>
<td>6, 9, 12 mg/day</td>
<td>No dietary restrictions at lower dose</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Non-Hydrazine</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Tranylcypromine (Parnate)</td>
<td>20-30 mg/d</td>
<td></td>
</tr>
</tbody>
</table>

- 1st one was iproniazid, developed as an anti-tuberculosis drug in 1950’s.
- Monoamine oxidase found primarily in outer membrane of the mitochondria; is the enzyme primarily responsible for the catabolism of biogenic amines.
- MAO metabolizes catecholamines found outside of their storage vesicles in presynaptic nerve terminals.

<table>
<thead>
<tr>
<th>Type</th>
<th>Location</th>
<th>Preferred Substrates</th>
<th>Select Inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>CNS, sympathetic nerve terminals, liver, gut, skin</td>
<td>NE, 5-HT, DA, tyramine, tryptamine</td>
<td>meclobemide**</td>
</tr>
<tr>
<td>B</td>
<td>CNS, liver, platelets</td>
<td>DA, tyramine, tryptamine, phenylethylamine</td>
<td>Selegiline*</td>
</tr>
</tbody>
</table>

*selectivity lost at higher doses (>10 mg/day) **reversible inhibition

Interactions with Foods, Other Drugs:

1) With inhibition of intestinal and hepatic MAO (i.e., type A) vasoactive amines (esp. tyramine) in foods, also phenylethylamine, pseudoephedrine can enter the bloodstream and are taken up by sympathetic nerve terminals, causing a release of endogenous catecholamines.
   - Can result in hyper-adrenergic crisis: severe hypertension, hyperpyrexia, tachycardia, diaphoresis, tremulousness and cardiac arrhythmias.

2) There is a pharmacodynamic interaction between MAOIs and serotonergic agents, such as SSRIs, clomipramine, venlafaxine, etc.
   - This can lead to the “serotonin syndrome” manifested by tachycardia, hypertension, fever, ocular oscillations, and myoclonic jerks. In the severe form, may include severe hyperthermia, coma, convulsions, and death.

*Due to these potential interactions, patients are instructed to avoid a tyramine-rich foods (aged cheeses or meat, fava beans, overripe fruits, yeast or soy, aged red wine, tap beer) along with the serotonergic drugs, bupropion, meperidine (Demerol) and any sympathomimetics (i.e. cold preparations). Need to be off of MAOI for at least two weeks before exposure to drugs listed above.*

4. Mood Stabilizers

Lithium Carbonate

A. Pharmacology
   1. Administered as dibasic salt Li$_2$ CO$_3$
   2. Good p.o. absorption
   3. Distributes in total body water space
   4. Renal excretion, approximately 80% absorbed proximally
   5. Half-life: 10 to 20 hours (increases with age; ↓ GFR)
B. Clinical Use
   1. Acute mania—Li blood level 0.8-1.2 meq/liter
   2. Manic depressive prophylaxis—Li blood level .6 to 1.0 meq/liter (some older people .5); elderly have higher CNS levels vs. serum
   3. Depression prophylaxis
   4. As an antidepressant adjunct (add to SSRI, tricyclic, MAOI)

C. Treatment
   1. Different people require different amounts
   2. Start low, work up following blood levels every 3-5 days
   3. Nighttime dosing is reasonable

D. Problems
   1. Li retention if Na lost (diarrhea, thiazide diuretics), NSAIDs
   2. Dangerous if patient not reliable

E. Side-effects and toxicity:
   1. GI; anorexia, metallic taste, weight gain, n & v
   2. Renal; diabetes insipidus, glycosuria, polyuria, dehydration, renal failure (rare)
   3. CNS; tremor, blurred vision, slurred speech, dizziness, mental dullness, drowsiness, confusion, stupor, coma (with overdose)
   4. N-M; ataxia, weakness, fascication’s, hyperactive reflexes, clonus
   5. Skin-rash, acne
   6. Endocrine; goiter, hypothyroidism (monitor TSH)
   7. Heme: ↑ WBC
   8. Secreted in breast milk; and ? risk of cardiac malformations (pregnancy)

Non-Lithium Mood Stabilizers (anti-epilepsy drugs: AEDs)

Uses
   a. rapid cyclers
   b. Li non-response
   c. dysphoric mania
   d. (although they have emerged as first line therapy)

1. Carbamazepine (Tegretol and Tegretol-XR) (CBZ)
   a. auto-induction
   b. complex drug interactions

<table>
<thead>
<tr>
<th>CBZ:</th>
<th>↑CBZ:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) ↓ antidepressant levels</td>
<td>1) cal - channel blockers</td>
</tr>
<tr>
<td>2) ↓ BZD</td>
<td>2) cimetidine</td>
</tr>
<tr>
<td>3) ↓ contraceptives</td>
<td>3) erythromycin</td>
</tr>
<tr>
<td>4) ↓ neuroleptics</td>
<td></td>
</tr>
<tr>
<td>5) ↓ theophylline levels</td>
<td>↓CBZ:</td>
</tr>
<tr>
<td>6) ↓ valproate levels</td>
<td>1) phenobarbital</td>
</tr>
<tr>
<td>7) ↓ warfarin level</td>
<td></td>
</tr>
</tbody>
</table>

Side Effects/Toxicity:
   1) sedation
   2) anticholinergic
   3) dizziness, ataxia
   4) rash, GI SE’s
   5) hyponatremia
   6) leukopenia
   7) thrombocytopenia
2. Valproate (Depakote and Depakote ER)

Clinical Issues
a) Rapid onset
b) FDA indication for acute BPAD (not maintenance, but used for this)
c) Complex drug interactions
   1) Alcohol \(^{↑}\) CNS depression
   2) Aspirin \(^{↑}\) bleeding time
   3) CBZ \(^{↑}\) Free CBZ and/or epoxide
   4) Dicumerol, warfarin \(^{↑}\) bleeding time
   5) Fluoxetine \(^{↑}\) VPA levels
   6) Phenobarb \(^{↑}\) CNS depression
   7) Phenytoin \(^{↑}\) Phenytoin levels

Side Effects/Toxicity
- Hair loss
- Weight gain
- Tremor
- Nausea
- Thrombocytopenia
- Hepatotoxicity, pancreatitis
- Encephalopathy (also rare)

3. Lamotrigine (Lamictal)
   a) Rash in up to 5%, severe in 0.03%: otherwise very well tolerated
   b) Need to increase dose slowly (especially with Depakote)
   c) FDA indication for maintenance treatment
   d) Probably best for those more depressed than manic

4. Investigational use for BPAD
   a) Gabapentin (Neurontin)
   b) Tiagabine (Gabitril)
   c) Topiramate (Topomax)
   d) Levetiracetam (Keppra)
5. Antipsychotics

Introduction

1. All fairly efficacious
2. Long half-life; can be used once a day (but often not)
3. Wide variation in patient tolerance
4. Symptom specific—can be used for almost any psychosis

A. List of Typical Neuroleptics/Antipsychotics

<table>
<thead>
<tr>
<th>Phenothiazines</th>
<th>Sedation</th>
<th>Anticholinergic</th>
<th>Extrapyramidal</th>
<th>Potency (mg of drug equiv. to 100mg chlorpromazine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aliphatic</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
<td>100</td>
</tr>
<tr>
<td>(chlorpromazine-Thorazine)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aromatic-Piperidine</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
<td>100</td>
</tr>
<tr>
<td>(thioridazine-Mellaril)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aromatic-Piperazine</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>1.5-3</td>
</tr>
<tr>
<td>(fluphenazine-Prolixin)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perphenazine-Trilafon</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Thioxanthenes</th>
<th>Sedation</th>
<th>Anticholinergic</th>
<th>Extrapyramidal</th>
<th>Potency (mg of drug equiv. to 100mg chlorpromazine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aliphatic</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
<td>50</td>
</tr>
<tr>
<td>(chlorprothixene-Taractan)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aromatic-Piperidine</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>2-5</td>
</tr>
<tr>
<td>(thiothixene-Navane)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dihydropindolones</th>
<th>Sedation</th>
<th>Anticholinergic</th>
<th>Extrapyramidal</th>
<th>Potency (mg of drug equiv. to 100mg chlorpromazine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(malindone-Moban)</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>6-10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Butyrophenones</th>
<th>Sedation</th>
<th>Anticholinergic</th>
<th>Extrapyramidal</th>
<th>Potency (mg of drug equiv. to 100mg chlorpromazine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(haloperidol-Haldol)</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>2-5</td>
</tr>
<tr>
<td>(droperidol-Inapsine)</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>10</td>
</tr>
</tbody>
</table>

B. Side Effects

- G.I. (jaundice and ileus), Heme (aplastic anemia), EKG changes, retinopathy (Mellaril), photosensitivity all relatively uncommon
- Prolactin elevation (leading to galactorrhea, amenorrhea, libido changes) due to dopamine block of tuberoinfundibular tract
- Neurologic side effects 1° related to D₂ block
  a. Dystonia
  b. Pseudoparkinsonism
  c. Akinesia
  d. Akathisia
  e. tardive dyskinesia

ATYPICAL/ SECOND GENERATION ANTIPSYCHOTICS: SGAs

1. Antipsychotic effects with less EPS
2. Mesolimbic (psychosis) vs. nigrostriatal (EPS) blockade (refer to figure 1)
3. Serotonin blockade is likely necessary for “atypicality” (5-HT₂A > D₂ blockaid)
### Receptor Affinity Table

<table>
<thead>
<tr>
<th>Receptor</th>
<th>D1</th>
<th>D2</th>
<th>D3</th>
<th>D4</th>
<th>5HT1a</th>
<th>5HT2a</th>
<th>5HT2c</th>
<th>α1</th>
<th>H1</th>
<th>M1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clozapine</strong></td>
<td>85</td>
<td>126</td>
<td>473</td>
<td>35</td>
<td>875</td>
<td>16</td>
<td>16</td>
<td>7</td>
<td>8</td>
<td>1.9</td>
</tr>
<tr>
<td><strong>Olanzapine</strong></td>
<td>31</td>
<td>11</td>
<td>49</td>
<td>27</td>
<td>&gt;10000</td>
<td>4</td>
<td>23</td>
<td>19</td>
<td>7</td>
<td>1.9</td>
</tr>
<tr>
<td><strong>Risperidone</strong></td>
<td>430</td>
<td>4</td>
<td>10</td>
<td>9</td>
<td>210</td>
<td>0.5</td>
<td>25</td>
<td>0.7</td>
<td>20</td>
<td>&gt;10000</td>
</tr>
<tr>
<td><strong>Quetiapine</strong></td>
<td>455</td>
<td>160</td>
<td></td>
<td></td>
<td>2800</td>
<td>31</td>
<td>3500</td>
<td>7</td>
<td>11</td>
<td>120</td>
</tr>
<tr>
<td><strong>Ziprasidone</strong></td>
<td>525</td>
<td>5</td>
<td>7</td>
<td>32</td>
<td>3</td>
<td>0.4</td>
<td>1</td>
<td>11</td>
<td>50</td>
<td>&gt;1000</td>
</tr>
<tr>
<td><strong>Aripiprazole</strong></td>
<td>265</td>
<td>0.45*</td>
<td>0.8</td>
<td>44</td>
<td>4.4*</td>
<td>3.4</td>
<td>15</td>
<td>47</td>
<td>61</td>
<td>&gt;10000</td>
</tr>
<tr>
<td><strong>Haloperidol</strong></td>
<td>210</td>
<td>0.7</td>
<td>2</td>
<td>3</td>
<td>1100</td>
<td>45</td>
<td>&gt;10000</td>
<td>6</td>
<td>440</td>
<td>&gt;1500</td>
</tr>
</tbody>
</table>

*Numbers are dissociation constants (Ki): lower # = higher receptor affinity

*Partial agonist

### SGAs (in order of FDA approval, mostly):

- **Clozapine (Clozaril)**
  1. No T.D., no EPSE, no ↑ in prolactin
  2. Seizures with high doses
  3. Salivation, drowsiness, dizziness, tremor, HA, ↓ BP
  4. Blood dyscrasia (up to 2%) requires weekly WBC at beginning of therapy
  5. Used for:
     - a. Neuroleptic non-response
     - b. T.D. patients

- **Risperidone (Risperdal)**
  1. Dosage = 2-10 mg, optimal probably 4-6 mg
  2. Dose-related EPSE, TD risk low
  3. Addresses positive and negative symptoms
  4. Will ↑ prolactin

- **Paliperidone (Invega)**
  1. Main active metabolite of risperidone
  2. Available in OROS SR formulation
  3. Dose 3-12 mg./day; fewer AEs than risperidone?

- **Olanzapine (Zyprexa)**
  1. Similar S.E.s to clozapine (look at Ki data)
  2. Usual dose 10-20 mg.

- **Quetiapine (Seroquel IR and XR)**
  1. Low potential for EPSE
  2. Need to titrate dose: 50 to 800+ mg
The last 3 approved SGAs (May, 2009 – October, 2010) have fewer problems with weight gain, less potential for adverse metabolic abnormalities.

**Ziprasidone (Geodon)**
1. “Weight neutral”?
2. May be problems with QTc prolongation
3. Need to use at least 80 mg BID vs daily dosing (with food)

**Aripiprazole (Abilify)**
1. Also may be “weight neutral” (but probably not)
2. Partial **agonist** at D2 and 5HT1a receptors
3. Usual dose is 10 to 30 mg
4. Tends to be not sedating, side effects may go down with time

**Iloperidone (Fanapt)**
1. 6-12 mg BID target dose: need to titrate from 1 mg BID due to concern over orthostatic hypotension
2. Use ½ the dose if poor metabolizer at CYP2D6
3. Dose related prolongation of QTc

**Asenapine (Saphris)**
1. Is administered as a sublingual formulation 5-10 mg in PM vs BID
2. Black cherry flavor is better tolerated than regular

**Lurasidone (Latuda)**
1. 40-120 mg per day with food, starting at 20mg for BPAD
2. Only atypical SGA with binding to DA receptors > 5-HT2A receptors

---

**SGAs and metabolic abnormalities**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Weight Gain</th>
<th>Risk for diabetes</th>
<th>Worsening lipid profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clozapine</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Risperidone</td>
<td>++</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>++</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>Aripiprazole*</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Ziprasidone*</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Newer drugs with limited long-term data.

+ = increase effect; - = no effect; D = discrepant results.

All SGAs have a black box warning regarding increased mortality in elderly patients with dementia-related psychosis (Diabetes Care, Volume 27, Number 2, February 2004)
6. Anti-Anxiety Drugs
   • SSRIs
     1. Commonly used for anxious depression: should now be first line treatment
   • SNRIs
     1. Venlafaxine (Effexor) and duloxetine (Cymbalta): FDA indications for GAD and panic
   • Buspirone HCl (Buspar)
     1. Non-BZD anxiolytic; does not impair cognition, motor control
     2. Has shown utility for depression augmentation
     3. Non-addicting
     4. Delay of onset of action (up to a month or so), need to up titrate dose
   • Tricyclic Antidepressants
     1. Pharmacology, other effects—see previous notes
     2. Appear to block panic attacks, particularly associated with agoraphobia
   • Benzodiazepines (also marketed as hypnotics): see table 1
   • Antipsychotics
     1. Effectiveness fairly well-established for anxiety
     2. Clinical evidence is beginning to support their use in anxiety disorders
     3. However, they are expensive, have side effects (EPSE, metabolic, etc.) that may limit their use

7. Sedatives/Hypnotics
   • Sedative Antidepressants; Trazodone
     1. Relatively safe, except for priapism risk
     2. Few sleep stage changes
     3. Tolerance minimal
     4. Bot well studied for sleep but used a lot
   • Sedative Antidepressants; TCAs
     1. Mix of H2 and anticholinergic effects
     2. Cardiac effects, O.D. risk
   • Benzodiazepine hypnotics
     1. Well-established, though dependence/abuse risk
     2. Amnesic, neurological side-effects with higher dosing, short acting (Triazolam > 1 mg)
     3. rebound insomnia
     4. ↓ stage 3, 4, and REM

Non-benzodiazepine hypnotics: bind omega-1 receptor of GABA-BZ complex; maybe lower abuse risk than benzodiazepines and sleep architecture preserved
   • Zolpidem (Ambien and Ambien CR)
     1. Half-life of 2.8 hrs.
     2. No apparent tolerance
     3. Dosage  5 mg = elderly, 10 mg = usual adult
     4. Occasional problems with confusion, agitation
5. Now marketed as sublingual formulation 1.75, 3.5 mg (Intermezzo) for middle of night awakening

- **Zaleplon (Sonata)**
  1. T½ of about 1 hr
  2. May be used for mid-night awakening
  3. Dose 5-20 mg

- **Eszopiclone (Lunesta)**
  1. T½ of 6 hours
  2. 6 month clinical trial
  3. Metabolized by CYP3A4

*All hypnotics have warnings about “complex sleep behaviors” (driving, eating, food, making phone calls, having sex, without remembering the events).

**Others:**
- **Ramelteon (Rozerem)**
  1. Melatonin receptor agonist
  2. 8 mg.; probably best for initial insomnia (not maintenance of sleep)
  3. Watch drug interactions (esp. Luvox)

- **Antihistamines**
  1. Mostly over-the-counter (except hydroxyzine: Vistaril)
  2. Mixed antihistamine and anticholinergic effects
  3. Clinical research doesn’t show significant efficacy (however, most commonly used by the consumer)

---

*The U.S. Food and Drug Administration (FDA) has requested that all manufacturers of sedative-hypnotic drug products, a class of drugs used to induce and/or maintain sleep, strengthen their product labeling to include stronger language concerning potential risks. These risks include severe allergic reactions and complex sleep-related behaviors, which may include sleep-driving. Sleep driving is defined as driving while not fully awake after ingestion of a sedative-hypnotic product, with no memory of the event.*

### 8. Benzodiazepines

<table>
<thead>
<tr>
<th>LONG-ACTING</th>
<th>Dosage Equivalent</th>
<th>Peak Blood Level (hrs)</th>
<th>Half-Life (hrs)</th>
<th>Steady State (days)</th>
<th>Active Metabolites</th>
<th>Half-Life (hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlordiazepoxide* (Librium)</td>
<td>10.0</td>
<td>2 - 6</td>
<td>12</td>
<td>2 - 3</td>
<td>Yes</td>
<td>6 - 96</td>
</tr>
<tr>
<td>Diazepam (Valium)*</td>
<td>5.0</td>
<td>36</td>
<td>36</td>
<td>6 - 9</td>
<td>Yes</td>
<td>6 - 96</td>
</tr>
<tr>
<td>Chlorazepate Monopotassium (Tranxene)*</td>
<td>7.5</td>
<td>Inactive Hydrolyzed</td>
<td>--</td>
<td>--</td>
<td>Yes</td>
<td>6 - 96</td>
</tr>
<tr>
<td>Prazepam (Centrax)*</td>
<td>10.0</td>
<td>6</td>
<td>63</td>
<td>8 - 12</td>
<td>Yes</td>
<td>50 - 96</td>
</tr>
<tr>
<td>Flurazepam (Dalmane)*</td>
<td>30.0</td>
<td>14</td>
<td>10</td>
<td>2 - 3</td>
<td>Yes</td>
<td>50 - 96</td>
</tr>
<tr>
<td>Clonazepam (Klonopin)</td>
<td>0.25</td>
<td>3-4</td>
<td>18-50</td>
<td>4-6</td>
<td>No</td>
<td>------</td>
</tr>
<tr>
<td>SHORT-ACTING</td>
<td>Dosage Equivalent</td>
<td>Peak Blood Level (hrs)</td>
<td>Half-Life (hrs)</td>
<td>Steady State (days)</td>
<td>Active Metabolites</td>
<td>Half-Life (hrs)</td>
</tr>
<tr>
<td>----------------------</td>
<td>-------------------</td>
<td>------------------------</td>
<td>----------------</td>
<td>---------------------</td>
<td>-------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Lorazepam (Ativan)</td>
<td>1.0</td>
<td>2</td>
<td>6 - 15</td>
<td>1 - 3</td>
<td>None</td>
<td>--</td>
</tr>
<tr>
<td>Oxazepam (Serax)</td>
<td>15.0</td>
<td>2</td>
<td>4 - 10</td>
<td>1 - 2</td>
<td>None</td>
<td>--</td>
</tr>
<tr>
<td>Temazepam (Restoril)</td>
<td>30.0</td>
<td>1</td>
<td>8 - 20</td>
<td>1 - 3</td>
<td>None</td>
<td>--</td>
</tr>
<tr>
<td>Triazolam (Halcion)</td>
<td>0.25</td>
<td>1</td>
<td>2.7</td>
<td>1</td>
<td>Unimportant</td>
<td>--</td>
</tr>
<tr>
<td>Alprazolam (Xanax)</td>
<td>0.5</td>
<td>2</td>
<td>6 - 20</td>
<td>2 - 3</td>
<td>Unimportant</td>
<td>--</td>
</tr>
<tr>
<td>Midazolam (Versed)</td>
<td>----</td>
<td>30 min</td>
<td>1.5</td>
<td>1</td>
<td>Unimportant</td>
<td>--</td>
</tr>
<tr>
<td>Estazolam (Prosom)</td>
<td>2.0</td>
<td>2</td>
<td>8 - 12</td>
<td>1 - 2</td>
<td>Unimportant</td>
<td>--</td>
</tr>
<tr>
<td>Quazepam (Doral)</td>
<td>15.0</td>
<td>2</td>
<td>20 - 40</td>
<td>2 - 3</td>
<td>Yes</td>
<td>50 - 160</td>
</tr>
</tbody>
</table>

**Figure 1**

Principle Dopaminergic Tracts

The four major dopamine tracts: (1) nigrostriatal tract—also known as the extrapyramidal pathway, begins in the substantia nigra and ends in the caudate nucleus and putamen of the basal ganglia; (2) mesolimbic tract—originates in the midbrain tegmentum and innervates the nucleus accumbens and adjacent limbic structures; (3) mesocortical tract—originates in the midbrain tegmentum and innervates anterior cortical areas; and (4) tuberohypophyseal tract—projects from the arcuate and periventricular nuclei of the hypothalamus to the intermediate lobe of the pituitary and to the portal blood system surrounding the anterior pituitary.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Daily Dosage</th>
<th>Anticholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Sexual Dysfunction</th>
<th>GI Effects</th>
<th>Agitation / Insomnia</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline</td>
<td>T, IM</td>
<td>100 – 300 mg/d</td>
<td>Very High</td>
<td>Very High</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td>Other common uses: chronic pain, hypnotic</td>
</tr>
<tr>
<td>Clomipramine</td>
<td>C</td>
<td>100 – 250 mg/d</td>
<td>Very High</td>
<td>Very High</td>
<td>Very High</td>
<td>Very High</td>
<td>Very Low</td>
<td>None</td>
<td>Approved for OCD; 250 mg daily maximum due to increased risk of seizures</td>
</tr>
<tr>
<td>Desipramine</td>
<td>T, C</td>
<td>100 – 300 mg/d</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Moderate</td>
<td>High</td>
<td>Very Low</td>
<td>Very Low</td>
<td>Slow cardiac conduction and lower seizure threshold; should not be used with MAOIs</td>
</tr>
<tr>
<td>Doxepin</td>
<td>C, L</td>
<td>100 – 300 mg/d</td>
<td>Very High</td>
<td>Very High</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Imipramine</td>
<td>T, C, IM</td>
<td>100 – 300 mg/d</td>
<td>Very High</td>
<td>High</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Nortriptyline</td>
<td>C, L</td>
<td>50 – 200 mg/d</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Moderate</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td>“therapeutic window” plasma level – must be within 50 – 150 ng/mL for efficacy</td>
</tr>
<tr>
<td>Protriptyline</td>
<td>T</td>
<td>20 – 60 mg/d</td>
<td>Very High</td>
<td>Very Low</td>
<td>Moderate</td>
<td>High</td>
<td>Very Low</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>Trimipramine</td>
<td>C</td>
<td>100 – 300 mg/d</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

2,000 mg can be fatal in overdose in adults; all TCAs cause slowed cardiac conduction; all TCAs may lower seizure threshold; some TCAs have established therapeutic plasma levels.

* Few drug-to-drug comparison trials of sexual dysfunction frequency have been done; due to the serotoninergic effects, clomipramine and SSRIs are more likely than other antidepressants to cause delayed ejaculation and anorgasmia.
### Antidepressants: SSRI S

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Daily Dosage</th>
<th>Anticholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Sexual Dysfunction</th>
<th>GI Effects</th>
<th>Agitation / Insomnia</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram</td>
<td>T</td>
<td>20-60 mg/d</td>
<td>none</td>
<td>Very Low</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>Approved for the treatment of generalized anxiety disorder</td>
</tr>
<tr>
<td></td>
<td>L, T</td>
<td>10-20 mg/d</td>
<td>none</td>
<td>Low</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td>Escitalopram</td>
<td>L, T</td>
<td>10-80 mg/d</td>
<td>none</td>
<td>none</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Very High</td>
<td></td>
</tr>
<tr>
<td>Escitalopram</td>
<td>L, T</td>
<td>10-80 mg/d</td>
<td>none</td>
<td>none</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Very High</td>
<td></td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>C, T, L</td>
<td>100-300 mg/d</td>
<td>none</td>
<td>moderate</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Low</td>
<td>Indicated for OCD</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>T</td>
<td>20-60 mg/d</td>
<td>none</td>
<td>none</td>
<td>none</td>
<td>Very High</td>
<td>High</td>
<td>Low</td>
<td>When stopping therapy, a gradual dose reduction is recommended; approved for the treatment of premenstrual dysphoric disorder</td>
</tr>
<tr>
<td>Paxil CR</td>
<td>T</td>
<td>12.5-75 mg/d</td>
<td>none</td>
<td>Very Low</td>
<td>none</td>
<td>Very High</td>
<td>Very High</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>Sertraline</td>
<td>T</td>
<td>50-200 mg/d</td>
<td>none</td>
<td>Very Low</td>
<td>none</td>
<td>Very High</td>
<td>Very High</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Few drug-to-drug comparison trials of sexual dysfunction frequency have been done; due to the serotoninergic effects, clomipramine and SSRIs are more likely than other antidepressants to cause delayed ejaculation and anorgasmia.
### Antidepressants: MAOIs

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Daily Dosage</th>
<th>Anticholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Sexual Dysfunction</th>
<th>GI Effects</th>
<th>Agitation / Insomnia</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenelzine Nardil, Parke-Davis</td>
<td>T</td>
<td>45-90 mg/d</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>None</td>
<td>Avoid high-tyramine foods and sympathomimetic agents to reduce the risk of hypertensive crisis</td>
</tr>
<tr>
<td>Tranylcypromine Parnate, SmithKline Beecham</td>
<td>T</td>
<td>25-50 mg/d</td>
<td>Moderate</td>
<td>None</td>
<td>Very High</td>
<td>High</td>
<td>Very Low</td>
<td>Very High</td>
<td>*Few drug-to-drug comparison trials of sexual dysfunction frequency have been done; due to the serotoninergic effects, clomipramine and SSRIs are more likely than other antidepressants to cause delayed ejaculation and anorgasmia.</td>
</tr>
</tbody>
</table>
## Antidepressants: Others

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Daily Dosage</th>
<th>Anticholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Sexual Dysfunction</th>
<th>GI Effects</th>
<th>Agitation / Insomnia</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxapine</td>
<td>Asendin, Lederle;</td>
<td>T</td>
<td>200-600 mg/d</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>None</td>
<td>None</td>
<td>Although not an antipsychotic, amoxapine has substantial neuroleptic activity and can cause tardive dyskinesia</td>
</tr>
<tr>
<td></td>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bupropion</td>
<td>T</td>
<td>150-450 mg/d</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Safer in overdose than TCAs; SR formulation offers bid (vs tid) dosing; avoid in patients with seizure disorders</td>
</tr>
<tr>
<td></td>
<td>Wellbutrin, GlaxoSmithKline; Various</td>
<td>T</td>
<td>150-300 mg/d</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wellbutrin SR, GlaxoSmithKline</td>
<td>T</td>
<td>150-300 mg/d</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Duloxetine</td>
<td>Cymbalta, Lilly</td>
<td>C</td>
<td>20-60 mg/d</td>
<td>Low</td>
<td>Low</td>
<td>None</td>
<td>Moderate</td>
<td>High</td>
<td>Do not use with MAOIs, thioridazine, or in the presence of uncontrolled narrow-angle glaucoma; also approved for the management of diabetic neuropathic pain</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>Remeron, Organon</td>
<td>T</td>
<td>15-45 mg/d</td>
<td>None</td>
<td>High</td>
<td>None</td>
<td>None</td>
<td>Very Low</td>
<td>Safer in overdose than TCAs; less sedation at doses&gt;15mg/d; weight gain</td>
</tr>
<tr>
<td>Trazodone</td>
<td>Desyrel, Apothecon;</td>
<td>T</td>
<td>200-600 mg/d</td>
<td>Very Low</td>
<td>Very High</td>
<td>Very High</td>
<td>None</td>
<td>Moderate</td>
<td>Safer in overdose than TCAs; pripism rare (1:1,000 – 1: 10,000); now well tolerated at antidepressant dosage; most commonly used as a hypnotic at 50-200 mg qhs</td>
</tr>
<tr>
<td></td>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>Effexor, Effexor XR,</td>
<td>C (XR), T</td>
<td>75-225 mg/d</td>
<td>None</td>
<td>Low</td>
<td>Very Low</td>
<td>High</td>
<td>Very High</td>
<td>Also indicated for generalized anxiety disorder and social anxiety disorder (XR); do not take with MAOIs hyponatremia may occur</td>
</tr>
<tr>
<td></td>
<td>Wyeth-Ayerst</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2,000 mg can be fatal in overdose in adults; all TCAs cause slowed cardiac conduction; all TCAs may lower seizure threshold; some TCAs have established therapeutic plasma levels.

* Few drug-to-drug comparison trials of sexual dysfunction frequency have been done; due to the serotoninergic effects, clomipramine and SSRIs are more likely than other antidepressants to cause delayed ejaculation and anorgasmia.
# Mood Stabilizers

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Daily Dosage</th>
<th>Therapeutic Plasma Level</th>
<th>Common Adverse Effects</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lithium Carbonate</strong></td>
<td>C, L, T (XR)</td>
<td>1,200 – 2,400 mg/d acute and 900-1,200 mg/d maintenance</td>
<td>0.8 – 1.2 mEq/L. 0.6 – 1.2 mEq/L.</td>
<td>nausea, fine hand tremor, increased urination and thirst, toxicity, slurred speech, confusion, severe gastrointestinal effects, weight gain, acne</td>
<td>Established standard treatment for bipolar disorder; risk of hypothyroidism with maintenance therapy; avoid in pregnancy, especially first trimester</td>
</tr>
<tr>
<td><strong>Carbamazepine</strong></td>
<td>C (XR), Ch, L, T</td>
<td>800-1200 mg/d</td>
<td>4-12 mg/L*</td>
<td>nausea, dizziness, sedation, headache, dry mouth, constipation</td>
<td>Hepatic enzyme inducer; alternative to lithium or valproic acid or adjunctive treatment for bipolar disorder; avoid in pregnancy, especially first trimester</td>
</tr>
<tr>
<td><strong>Gabapentin</strong></td>
<td>C, L, T</td>
<td>900 – 1,800 mg/d divided tid</td>
<td>n/a</td>
<td>dizziness, headache, sedation, tremor, nausea, vomiting, ataxia, rash</td>
<td>No more effective than placebo in acute mania as monotherapy; may help with associated anxiety or sleep difficulty as add-on agent; adjust dosing in renal impairment</td>
</tr>
<tr>
<td><strong>Lamotrigine</strong></td>
<td>Ch, T</td>
<td>100-400 depending on concomitant medication use; start at 25-50</td>
<td>n/a</td>
<td>dizziness, headache, sedation, tremor, nausea, vomiting, ataxia, rash</td>
<td>Clinical data indicate role in depressed phase of bipolar disorder or as adjunctive add-on in acute mania; caution: potential for severe systemic rash; increased risk of rash in children and adolescents with rapid titration, or if given in combination with P450 inhibitors (e.g., valproic acid); titrate dose very slowly with minimal incremental increases every two weeks: may take weeks to get to therapeutic dose</td>
</tr>
<tr>
<td><strong>Oxcarbazepine</strong></td>
<td>L, T</td>
<td>600 – 2,400 mg/d divided bid</td>
<td>n/a</td>
<td>cognitive changes (difficult with concentration, speech); somnolence or fatigue; ataxia and gait disturbances</td>
<td>Significant hyponatremia can develop; start at one half of usual starting dose for patients with renal impairment</td>
</tr>
<tr>
<td><strong>Valproic Acid</strong></td>
<td>T, C, L</td>
<td>500-1800 mg/d</td>
<td>50-100 mg/</td>
<td>nausea, vomiting, diarrhea, abdominal cramps, sedation, tremor, weight gain, thrombocytopenia, increased liver function tests, hair loss</td>
<td>Hepatotoxicity and pancreatitis can occur; avoid in pregnancy; avoid in hepatic disease</td>
</tr>
</tbody>
</table>

*Levels are not established for therapeutic efficacy, but rather are used to monitor for toxicity.*
## Hypnotics

### Benzodiazepines

<table>
<thead>
<tr>
<th>Agent</th>
<th>Daily Adult Dosage Range</th>
<th>tpk (h)</th>
<th>t1/2 (h)</th>
<th>Metabolic Pathway</th>
<th>Active Metabolites</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Estazolam</strong></td>
<td>ProSom, Abbott</td>
<td>1-2 mg/d</td>
<td>2</td>
<td>12-15</td>
<td>oxidation</td>
<td>None</td>
</tr>
<tr>
<td><strong>Flurazepam</strong></td>
<td>Dalmane, Roche: Others</td>
<td>15-30 mg/d</td>
<td>1</td>
<td>40-150</td>
<td>oxidation N-dealkylation</td>
<td>hydroxyethyl-flurazepam flurazepam aldehyde N-desalkyl-flurazepam</td>
</tr>
<tr>
<td><strong>Quazepam</strong></td>
<td>Doral, Wallace</td>
<td>7.5-15 mg/d</td>
<td>2</td>
<td>39</td>
<td>oxidation</td>
<td>2-oxoquazepam</td>
</tr>
<tr>
<td><strong>Temazepam</strong></td>
<td>Restoril, Novartis, Others</td>
<td>15-30 mg/d</td>
<td>1.5</td>
<td>10-15</td>
<td>conjugation</td>
<td>None</td>
</tr>
<tr>
<td><strong>Triazolam</strong></td>
<td>Halcion, Pharmacia &amp; Upjohn</td>
<td>0.125-0.25 mg/d</td>
<td>2</td>
<td>2</td>
<td>oxidation</td>
<td>None</td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.

### Antihistamines

<table>
<thead>
<tr>
<th>Agent</th>
<th>Daily Adult Dosage Range</th>
<th>Onset (min)</th>
<th>Duration (h)</th>
<th>t1/2(h)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diphenhydramine</strong></td>
<td>Benadryl, Parke-Davis; Others</td>
<td>25-100 mg/d</td>
<td>N/A</td>
<td>N/A</td>
<td>3-10</td>
</tr>
<tr>
<td><strong>Doxylamine</strong></td>
<td>Unisom, Pfizer</td>
<td>25-100 mg/d</td>
<td>N/A</td>
<td>N/A</td>
<td>10</td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.
### Omega-1 Receptor Agonists (non-benzodiazepine)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Daily Adult Dosage Range</th>
<th>Onset (min)</th>
<th>Duration (h)</th>
<th>t1/2(h)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zaleplon</td>
<td>5-10 mg/d</td>
<td>30-60</td>
<td>1</td>
<td>1</td>
<td>Very short duration of effect allows dosing during night up to 4 hours before arising</td>
</tr>
<tr>
<td>Zolpidem</td>
<td>5-10 mg/d</td>
<td>30-60</td>
<td>2-4</td>
<td>4</td>
<td>Very short duration of effect allows dosing during night up to 4 hours before arising</td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.

### Other

<table>
<thead>
<tr>
<th>Agent</th>
<th>Daily Adult Dosage Range</th>
<th>Onset (min)</th>
<th>Duration (h)</th>
<th>t1/2(h)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Chloral Hydrate</em></td>
<td>500-2,000 mg/d</td>
<td>30</td>
<td>N/A</td>
<td>8</td>
<td>Loses hypnotic efficacy within first week; gastrointestinal irritation</td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.
### Anxiolytics

#### Benzodiazepines

<table>
<thead>
<tr>
<th>Agent</th>
<th>Schedul  e</th>
<th>Approved Indications</th>
<th>Approved Oral Adult Dosage Range</th>
<th>Approximate Equivalent Doses</th>
<th>Dosage Forms</th>
<th>Onset (PO)</th>
<th>t1/2</th>
<th>Metabolic Pathway</th>
<th>Active Metabolites</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alprazolam</strong></td>
<td>I.V.</td>
<td>anxiety disorders; panic disorder</td>
<td>0.75-4 mg/d; 1.5-10 mg/d</td>
<td>0.5 mg/d</td>
<td>T</td>
<td>Fast</td>
<td>Intermed</td>
<td>Oxidation</td>
<td>alpha-hydroxy-alprazolam</td>
<td>Anterograde amnesia; tid-quid dosing necessary</td>
</tr>
<tr>
<td><strong>Chlordiazepoxide</strong></td>
<td>I.V.</td>
<td>anxiety; alcohol withdrawal; preoperative sedation</td>
<td>5-300 mg/d</td>
<td>10 mg/d</td>
<td>T, C, I</td>
<td>Fast</td>
<td>Long</td>
<td>N-dealkylation</td>
<td>desmethyldiazepamoxide, demoxepam, desmethyldiazepam, oxazepam</td>
<td>Variable bioavailability with IM dosing</td>
</tr>
<tr>
<td><strong>Clonazepam</strong></td>
<td>I.V.</td>
<td>seizure disorders; panic disorder</td>
<td>1-6 mg/d</td>
<td>0.25 mg/d</td>
<td>T</td>
<td>Very Fast</td>
<td>Long</td>
<td>Oxidation</td>
<td>desmethyldiazepam, oxazepam</td>
<td>Anterograde amnesia; adjunctive for bipolar disorders and psychoses</td>
</tr>
<tr>
<td><strong>Clorazepate</strong></td>
<td>I.V.</td>
<td>anxiety; seizure disorders; alcohol withdrawal</td>
<td>7.5-90 mg/d</td>
<td>7.5 mg/d</td>
<td>T, C</td>
<td>Very Fast</td>
<td>Long</td>
<td>Oxidation</td>
<td>desmethyldiazepam, 3-hydroxydiazepam, oxazepam</td>
<td>Variable bioavailability with IM dosing</td>
</tr>
<tr>
<td><strong>Diazepam</strong></td>
<td>I.V.</td>
<td>anxiety; alcohol withdrawal; muscle spasm; preoperative sedation; status epilepticus</td>
<td>2-40 mg/d</td>
<td>5 mg/d</td>
<td>T, SR, L, I</td>
<td>Fast</td>
<td>Long</td>
<td>Oxidation</td>
<td>desmethyldiazepam</td>
<td>Reliable bioavailability with IM dosing</td>
</tr>
<tr>
<td><strong>Lorazepam</strong></td>
<td>I.V.</td>
<td>anxiety; preoperative sedation</td>
<td>0.5-10 mg/d</td>
<td>1 mg/d</td>
<td>T, L, I</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Oxazepam</strong></td>
<td>I.V.</td>
<td>anxiety disorders; alcohol withdrawal</td>
<td>30-120 mg/d</td>
<td>15 mg/d</td>
<td>T, C</td>
<td>Slow</td>
<td>Short</td>
<td>Conjugation</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.
**Others**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Schedule</th>
<th>Approved Indications</th>
<th>Approved Oral Adult Dosage Range</th>
<th>Approximate Equivalent Doses</th>
<th>Dosage Forms</th>
<th>Onset (PO)</th>
<th>t1/2</th>
<th>Metabolic Pathway</th>
<th>Active Metabolites</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Buspirone</strong>&lt;br&gt; BuSpar, Bristol-Myers Squibb</td>
<td>No</td>
<td>anxiety</td>
<td>15-60 mg/d</td>
<td>5 mg/d</td>
<td>T</td>
<td>0.5-1.5 h</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>No sedation, no dependence; not useful on prn basis; nausea, dizziness, agitation</td>
</tr>
<tr>
<td><strong>Hydroxyzine</strong>&lt;br&gt; Atarax, Pfizer; Vistaril, Pfizer; Others</td>
<td>No</td>
<td>anxiety; pruritus; preoperative and postoperative sedation</td>
<td>50-400 mg/d</td>
<td>N/A</td>
<td>T, C, L, I</td>
<td>2 h</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Anticholinergic; sedation</td>
</tr>
</tbody>
</table>

Avoid alcohol and other CNS depressants with these agents (except buspirone); drowsiness may impair ability to drive. Use caution.
# Antipsychotics

**Typical—Dopamine receptor antagonists**

<table>
<thead>
<tr>
<th>Class</th>
<th>Agent</th>
<th>Dosage Designs</th>
<th>Dosage Equivalents (mg)</th>
<th>Extrapyramidal Side Effects</th>
<th>Anti-cholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>PHENOTHIAZINES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piperazine</td>
<td>Mesoridazine</td>
<td>L, T</td>
<td>50</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>Patients on oral thioridazine who need injectable form may receive mesoridazine injection; ECG changes</td>
</tr>
<tr>
<td></td>
<td>Chlorpromazine</td>
<td>I, L, T</td>
<td>100</td>
<td>Moderate</td>
<td>Moderate</td>
<td>High</td>
<td>High</td>
<td>Allergic dermatitis; photosensitivity; ECG changes</td>
</tr>
<tr>
<td>Butyrophenone</td>
<td>Haloperidol</td>
<td>I, L, T</td>
<td>2</td>
<td>Very High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td>Thioxanthene</td>
<td>Thiothixene</td>
<td>C</td>
<td>4</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fluphenazine</td>
<td>I, L, T</td>
<td>2</td>
<td>Very High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perphenazine</td>
<td>I, T</td>
<td>8</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trifluoperazine</td>
<td>I, T</td>
<td>5</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Piperazine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thioridazine</td>
<td>L, T</td>
<td>100</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td></td>
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<tr>
<td></td>
<td>Piperazine</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mesoridazine</td>
<td>L, T</td>
<td>50</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>Butyrophenone</td>
<td>Haloperidol</td>
<td>I, L, T</td>
<td>2</td>
<td>Very High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td>Thioxanthene</td>
<td>Thiothixene</td>
<td>C</td>
<td>4</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fluphenazine</td>
<td>I, L, T</td>
<td>2</td>
<td>Very High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perphenazine</td>
<td>I, T</td>
<td>8</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trifluoperazine</td>
<td>I, T</td>
<td>5</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Piperazine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thioridazine</td>
<td>L, T</td>
<td>100</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td></td>
</tr>
</tbody>
</table>

61 | Page
**Atypical**

Dopamine and serotonin receptor antagonists; more effective for negative symptoms of schizophrenia (amotivation, affect, isolation); significantly less EPS and lower risk for tardive dyskinesia than with typical antipsychotics.

<table>
<thead>
<tr>
<th>(Class) Agent</th>
<th>Dosage Forms</th>
<th>Usual Adult Dosage Range</th>
<th>Extra-pyramidal Side Effects</th>
<th>Anticholinergic</th>
<th>Sedation</th>
<th>Orthostatic Hypotension</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aripiprazole</strong></td>
<td>Abilify, Bristol-Myers Squibb/Otsuka America</td>
<td>T 10-30 mg/d</td>
<td>Very Low</td>
<td>Very Low</td>
<td>Low</td>
<td>Low</td>
<td>Adverse events include headache, nausea, vomiting, anxiety, insomnia; dosed once daily</td>
</tr>
<tr>
<td><strong>Olanzapine</strong></td>
<td>Zyprexa/Zyprexa Zydis, Lilly</td>
<td>T 10-20 mg/d</td>
<td>Low</td>
<td>Moderate</td>
<td>Moderate/High</td>
<td>Moderate</td>
<td>Monitor blood glucose levels at baseline and periodically; periodic assessment of transaminases is recommended in patients with significant hepatic disease; elevates prolactin levels; approved as a treatment for both acute mania and maintenance treatment in bipolar disorder</td>
</tr>
<tr>
<td><strong>Quetiapine</strong></td>
<td>Seroquel, AstraZeneca</td>
<td>T 400-800 mg/d</td>
<td>Rare</td>
<td>Moderate</td>
<td>Moderate/n/a</td>
<td>Initiating dose at 25 mg bid to reduce orthostatic hypotension; potential for lipid abnormalities; indicated as monotherapy and adjunct therapy with lithium or divalproex for the short-term treatment of acute manic episodes associated with bipolar disorder; indicated for schizophrenia</td>
<td></td>
</tr>
<tr>
<td><strong>Risperidone</strong></td>
<td>Risperdal/ Risperdal/Risperdal Consta, Janssen</td>
<td>T 2-8 mg/d</td>
<td>Low</td>
<td>Low</td>
<td>Low/Moderate</td>
<td>Low/Moderate</td>
<td>Dosage should be reduced in patients with renal or hepatic disease; indicated for bipolar mania and schizophrenia; initiate therapy at lower doses to avoid orthostatic hypotension; IM dose 25 mg every 2 wk</td>
</tr>
<tr>
<td><strong>Clozapine</strong></td>
<td>Clozaril, Novartis, various</td>
<td>T 300-900 mg/d</td>
<td>Very Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>WBC monitoring required due to risk of agranulocytosis; dose related seizure risk; little or no prolactin elevation; monitor for systems of myocarditis; severe hyperglycemia has been reported</td>
</tr>
<tr>
<td><strong>Ziprasidone</strong></td>
<td>Geodon, Pfizer</td>
<td>T 40-160 mg/d</td>
<td>Very Low</td>
<td>Very Low</td>
<td>Low to Moderate</td>
<td>Low to Moderate</td>
<td>Avoid use with other drugs known to prolong the QTc interval</td>
</tr>
</tbody>
</table>

5 mg, 10 mg, 15 mg, 20 mg, 30 mg
I. Objectives

A. To recognize a number of psychometric techniques commonly used in assessing cognitive and personality characteristics and functioning of psychiatric patients.

B. To be aware of some of the strengths and weaknesses of psychometric techniques in the assessment of psychological functioning.

C. To recognize some of the psychometric signs of psychopathology and/or cognitive dysfunction.

D. To understand how psychometric data can supplement other kinds of medical and psychiatric data in the diagnosis of mental disorders.

II. Neuropsychological Techniques - Cognitive Functioning

A. Intellectual and neuropsychological evaluations are not synonymous.

B. Indications for evaluation vary and should be explicitly stated.

1. Cognitive assessments may be helpful in questions involving diagnosis.

2. Increasingly, the value of cognitive assessments resides with treatment and disposition recommendations.

C. Intellectual evaluations usually use a form of the Wechsler scales.

1. Advantages of the WPPSI, WISC, and WAIS format include factoring the Full Scale IQ into Verbal and Performance (nonverbal) components as well as in providing a subtest profile.

2. A single scale or test is not adequate to make distinctions about impairment.

D. Neuropsychological evaluations may be battery oriented or problem oriented.

1. Halstead-Reitan, Luria-Nebraska, and eclectic methodologies are the three main assessment approaches used in this country.

2. A comprehensive neuropsychological evaluation examines the various domains of cognitive functioning including orientation and attention, perception and receptive ability, memory, verbal conceptual ability, nonverbal conceptual ability, and executive/expressive functioning.

E. Some caveats to consider in requesting and using information from a cognitive evaluation include issues related to the service provider, the nature of the evaluation per se, and the myriad of possibly relevant subject variables.
III. Projective Techniques

A. Types
   1. Rorschach
   2. Thematic Apperception Test (TAT)
   3. Projective Drawings

B. Uses of Projective Tests
   1. Forensic evaluations
   2. Complete psychological evaluations
   3. Therapy recommendations

IV. The Mmpi

A. Rationale for the MMPI
   1. To identify patients likely to have psychological or psychiatric problems.
   2. To assist in differential psychiatric diagnosis.

B. Method of MMPI Scale Development
   1. Empirical, using carefully diagnosed psychiatric patients as criterion groups.
   2. Intuitive and Statistical (the validity scales).

C. Methods of MMPI Profile Interpretation
   1. Actuarial: for the most commonly occurring profile types.
   2. Empirical: based on research identifying the major psychological or psychiatric correlates of scale scores and profile patterns.

D. Strengths and Weaknesses of the MMPI
   1. A convenient screening test - a wealth of information with a minimum of professional time expenditure.
   2. A useful supplement to the Mental Status Interview - may pick up problems not revealed in a face-to-face interview.
   3. Useful in evaluating response to treatment (before and after comparisons).
   4. Ten to twenty percent false positives/negatives.
   5. Moderately susceptible to conscious bias (“faking”).
Introduction

Attitudes toward psychiatry and psychiatric patients are often enhanced when students develop competence in interviewing patients about sensitive issues. The following psychiatric “screens” are useful in general medical practice. The psychiatry clerkship would be an appropriate time for students to learn these (or other) sets of questions. Some screens may be more appropriate in different clinical sites such as psychiatric emergency departments, consultation services or outpatient departments. However, the sites where they are practiced are less important than the fact that students achieve familiarity and comfort with using them.

Psychiatric Work-Up

Seven questions should be asked about any psychiatric patient:

1. Why is the patient here now?
2. What does the patient want/expect?
3. Is a general medical illness contributing to the patient’s difficulties?
4. How lethal is the situation?
5. In what ways are the patient’s relationships helping or exacerbating the problem?
6. What are the patient’s cultural expectations/explanations/treatments for their illness?
7. What is the psychiatric diagnosis?

Alcohol And Drug Abuse Screen

Have you ever had a drinking or drug problem?
(Yes: 70% of alcoholics, 1% of nonalcoholics; JAMA 259:51, 1988.)

Has anyone else ever worried that you had a drinking or drug problem?

Did you ever use sleeping pills, weight loss medication or painkillers?

CAGE questions (JAMA 252:1905, 1984)

C - Have you ever felt you ought to cut down on your drinking?
A - Have people Annoyed you by criticizing your drinking?

G - Have you ever felt bad or Guilty about your drinking?

E - Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover -- an Eye opener?

(A positive answer on two or more will identify the majority of people with alcohol abuse or dependence)

When is the last time you used any tobacco?

How much are you using now (were you using then)?

Have you used any other forms of tobacco (chew, cigarettes, cigars, pipes)?

____________________________________________________________________________________

Anxiety Disorders Screen

Do you ever feel nervous or tense?

Have you ever felt extremely frightened, physically uncomfortable, or worried that something terrible was going to happen?

If yes: Tell me about that.

Did you expect to feel that way?

Are there situations or activities that cause you a lot of anxiety or that you are more afraid of than most people would be?

If yes: What happens when you ____________________________?

Do you avoid that/those situations/activities?

Do you worry a lot or have trouble getting things off your mind?

If yes: What do you worry about?

What do you have trouble getting off your mind?

Is there anything you have to do over and over again and cannot stop yourself from doing?

If yes: Tell me about that.

____________________________________________________________________________________

Depression Screen

How would you describe your mood?

A. In the past month, have you felt down, depressed, or hopeless most of the day nearly every day?
If yes:  Describe what that is like for you.
Do you feel that way now?
How long have you felt depressed?

If no: When did you last feel down, depressed or hopeless?
How long did you feel depressed?

B. Have you lost interest or pleasure in doing things you used to enjoy?

If yes:  What do you usually enjoy doing?
When was the last time you did one or more of those things?
Was it enjoyable?
How long have you had difficulty getting interested in or enjoying activities?

If no: What do you enjoy doing?
When was the last time you did one or more of those things?

If A or B is positive:

Sleep, increase or decrease
Interest (previously determined)
Guilt, hopelessness, helplessness
Energy, decreased
Concentration, decreased
Appetite, increased or decreased
Psychomotor, retardation or agitation
Suicidality, active vs. passive

Hypomania Screen

Have you had periods of needing very little sleep and not feeling tired?

Has anyone ever worried that you were excessively happy or so energetic that you were not your normal self?

Have your thoughts ever raced and you could not control them?

Have you ever had periods of greatly increased energy when you felt you could accomplish almost anything?

Have you ever had periods of thrill seeking when you took physical risks, such as speeding or doing other dangerous things?
Eating Disorders Screen

Have you ever lost or gained weight in the last year? How much?

How many times have you started a diet in the last year?

Have you ever felt that your eating was out of control? Have you gone on eating binges?

Have you ever vomited or spit out food after eating to get rid of it?

Have you ever used diuretics or laxatives? How often?

Have people ever given you a hard time about being too thin?

Family Violence Screens

A. CHILD ABUSE (MODIFY FOR MALE PERPETRATORS)

How did you feel during your pregnancy?

Has your child lived up to your expectations?

At what age do you think children know right from wrong?
(Complain about unrealistic expectations of children)

How do you feel when your child behaves badly? What do you do?

Is there anyone you can turn to for help?

Have you ever been concerned that anyone would hurt your child?

Have you ever been frightened with thoughts of hurting your child?

Have you or anyone else hurt your child?

B. SEXUAL ABUSE VICTIMS

Are there things going on in your home that you are uncomfortable with or ashamed to talk about?

Has there been any sexual contact between family members in your home besides your parents?

Have you been involved sexually with any adult, including either of your parents?
C. PARTNER/ELDER ABUSE VICTIMS

I know that you may be ashamed of what happened (or might have happened), but could it be that this injury did not happen by accident?

Is your family under a lot of stress?

What happens when you and your partner argue?

Do either of you have trouble with your temper?

Have you ever fought physically with your partner? How badly have you been hurt?

Is there a weapon in the house?

Are you afraid to go home?

D. ABUSE HISTORY

Did you ever witness any violence in your home when you were growing up?

How were you disciplined as a child?

Were you ever physically hurt by a family member?

During your childhood or adolescence, did a relative, family friend or stranger ever touch your body, or have you touch them in a sexual way?

Did anyone attempt or succeed in having sexual intercourse with you?

Did you ever have an unwanted sexual experience of any kind?

---

Psychosis Screen

Have you ever had trouble with your thinking?

Has your thinking ever been so confused that you lost track of your ideas?

Have any of your thoughts seemed frightening or disturbing to you?

Have you ever felt like people were watching or following you, or that they wanted to hurt you?

Have your eyes or ears ever played tricks on you?

Have you ever had the experience of hearing a voice when nobody else was around, or of seeing things that weren’t there?
A. GENERAL SCREEN

Are you sexually active at the present time?
   If no - have you ever been?

Are (were) your partners men, women or both?
   If both - which do you prefer?

What means of birth control do you (have you) use(d)?
   Ask both males and females

Do you have any concerns or problems with your sexual life?

Have there been any changes in your sexual activity?
   Changes in level and frequency of interest?
   Changes in type of interest?
   Do you or have you ever engaged in anal intercourse?
   Are there any ways in which you would like your sexual life to be different?

Have any bad or frightening things ever happened to you sexually? For example: rape, sexual abuse or molestation? (See Abuse Screen)

Have you had any sexually transmitted diseases such as herpes, chlamydia, gonorrhea, syphilis or AIDS? (See HIV Screen)

Have you ever been treated for a sexually transmitted disease?

B. HIV RISK FACTORS

Do you worry about getting AIDS? Why? or Why not?

Do you practice safe sex? (Explain)

Have you ever injected (or shot up) drugs into your veins?

(If male) Have you ever had sexual contact with another man or with someone who used I.V. drugs?

(If female) Have you ever had sexual contact with someone who was bisexual or someone who used IV drugs?

How many sexual partners have you had in the last ten years?

Have you ever needed a blood transfusion? What year? (1979-1985 is risk period)
Sleep Disorders Screen

Are you comfortable with your sleep pattern?

Are you excessively tired during the day?

Does your bed partner complain about your sleep pattern?

---

Suicide And Violence Screen

Have you ever had thoughts that life is not worth living?

Have you ever had thoughts of killing yourself? (Now?)

How would you do it?

Have you taken steps to carry out your plan? (Collected weapons, pills, etc.)

Patients who are suicidal may also be homicidal and vice versa, so ask:

Have you ever had thoughts of hurting anyone else? (Now?)

Have you ever hurt anyone else?

What plans do you now have to hurt anyone?

---

Trauma Screen

Have you ever had anything happen to you where you thought you would be seriously injured or might die?

Have you ever been in a life threatening accident? Fire? Disaster?

Have you ever been attacked or raped?

Have you ever seen these things happen to someone else?
Sheila Specker, M.D.

**Definition**

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), “the essential feature of a **substance use disorder** is a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems.” It goes on to state that the “diagnosis of a substance use disorder can be applied to all 10 classes…except caffeine.”

Diagnostic criteria for Substance Abuse according to DSM-5 are:

**Impaired Control**

1. The individual may take the substance in larger amounts or over a longer period than was originally intended.
2. The individual may express a persistent desire to cut down or regulate substance use and may report multiple unsuccessful efforts to decrease or discontinue use
3. The individual may spend a great deal of time obtaining the substance, using the substance, or recovering from its effects
4. Craving

**Social Impairment**

5. Failure to fulfill major role obligations at work, school, or home
6. Continue substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance
7. Important social, occupational, or recreational activities may be given up or reduced because of substance use, withdrawing from family activities and hobbies in order to use the substance

**Risky Use**

8. Substance use in situations in which it is physically hazardous
9. Continue substance use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.

**Pharmacological Criteria**

10. Tolerance, which is signaled by requiring a markedly increased dose of the substance to achieve the desired effect or a markedly reduced effect when the usual dose is consumed.
11. Withdrawal, which occurs when blood or tissue concentrations of a substance decline in an individual who had maintained prolonged heavy use of the substance.
Polysubstance abuse is not a diagnosis. Individual substances should be listed instead through multiple substances of abuse. Clinical judgment must still be applied regardless of dosage. For example, mixed drug abuse, mental retardation, neurocognitive disorders, other psychiatric conditions, or extreme youth or old age may lead to drug-related problems at lower doses. Even relatively mild psychoactive compounds, such as caffeine, can lead to disabling symptoms in sensitive patients in large doses. Episodes of opioid or cocaine overdose, amphetamine delusional disorder, phencyclidine delirium, or cannabis delusional disorder also exemplify other types of pathological drug use.

Different drugs produce withdrawal symptoms as follows:

1. **Opioids**: lacrimation, rhinorrhea, mydriasis, piloerection, sweating, diarrhea, yawning, mild hypertension, tachycardia, fever, insomnia.

2. **Sedatives** (including alcohol): nausea, vomiting, malaise, weakness, tachycardia, sweating, hypertension, anxiety, depressed mood or irritability, orthostatic hypotension, coarse tremor.

3. **Amphetamines**: fatigue, disturbed sleep, increased dreaming.

4. **Tobacco**: craving, irritability, anxiety, difficulty concentrating.

Onset of withdrawal symptoms following the last dose varies with the drug’s duration of action. Withdrawal can begin in four to six hours with short acting drugs (e.g., heroin, morphine), in eight to sixteen hours with intermediate acting drugs (e.g., oxycodone, alprazolam, alcohol), or in a few to several days with long acting drugs (e.g., diazepam, methadone).

Other substance related conditions include substance intoxication, substance withdrawal, substance induced delirium, persisting dementia, persisting amnestic disorder, psychotic disorder, mood disorder, and anxiety disorder.

**Etiology And Pathogenesis**

Other than excessive or problematic drug use as a final common pathway, there is no one cause of drug abuse. Rather, it is multifactorial in its etiology and has biologic, genetic, psychosocial, and environmental influences.

**Individual Factors**

Family/Genetic Influences

Like many other psychiatric disorders, drug abuse tends to reoccur within the same family. This suggests that genetic factors may play a role in drug abuse. Offspring of heavy tobacco smokers are considerably more apt to become tobacco dependent than the general population (Sarvik et al 1977, Krasnagor 1979). Opium dependent persons in Asia show a higher rate of opium dependence among their siblings and relatives than does the general population (Westermeyer 1974). Similarly, drug dependent persons in the United States often have alcoholic relatives as
well as depressed or manic relatives. Thus far, adoptive and twin studies have indicated that alcoholism is genetically transmitted to some extent; however, the extent to which family prevalence is due to genetics and/or environment is not known.

**Neurotransmitters**

Specific opiate receptors exist in several areas of the brain, including the limbic system. Endogenous morphine-like substances, called endorphins, have been shown to exist in several mammalian species. Opioid drugs probably interact with this system directly, thereby replacing endogenous or host rewards (e.g., from food, social approval, sex, exercise) with exogenous or drug rewards. This same mechanism may also operate for other drugs. For example, condensation products of alcohol metabolism with dopamine include tetra-hydroisoquinolines (TIQ’s) which are also the intermediary products of morphine in opium poppy (Simon 1980).

Benzodiazepine receptors in the brain have also been demonstrated. These probably play a role in sedative abuse. Like other biological systems, these may be influenced by genetic as well as environmental factors.

Elucidation of these neurotransmitters and their locus of action in the brain has contributed much to current thinking regarding drug abuse. It has helped us to understand why drug dependent persons often ignore other personal and social needs to seek drug induced effects. As the drug abusing person becomes increasingly reliant on drugs as a means of functioning and enjoying life, other means for enhancing life diminish. Consequently the drug dependent person pays less attention to food, exercise, work, recreation, friends, and family.

**Psychological Variables**

Psychological and personality variations usually accompany drug abuse, although it is difficult to ascertain whether these are primary and etiologic, or secondary to drug abuse. Factors which initially lead a person to start drug use may change over time, so that the original causes may be replaced by different or altered factors which govern continued or increased drug use. Most clinicians and researchers agree that no one personality type predates drug abuse, although those with chronic pain, anxiety, depression, impulsiveness, and/or antisocial attitudes appear to be at greater risk. Personality characteristics of drug abusers, perhaps as much acquired as primary, typically include impulsivity, suppression of emotion, anxiety or anger in interpersonal relationships, low frustration tolerance, limited flexibility and adaptiveness, and low self-esteem. Some individuals use drugs as a medicine such as to reduce anxiety or depression, treat pain, or insomnia while others use drugs for their euphoric properties. The “anxiety reduction” theory states that some people take drugs initially to reduce tension, especially in social settings.

**Agent or Drug Factors**

**Pharmacological Considerations**

Pharmacological properties of drugs themselves affect their propensity to be abused. Opioids and sedatives produce rapid, albeit temporary, relief of anxiety, fear, and insomnia. Stimulants relieve boredom, somnolence, anergy, and fatigue. Drugs altering perceptions may aid in
blocking out undesirable thoughts or feelings. Physiological symptoms relieved by drugs of potential abuse include pain, nausea, vomiting, cramps, diarrhea, and coughing.

Drugs with more rapid onset of action and briefer actions (e.g., heroin, cocaine, methamphetamine) are more addictive over more delayed, longer acting drugs (e.g., methadone, phenobarbital).

Modes of administration also govern rate of drug effect and thereby the liability for drug abuse. Intravenous, smoking, and sniffing produce faster drug effects than ingestion.

Tolerance and withdrawal phenomena also contribute to drug abuse syndromes. Tolerance relates to the need for increasing doses to have the same effect. It is particularly characteristic of opioids and sedatives, but also has been observed with stimulants, cannabis, and tobacco. Cessation of drug use in the tolerant individual precipitates withdrawal, which in its acute phase, usually persists days or weeks depending on the drug. Subclinical abstinence symptoms can continue for months in the chronic phase of withdrawal. These chronic abnormalities, best described for opioid drugs, consist of altered sleep patterns, vital signs, and endocrine functions which may persist for up to a year. Anxiety symptoms, panic attacks, irritability, suspiciousness, low pain tolerance, depressive symptoms, and sometimes manic symptoms may persist for several weeks to several months following abstinence. Stimulant withdrawal tends to be marked by fatigue, hyperphagia, bradycardia and somnolence. Sedative and opioid withdrawal are accompanied by weakness, anorexia, tachycardia, agitation, and insomnia. Irritability, social withdrawal, and remorse may occur with either category of drug. In the chronic stages of drug dependence, drug usage is often continued more to avoid the withdrawal syndrome than to obtain the acute effects of the drug.

Availability

As availability of a drug increases in the environment, the prevalence of its use tends to increase (Hughes 1972). Availability of licit drugs (such as tobacco) may be governed by such factors as distance between sales outlets, hours of sale, and restrictions on sale to minors. In the case of prescribed drugs, availability may be largely due to prescribing habits among physicians. The greatly increased use of benzodiazepines in the late 1960s and 1970s, and their waning use in the 1980s, has hinged largely on physician prescribing practices. This has also formerly been true of amphetamine prescribing, which was prevalent during the 1950s and 1960s in many countries including the United States (Smart 1980).

The cost of illicit drugs also influences their use. As price increases, drug use tends to decrease even if availability is held constant. This is one argument for drug prohibition laws, which often increase the cost of drugs considerably (since they are illicit) but may not greatly reduce availability. Both price and availability affect society-drug interactions.

Environmental or Social Factors

Cultural Factors in Drugs of Abuse

Cultures that effectively prohibit or preferentially ignore certain drugs have little or no problems with them. For example, alcohol abuse is rare in certain Muslim nations which forbid beverage
alcohol for religious reasons. Certain Middle Eastern countries have economic problems with widespread importation of khat, a stimulant somewhat stronger than caffeine but weaker than amphetamines. It is a substance of abuse in Minneapolis in certain ethnic groups (e.g. Somalis).

Patterns of use for a particular drug determine the likelihood that the drug will be associated with abuse. Problematic use is more apt to attend non-ritual use in which some (but not all) adolescents are introduced to the drug by slightly older adolescents away from family and in a surreptitious fashion with intoxication as a goal. Use without abuse is more apt to occur when all children or adolescents in the society are introduced to the drug experience in a family-sponsored, multi-generational, socially approved setting with ritual feasting and celebration.

**Drug Laws**

Drug laws and policies focusing on the importation of drugs have been ineffective strategies to decrease addiction. Certain substances (e.g. cocaine) may carry greater penalties for selling and possession possibly related to the societal and federal pressures. Certain states (Minnesota included) have enacted laws requiring physicians to report use of certain substances during pregnancy. While this may help the women obtain more services, it may also discourage drug addicted pregnant women from seeking care.

**Epidemiology**

**Rates of Drug Abuse**

Rates of drug abuse often fluctuate widely over time and from place to place in the United States. There have been several opioid “epidemics” in the twentieth century, especially during and after war actions. An amphetamine “epidemic” occurred in the late 1950s and early 1960s. Tobacco dependence has increased progressively over the last century, first among men and more recently among women and is now decreasing. Cannabis abuse increased markedly during the late 1960s and did not begin to decline until the early 1980s. Cocaine abuse and dependence continued at stable levels during the 1970s, increased dramatically in the early 1980s, and showed some decline in the late 1980s. A fairly constant population continues chronic cocaine use. Methamphetamine use has increased over the last several years. Prescription opioid and heroin use has increased dramatically in the past several years.

**Demographic Characteristics**

Men are generally affected by drug and alcohol abuse more frequently than women, although there are exceptions. Betel-areca dependence in parts of Asia and sedative abuse in North American and Europe have occurred predominantly among women. In recent years, the rates of tobacco and alcohol dependence have been increasing more among American women than men.

Cannabis is the primary drug of abuse among adolescents presenting for treatment. Heroin, cannabis, tobacco, volatile hydrocarbons, and hallucinogens have had a upsurge among youths. Elderly people have shown may develop problems with alcohol and sedatives, in association
with death of a marital partner, isolation from friends and family in old age residences, major depression, chronic pain, or disabling medical conditions (Cohen 1981).

Socioeconomic variables affect the availability and type of drugs. For example, successful athletes and entertainers have had both the money for, and access to, such drugs as cocaine and heroin.

Healthcare workers are especially liable to abuse opioids and sedatives. Availability of these substances in the work setting is likely a risk factor. Use of the short acting opiates is particularly a problem among anesthetists and anesthesiologists. However, alcohol is still the primary substance of abuse among healthcare workers. Physicians have very high rates of recovery (70-85%), much higher than the general population, likely related to good social stability, risk of license loss, and close monitoring being the standard of care.

Pathology

Pathological consequences from drug abuse vary widely with the drug, dosage, and duration of use. Route of administration is also an important consideration.

Alcohol Use Disorder

The presence of at least 2 of these symptoms indicates an Alcohol Use Disorder (AUD). The severity of the AUD is defined as:

**Mild:** The presence of 2 to 3 symptoms  
**Moderate:** The presence of 4 to 5 symptoms  
**Severe:** The presence of 6 or more symptoms:

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Alcohol is often taken in larger amounts or over a longer period than was intended.</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>2. There is a persistent desire or unsuccessful efforts to cut down or control substance use.</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>4. Craving, or a strong desire or use to use alcohol</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>5. Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home.</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>7. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>8. Recurrent alcohol use in situations in which it is physically hazardous</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>9. Alcohol use is continued, despite knowledge of having a persistent or recurrent physical or psychological problem that</td>
<td>?</td>
<td>No</td>
</tr>
</tbody>
</table>
is likely to have been caused or exacerbated by alcohol.

<table>
<thead>
<tr>
<th>Question</th>
<th>Tolerance</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>10. Tolerance, as defined by either of the following:</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>a. A need for markedly increased amounts of alcohol to achieve intoxication or desired effect.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Markedly diminished effect with continued use of the same amount of alcohol.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Withdrawal, as manifested by:</td>
<td>?</td>
<td>No</td>
</tr>
<tr>
<td>a. At least two substance-specific withdrawal symptoms/signs, or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Taking the substance to relieve or avoid withdrawal symptoms.</td>
<td></td>
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</tbody>
</table>

Alcohol

The second most commonly abused drug next to nicotine is alcohol. The clinician should ask precisely how many beers, wine, wine coolers, or hard liquor the patient drinks per day and how many days a week, month, or year. 1 drink is defined as 12oz beer, 5oz wine, or 1.5oz of spirits (gin, vodka, etc.). According to the National Institute of Abuse on Alcohol and Alcoholism (NIAAA) nearly 3 in 10 US adults engage in at risk drinking patterns. These are defined as greater than 4 drinks on any day for men (3 for women) or greater than 14 drinks/week for men (7 for women). If one exceeds both daily and weekly limits, there is a 50% chance of having alcohol abuse or dependence.

Acute alcohol withdrawal can be mild, moderate, or severe. Long-acting benzodiazepines are preferred (e.g. valium) for treatment because of smoother withdrawal and longer half-life. Severe withdrawal symptoms include seizures and alcohol withdrawal delirium (delirium tremens). The latter develops after cessation of heavy alcohol ingestion or a reduction in the amount of alcohol ingested, usually within one week, and is associated with marked autonomic hyperactivity (e.g., tachycardia, hypertension, diaphoresis). DTs can be fatal especially if the patient has cardiovascular compromise. DT’s is preventable by treating withdrawal with a benzodiazepine or barbiturate. Treatment should begin on a medical unit with intravenous long acting benzodiazepines.

Wernicke

This is a encephalopathy complication of severe alcohol dependence. Wernicke characterized a disorder of acute onset with mental disturbance, paralysis of eye movements, and ataxia of gait. The eye motor signs are prominent with weakness or paralysis of abduction (sixth nerve palsy), usually symmetric, and accompanied by nystagmus, strabismus, and diplopia.

Wernicke’s disease represents a medical emergency. Give thiamine 100 mg IV or IM to alcoholic patients to prevent onset of acute Korsakoff’s psychosis. Administer thiamine in ER before giving any glucose solution.
Pharmacotherapies

Medications for the treatment of alcohol dependence are disulfiram, naltrexone, or acamprosate. All medications are adjuncts to treatment and should not be used as the sole treatment. Disulfiram is used primarily for the impulsive drinker, Naltrexone if there are strong cravings and Acamprosate the newest agent probably aids reduction in desire to use. See table for further details.

Self-help groups such as AA and NA are often the mainstream of recovery. They provide ongoing support in the life change necessary for sobriety (twelve steps, use of sponsors).

<table>
<thead>
<tr>
<th>Medications for Treating Alcohol Dependence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Naltrexone</strong> (Depade®, Revia®)</td>
</tr>
<tr>
<td><strong>Action</strong></td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
</tr>
<tr>
<td><strong>Precautions</strong></td>
</tr>
<tr>
<td><strong>Serious adverse reactions</strong></td>
</tr>
<tr>
<td><strong>Common side effects</strong></td>
</tr>
<tr>
<td><strong>Examples of drug interactions</strong></td>
</tr>
<tr>
<td><strong>Usual adult dosage</strong></td>
</tr>
</tbody>
</table>

Opioids

Acute effects of opioids include meiosis or pinpoint pupils (which occur with most, but not all, opioids), constipation, hypotension, lethargy, coma, and possible death by respiratory depression. The withdrawal syndrome, beginning four to twelve hours after the last dose (depending on the drug), consists of agitation, piloerection, dilated pupils, muscle aches, and abdominal cramps. A subclinical withdrawal syndrome consisting of sleep disturbance, irritability, vital sign fluctuations, and autonomic system lability may persist for several months in tolerant.
individuals. Pharmacotherapies for opioid dependence are: methadone (through methadone maintenance programs), naltrexone (opiate antagonist), and the newest, buprenorphine (suboxone, partial agonist). Methadone Programs are clearly effective but are limited by their availability, possible exposure to drug using persons, and the requirements for frequent attendance. Buprenorphine is as effective as low-moderate doses of methadone; a special waver is required to prescribe this medication. Naltrexone’s utility is limited to those persons highly motivated who have ongoing exposure to narcotics (e.g. healthcare workers).

Sedatives

These drugs include the benzodiazepines and barbiturates. While showing cross tolerance with alcohol, they are synthetic and chemically dissimilar to each other. Sedatives with shorter half-lives tend to be more readily abused. Longer acting sedative drugs produce a safer, more stable withdrawal regimen.

Duration of action and margin of safety differ widely among the sedatives. Like the opioids and alcohol, they can produce tolerance if taken chronically in increasing doses. Acute effects include incoordination, dysarthria, lethargy, somnolence, coma, and death by respiratory depression. The withdrawal syndrome consists variably of tachycardia, fever, hypertension, headache, agitation, tremor, seizure, confusion, delusions, and hallucinations. Onset of withdrawal can occur within several hours after the last dose in the case of short acting barbiturates, or several days with the long acting diazepines.

Amphetamines and Similar Drugs

Amphetamines facilitate the release of noradrenalin thereby increasing pulse, blood pressure, metabolic rate, and sometimes temperature. Stimulant effects on the central nervous system include mydriasis, tachycardia, elevated mood, heightened self-confidence, alertness, and wakefulness with a decrease in rapid eye movement (REM) sleep.

Tolerance and increased daily doses occur in chronic users. Confusion, panic, and paranoia may ensue accompanied by a psychotic state similar to schizophrenia or mania which can persist for days, weeks, or months. Hyperthermia, arrhythmias, convulsions, and cerebrovascular accidents accompany overdose. A withdrawal consists of lethargy and increased REM sleep. Depression often appears following withdrawal; this may be a withdrawal effect, emergence of a primary depression, or some combination of both.

Cocaine

One form of cocaine is the hydrochloride, taken by injection or snorting. Crack cocaine is smoked and is relatively cheap and available. The paste form, used for smoking, involves an extraction from coca leaves using kerosene and sulfuric acid. Cocaine potentiates catecholamine effect by interfering with reuptake. Its effects are similar to those of amphetamines, but with a half-life persisting over minutes rather than hours. Certain complications resemble those of amphetamine, such as paranoia, hallucinations, or hypertension.
**Cannabis**

Although numerous psychoactive compounds exist in cannabis, most of its effect appears due to delta-9-tetrahydrocannabinol. Effect persists for a few to several hours depending on dose, tolerance, and pattern of use.

Many people appear able to consume small amounts of cannabis at infrequent intervals (i.e., weekly or monthly) without ill effect. Vulnerable individuals may experience hallucinations, delusions, or confusion at low doses. With chronic, heavy use, the percentage of impaired users probably increases.

Intoxication involves aspects of both stimulation and depression, sympathetic and parasympathetic manifestations. These include dry mouth, increased appetite, tachycardia, injected conjunctivae, and relaxation. Coordination for simple tasks is not impaired at lower doses, although balance and complex tasks become increasingly impeded with higher doses.

Some tolerance occurs with chronic use, but a distinct withdrawal syndrome has not been described. Since tetrahydrocannabinol is stored in fat, chronic users may demonstrate cannabis effect and excrete the drug for days following the last use.

**Tobacco**

Whether consumed by smoking, snuffing, or chewing, tobacco’s psychoactive effect is largely due to nicotine. Like cocaine, the half-life of nicotine is brief (under an hour). Many carcinogens also coexist in tobacco. Nicotine, which mimics the effects of acetylcholine, acts as a mild stimulant. Although smoking produces almost instantaneous effect, absorption after oral ingestion is slow. Effects include increased heart rate, gastric atony, and peripheral vasoconstriction. Large doses may produce nausea, emesis, and convulsions. Withdrawal effects include bradycardia, irritability, and increased appetite.

As a mild intoxicant with few or mild effects on cognition, mood, and coordination, tobacco rarely produces acute problems. However, it can produce numerous, sometimes catastrophic, adverse effects, including heart disease and lung cancer. Health complications increase markedly after twenty pack-years of smoking. Although smoking cessation is difficult, physician recommendations to cease tobacco use are effective and nicotine replacements and bupropion can be helpful pharmacotherapies.

**Caffeine**

In lower doses caffeine reduces fatigue and enhances mental activity while causing some tachycardia, vasodilatation, and diuresis. It produces these effects by stimulating catecholamine release. Higher doses (i.e., over 600 mg per day) may produce excitement, agitation, headache, irritability, and insomnia. Withdrawal symptoms in high dose users can include fatigue and somnolence (Dreisbach & Pfeiffer 1943, Greden 1981). Caffeine is present in many common beverages, including coffee, tea, cocoa, colas, and other soft drinks. It is also present in many over-the-counter and prescription drugs taken for pain, appetite suppression, and the common cold.
Volatile Hydrocarbons

These have the same psychotoxic effects as alcohol, but with a shorter half-life, often under an hour. Special populations, such as prisoners or children, sniff them because they are available, inexpensive, and short acting. Aerosols, glue, cleaning and industrial solvents, and paint thinners can produce hepatic, renal, hematologic, or neurological damage depending on the chemical, pattern of use, and individual propensity. Early symptoms which may come to the attention of a pediatrician or psychiatrist are irritability, declining academic or occupational performance, memory loss, and personality change.

Phencyclidine (PCP)

This drug may be ingested, snuffed, smoked, or injected. Its effects are variable so that it may produce relaxation or panic, hypotension or hypertension, decreased reflexes or status epilepticus. In general, however, it potentiates adrenergic effects. Impurities from illicit production may cause anticholinergic effects. Body image distortions, agitation, and hallucinations are common in PCP users coming to clinical attention. Vertical or horizontal nystagmus, muscular rigidity, and dystonic reactions are clues to the diagnosis. Half-life is relatively short, but after-effects can continue for hours or a few days. Acute and chronic users may present to emergency rooms with psychiatric syndromes varying from panic attack, mania, schizophreniform psychosis, and delirium.

Hallucinogens

These include natural substances (e.g., peyote, morning glory seeds) and synthetic compounds (e.g., D-lysergic acid or LSD). Altered perceptual states are produced; panic, hallucinations and delusions may occur. While the half-life of these drugs is only a few hours, psychic effects may persist for six to twelve hours. Hallucinosis may continue for a few to several days in unusual cases. In vulnerable individuals, mania or schizophreniform psychosis may ensue. Physical manifestations are few except when anticholinergic properties are present (Cohen 1981).

Clinical Evaluation

Patients may hide, alter, or accurately describe their drug use and associated problems depending on their openness, wish for help, and extent of discomfort. A key factor is the clinician’s comfort and skill in aiding patients to relate their history. A nonjudgmental attitude toward patients is also critical. Patients may present from early in their course to the severely advanced stages.

Drug abuse patients usually seek clinical help due to some coercive force, either external (e.g., family, work supervisor) or internal (e.g., malaise, depression). An important step in management involves delineating this coercive force. Complicating this process is the patient’s frequent lack of awareness regarding the relationship between the current problem and the drug use. Another obstacle may be the patient’s tendency to blame others for the current problems rather than to take responsibility for the problem.

Drug abusing patients often do not volunteer symptoms indicative of depression, anxiety, panic, or psychosis; specific inquiry is necessary.
Formal mental status exam may reveal unsuspected deficits in orientation, memory, or cognition. A physical exam can demonstrate evidence of parenteral injection (e.g., venous tracks, skin popping scars), chronic smoking (e.g., rales and rhonchi), malnutrition, infectious diseases, and traumatic sequelae. Neurological findings (e.g., ataxia dysartrhia, pupillary changes), autonomic signs (e.g., flushing, perspiration, piloerection), and vital sign abnormalities (e.g., tachycardia, hypertension) provide valuable clues. See Table 1 for signs associated with various drugs (Westermeyer 1976).

**The Diagnostic Interview**

The diagnostic interview aims to establish the extent of substance use, consequences caused by substance use, determine if there is loss of control and gather enough information to make diagnoses. Possible consequences to inquire about are social, family, financial, health, legal and occupational.

The patient may be defensive, in denial, or exaggerate the extent of use. Being persistent in the interview is important and obtaining history from other sources is valuable.

**Clinical Course**

The typical course of untreated, chronic drug abuse is deterioration over a period of years, often with periods of relative stabilization or brief improvement followed by further deterioration. Acute problems associated with recent drug abuse may cause the disorder to be self-limiting, if the consequences motivate the user to moderate or cease drug usage. Spontaneous abstinence from drugs does occur but is infrequent among those with recurrent episodes of drug abuse or with chronic drug dependence.

Duration of course, like the clinical picture, varies with the drug, route of administration, and various host and environmental factors. Other things being equal, routes with rapid drug onset (i.e., injection, smoking, snuffing) hasten the morbid course over slower routes of administration (e.g., ingestion, chewing). Drugs with shorter half-lives (e.g., heroin, cocaine) lead to a more rapid course than those with longer half-lives (e.g., opium, diazepam, amphetamine). More potent drugs (e.g., morphine, methadone) hasten and increase the morbid effects over weaker drugs in the same category (e.g., codeine, propoxyphene). Some drugs usually produce medical complications (e.g., tobacco) or neuropsychiatric complications (e.g., phencyclidine) as their first manifestation, while others are more apt initially to produce psychosocial consequences (e.g., sedatives, opioids).

Age at onset influences the course, such that opioid dependence beginning at age fifteen affects the patient’s life course differently when compared to opioid dependence beginning at age thirty-five. Younger individuals have not yet had the opportunity to complete their education, learn an occupation, become employed, marry, have children, or otherwise establish some social competency. Older drug abusers coming to treatment usually have more biomedical problems and social isolation, while younger drug abusers gradually experience more legal, occupational, and marital problems. Women show a more rapid progression than men in both drug and alcohol abuse.
Treatment is effective and cost-effective. The evidence is clear that primary care physicians can effect change in reducing drinking in the hazardous drinker by brief interventions. In general, treatment earlier in the course tends to be more effective and less costly.

Acute phases of recovery, from medical, psychiatric, or social crises, usually take place over several weeks. Late recovery from autonomic instability, remorse, and social isolation occurs over several months. Psychological well-being, social fulfillment, and occupational stability may require a few to several years. The highest rates of relapse occur in the first 6 months after treatment (Figure 1). Although pharmacological factors greatly influence the pre-treatment course, the post-treatment relapse rates for heroin, alcohol, and tobacco (in the absence of ongoing outpatient treatment) are remarkably similar (Figure 1).

**Differential Diagnosis**

Differentiating drug abuse from other psychiatric disorders is often difficult. Substance abuse and psychiatric disorders coexist in one-quarter to one-third of psychiatric patients and in about the same proportion of substance abuse patients. Drug abuse may develop as an attempt at self-treatment for a preexisting disorder (e.g., stimulant abuse for depression, sedative abuse for anxiety or mania). Secondary psychiatric disturbances (e.g., reactive depression, panic disorder) may appear in the later stages of drug abuse. Secondary sociopathy may attend the disinhibiting effects of certain drugs.

Drug effects may mimic psychiatric disorder. For example, caffeine, cocaine, or amphetamine intoxication can produce symptoms like those of anxiety or mania (Greden 1974). Withdrawal from these drugs may resemble depression or, less often, paranoia. Acute cannabis, phencyclidine, or hallucinogen intoxication may present clinically as acute schizophreniform psychosis, manic psychosis, or organic delirium. (see Table 1 for specifics)

**Treatment**

**Drug Related Emergencies**

Intoxication is managed simply by observing and protecting the individual until the drug is metabolized or excreted. It is important to ensure that the patient does not injure self or others while the drug is being metabolized and/or excreted. Involuntary hospitalization may be necessary for two or three days during this phase. Overdose management depends on the specific drug and dose.

Withdrawal treatment lessens discomfort, reduces mortality, and can aid in establishing the doctor-patient relationship but it does not deter substance abuse. It does offer the opportunity to engage the patient and help motivate the patient for treatment.

**Treatment Modalities**

Treatment programs vary in intensity and duration from the most intense being inpatient followed by intensive outpatient, standard outpatient, halfway house and community supports.
Table 2 summarizes the American Society of Adduction Medicine criteria to match patients to the appropriate level of care.

Modalities for treatment of drug abuse are numerous and include the following:

1. Psychotherapies and Sociotherapies: individual, couples, family and group; verbal aversion; contingency contracting; social skills learning; day, evening, or weekend programs.

2. Self-Help: Narcotics Anonymous (primarily for illicit drug abusers), Alcoholics Anonymous (primarily for abusers of prescribed or licit drugs as well as alcohol), Al-Anon (for friends and relatives of drug abusers).

3. Pharmacotherapies: methadone, buprenorphine, naltrexone orally (for opioid or alcohol dependent patients or monthly injection (Viritrol®), disulfiram, acamprosate (for alcohol dependent patients).


If major psychiatric problems persist beyond a few to several days, they will probably not resolve spontaneously. Continuation of major depression, schizophreniform psychosis, mania, and other major disorders beyond two weeks almost always calls for specific treatment rather than expectant observation. If the patient responds rapidly and completely to low doses of medication, a lengthy course of medication may not be needed.

Minor or less disabling psychiatric syndromes are common in the early weeks of recovery. These include adjustment reactions, generalized anxiety, and panic disorder. If these are decreasing in severity and becoming less frequent, specific treatment may not be necessary. On the other hand, increasing, severe, or disabling symptoms generally require psychiatric treatment.

**Treatment Goals, Outcome, and Efficacy**

Treatment for drug abuse may be aimed at total abstention, reduction of drug use, or removal of problematic aspects from continued drug use. Generally, abstention (temporary or permanent) is the explicit goal. Still, many favorable outcomes from treatment involve reduction in associated problems or in amounts of drug used despite continued or episodic use. Assessment of treatment outcome must take into account these diverse goals of treatment.

Patients with the best prognosis are those with good social support and have ongoing recovery activities. Physicians have a unique role in providing motivation for patients to move from an indecision (or rejection of help) phase to that of active recovery and ultimately maintenance of behavioral change.

Directed motivational interviewing & treatment uses the five stages of change as principles. They are:

- Precontemplation, in which the patient denies that substance use is a problem.
- Contemplation, in which the patient realizes that there is a problem but is ambivalent about making the change.
- Preparation, in which the patient is becoming more ready to take action.
- Action, in which the patient takes specific steps towards cessation of use.
- Maintenance, in which the patient focuses on continuing positive changes and acquires relapse prevention skills.
Table 1. Drug Signs

<table>
<thead>
<tr>
<th></th>
<th>Intoxication</th>
<th>Overdose</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alcohol</td>
<td>Stimulants</td>
<td>Sedatives</td>
</tr>
<tr>
<td>Vital signs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circulatory collapse</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Hypertension</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Respiration, slow and shallow</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Appearance, Behavior, Mental Status</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Affect, labile</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Comprehension, slow</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Delirium</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Delusions</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Depressed mood</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Euphoria</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Hostile, assaultive</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Irritability</td>
<td>●</td>
<td>●</td>
<td>●</td>
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<tr>
<td>Lethargy</td>
<td>●</td>
<td>●</td>
<td>●</td>
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<tr>
<td>Memory, poor</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Restlessness</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Skin picking</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Suspiciousness</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Sweating</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Talkativeness</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Vomiting</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Yawning</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td></td>
<td>Intoxication</td>
<td>Overdose</td>
<td>Withdrawal</td>
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</tr>
<tr>
<td>Alcohol</td>
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<tr>
<td>Stimulants</td>
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<tr>
<td>Sedatives</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Opiates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hallucinogens</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phencyclidine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulants</td>
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<td></td>
<td></td>
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<tr>
<td>Sedatives</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Opiates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hallucinogens</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phencyclidine</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Eyes, Ears, Nose and Throat**

<table>
<thead>
<tr>
<th></th>
<th>Intoxication</th>
<th>Overdose</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coryza</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lacrimation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouth dry</td>
<td>●</td>
<td>●</td>
<td></td>
</tr>
<tr>
<td>Nystagmus</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Pupils - dilated</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Pupils-pinpoint</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhinorrhea</td>
<td>●</td>
<td>●</td>
<td></td>
</tr>
</tbody>
</table>

**Nerological Exam**

<table>
<thead>
<tr>
<th></th>
<th>Intoxication</th>
<th>Overdose</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesia to pinprick</td>
<td>● ● ● ● ●</td>
<td>●</td>
<td></td>
</tr>
<tr>
<td>Coma</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Convulsions</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Facial grimacing</td>
<td>●</td>
<td>●</td>
<td></td>
</tr>
<tr>
<td>Hypotonia</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Muscle spasms (rigidity)</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Reflexes, hyperactive</td>
<td>● ● ● ● ●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Speech, slurred</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Stare, blank</td>
<td>●</td>
<td>●</td>
<td></td>
</tr>
<tr>
<td>Tremor</td>
<td></td>
<td>●</td>
<td>●</td>
</tr>
</tbody>
</table>

**Skin**

<table>
<thead>
<tr>
<th></th>
<th>Intoxication</th>
<th>Overdose</th>
<th>Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flushing</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Piloerection</td>
<td></td>
<td>●</td>
<td></td>
</tr>
</tbody>
</table>
This overview of the Adult Admission Criteria is an approximate summary to illustrate the principal concepts and structure of the criteria.

<table>
<thead>
<tr>
<th>Levels of Care/Criteria Dimensions</th>
<th>Level I Outpatient Treatment</th>
<th>Level II Intensive Outpatient Treatment</th>
<th>Level III Medically Monitored Intensive Inpatient Treatment</th>
<th>Level IV Medically Managed Intensive Inpatient Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Acute Intoxication And/Or Withdrawal Potential</td>
<td>No withdrawal risk</td>
<td>Minimal withdrawal risk</td>
<td>Severe withdrawal risk, but manageable in Level III</td>
<td>Severe withdrawal risk</td>
</tr>
<tr>
<td>2 Biomedical Conditions and Complications</td>
<td>None or very stable</td>
<td>None or non-distracting from addiction treatment and manageable in Level II</td>
<td>Require medical monitoring but not intensive treatment</td>
<td>Require 24 hour medical and nursing care</td>
</tr>
<tr>
<td>3 Emotional/Behavioral Conditions and Complications</td>
<td>None or very stable</td>
<td>Mild severity, with potential to distract from recovery</td>
<td>Moderate severity, needing a 24 hour structured setting</td>
<td>Severe problems requiring 24 hour psychiatric care, with concomitant addiction treatment</td>
</tr>
<tr>
<td>4 Treatment Acceptance/Resistance</td>
<td>Willing to cooperate, but needs motivating and monitoring strategies</td>
<td>Resistance high enough to require structured program, but not so high as to render outpatient treatment ineffective</td>
<td>Resistance high enough despite negative consequences; needs intensive motivating strategies in 24 hour structure</td>
<td>Problems in this dimension do not qualify patient for Level IV treatment</td>
</tr>
<tr>
<td>5 Relapse Potential</td>
<td>Able to maintain abstinence and recovery goals with minimal support</td>
<td>Intensification of addiction symptoms and high likelihood of relapse without close monitoring and support</td>
<td>Unable to control use despite active participation in less intensive care; needs 24 hour structure</td>
<td>Problems in this dimension do not qualify patient for Level IV treatment</td>
</tr>
<tr>
<td>6 Recovery Environment</td>
<td>Supportive recovery environment and/or patient has skills to cope</td>
<td>Environment unsupportive but with structure or support, patient can cope</td>
<td>Environment dangerous for recovery, necessitating removal from environment; logistical impediments to outpatient treatment</td>
<td>Problems in this dimension do not qualify patient for Level IV treatment</td>
</tr>
</tbody>
</table>
Figure 1. Relapse Rate Over Time

References


