Unit 5 - Anxiety Disorders

I. Introduction
Reading: Brown et al. (2001); Wittchen & Essau (1993); Maxmen Ch 11; DSM-IV

A. Description
1. Symptoms of anxiety
   a. physiological arousal: autonomic arousal
   b. Maladaptive cognitions (differ according to disorder: e.g., social failure social phobia)
   c. Behavioral disturbance (e.g., avoidance)
   d. Subjective distress: feelings anxiety, tension

2. Normal anxiety vs. pathological
      1) normal fear is adaptive response: escape from or avoidance of dangerous situation
      2) pathological fear/anxiety: exaggerated fear with high negative affect & hyperarousal; is irrational

3. Epidemiology: among most frequent disorders: lifetime: 20% males, 30% females (Kessler et al., 1994; National Comorbidity study)

B. Assessment
1. General strategies:
   a. triple response mode for evaluation: evaluate cognitive, behavioral and physiological aspects
   b. self-report measures: Beck Anxiety Inventory (BAI): can ask about each of 3 aspects
   c. symptom rating scales: Hamilton Scales (anxiety, dep): rate after an interview; also address the 3 aspects
   d. structured interviews: Anxiety Disorders Interview Schedule -Revised(ADIS-R; DiNardi et al., 1987)
   e. behavioral assessment: behavioral avoidance test (BAT)
   f. physiological measures: HR, BP, GSR with exposure, approach of anxiety provoking stimuli

C. History and Classification
1. DSM-II (1968)
   a. 3 categories: anxiety neurosis; phobic neurosis; obsessive-compulsive neurosis
   b. were in Neurosis section with some mood, dissociative, and somatoform disorders

2. Feighner criteria (Feighner et al., 1972)
   a. criteria for 3 DSM-II cats: anxiety, phobic, and obsessive-compulsive neuroses
   b. same names as DSM-II but provided more specific, operational criteria

3. Research Diagnostic Criteria (RDC; Spitzer, Endicott & Robins, 1975)
   a. Provided criteria for 4 disorders: Panic disorder, GAD, OCD, Phobic disorder
   b. introduced current terminology (i.e., Panic dis, Generalized Anxiety dis, Obsessive-comp dis, Phobic dis)
   c. more specific categories and criteria than Feighner et al.

4. DSM-III (1980): anxiety disorders became a separate chapter for first time
   a. increased specificity: allowed distinguishing between specific anxiety disorders, from mood disorders
   b. allowed for multiple diagnoses: reduced diagnostic hierarchy, allowed examination of comorbidity
   c. divided into two major groups:
      1) Phobic Disorders: agoraphobia, social phobia, simple phobia
      2) Anxiety States: panic dis, GAD, OCD, PTSD (PTSD included for the first time)

5. DSM-III-R (1987):
a. changes to specific criteria  
b. eliminated division into phobias and anxiety states  
c. linked together agoraphobia and panic disorder  
d. added generalized subtype of social phobia—confusion w/ avoidant pd

a. changes to specific criteria for some disorders  
b. added Acute Stress Disorder - like PTSD but shorter duration (>2 days, < 1 mo) and additional dissociative sx  
c. changes in other categories:  
   1) Panic disorder and agoraphobia: gives criteria for panic attacks and agorophobia first, then can dx: panic dis w/ or w/out agorophobia; agorophobia w/out hx of panic  
   2) Social phobia: kept generalized subtype - further increased the overlap with avoidant pd  
   3) Simple phobia became specific phobia (now can also specify type)  
   4) Obsessive compulsive disorder: added specification w/ poor insight  
   5) Generalized anxiety disorder: less on physiological arousal, more focus on general worry and distress  
   6) Post-traumatic stress disorder: added specifiers  
      a) acute (>1 mo but < 3 mos) vs. chronic (>3 mos)  
      b) delayed onset: onset 6 mos or more after the trauma

D. Comorbidity  
1. Comorbidity and sx overlap among anxiety disorders  
   a. Davidson & Foa (1991) - PTSD criteria overlap with phobias, GAD, OCD (as well as dep); high comorbidity  
   b. Brown & Barlow (1992): high comorbidity among anxiety dis and overlapping sx: panic, anxious apprehension/worry common to all anxiety dis; DSM-IV criteria attempted to reduce the sx overlap  
   c. Marks (1986); Beidel & Turner (1997): theories of etiology suggest what is inherited is anxiety proneness, not risk for specific disorders, familial basis of anxiety disorders is nonspecific: Stein, Jang & Livesley (1999)

2. Overlap and comorbidity of social phobia (especially generalized subtype) with avoidant pers dis  
   a. problems with DSM-IV definitions - encourages assignment of both dx (violates principle of parsimony in dx)  
   b. Barlow (1989) and Marks (1985) general criteria to differentiate social phobia from avoidant pd  
      - social phobia not have social skills deficit, avoidant does  
      - onset more acute with social phobia but early (like avoidant pd)  
      - equal sex ratio for social phobia (social skills deficit more apparent for men)  
      - more focal for social phobia (sx are more diffuse, pervasive for avoidant pd)  
      - tx: exposure, anxiety management for social phobia (vs social skills training for avoidant and those with social skills deficit)  
      - these guidelines published before generalized type - harder to distinguish now

3. Comorbidity with other disorders: depression, personality disorders, substance use disorders  
      1) high rate of comorbid depression (although findings vary): anxiety typically precedes depression (may be different points on helplessness-hopelessness continuum)
2) alcohol and other subs - usually anxiety precedes (self-med hypo), but subs may induce anxiety (e.g., caffeine)

3) personality disorders: 27-65% panic disorder pts have pers dis; may also be assoc w/ increased dysthymia; could be high because of overlapping sx cluster C pers dis and anxiety dis

4) prognosis: comorbid depression or personality disorder -> poorer tx response

1) high comorbidity dep and anxiety; many common sx (e.g., irritability, reduced concentration, insomnia, etc), lack of clear boundaries between dep and anxiety (and among anxiety dis);

2) principal components analysis sx pts major depression, dysthymia, GAD, panic disorder: suggest can differentiate major dep and panic; differentiating GAD from dysthymia is less clear

3) anxiety: specific sx = physiological arousal, panic, avoidance, threat-related cognitions, worry

4) dep: specific sx = psychomotor retardation, crying, anhedonia, SI, early morning awakening, pessimism, feelings of loss, hopelessness

5) dep and anxiety each have specific sx and nonspecific shared sx

1) Munich, follow-up study for 7 yrs examining comorbidity depression and anxiety and support for MAD

2) high comorbidity: 68% major dep pts had 1 anxiety dis in lifetime (highest simple phobia); 1/3 x-sectionally

3) overlapping sx: self-report data esp supports dimensional model and overlap (less for clinician ratings)

4) overlap of risk factors for anxiety and depression

5) Mixed Anxiety-Depression: DSM-IV research category needing further validation
   a) not comorbid dep + anxiety (if comorbid, would dx both); is dep + anxiety that fails to meet criteria for either dep or anxiety disorder -> MAD

   b) variable criteria used in research -> diff estimates (1-3%); Wittchen & Essau found .8% MAD; 22% only subthreshold anxiety, 2.5% only subthreshold dep

   c) some evidence MAD is frequent in medical patients and results in impaired functioning and distress but because not meet criteria for a mood or anxiety disorder, are not treated

4. Dimensional model of anxiety and depression:
   a. Brown, Chorpita & Barlow (1998); Clark, Beck & Beck (1994): suggest dimensional models of anxiety and depression; believe DSM classification makes fine distinctions between categories w/out meaning:

   1) pts respond same meds (antidep, antianxiety), both respond to cog therapy

   2) similar family histories: mood disorders in families anxiety disorder pts

   b. Clark and Watson's (1991) Tripartite model:
1) 3 factors: **Negative affectivity (NA):** general nonspecific distress, common factor present in dep and anxiety
   **Positive affectivity (PA):** low PA=anhedonia – specific to depression
   **Autonomic arousal (AA):** high AA – specific to anxiety

2) high NA may be general vulnerability factor for dep and anxiety (Watson et al., 1994)

3) Using the three dimensions:
   - depression: high NA + low PA (anhedonia)
   - anxiety: high NA + high AA
   - comorbid anxiety and dep: high NA + low PA + high AA
   - MAD: high NA only (moderate low PA, moderate high AA)

4) Clark, Beck & Beck (1994): found support for model:
   a) anxiety: specific arousal (AA), threat-related cognitions and subjective distress
   b) depression: anhedonia (low PA), cognitions of personal loss and failure, subjective distress

4. **Brown et al. (2001): SCOTT:** Reliability of diagnosis of anxiety and mood disorders and differential diagnosis
II. Anxiety Disorders in a Diverse Society
Reading: Pigott (1999); Wetherell et al. (2003)

A. Race/ethnicity: not a lot of research
   a. lack of information; few non-majority pts in samples
   b. ECA study: more representative sample; suggest higher anxiety disorders — phobias, agoraphobia — in African Americans
   c. may be most common presenting problem of African American women seeking tx
   d. may not respond as well to meds

B. Gender Differences
1. Increased rate of anxiety disorders in women (up to 3:1) but less consistent evidence than for depression: Q of role of environment, socialization, hormones, common factors with vulnerability to depression
   a. Panic Disorder without Agoraphobia: 2:1, with Agoraphobia: 3:1, (also Agoraphobia without hx of panic dis dxed much more often in women, specific data lacking)
   b. Specific Phobias: ratio of 2:1; however, depends on the nature of the phobia:
      1)75-90% animal and environmental phobias, situational phobias female
      2) 55-70% fear of heights, fear of blood-injection-injury female
   c. PTSD and Acute Stress Disorder: ?? but gender-specific types of traumas: rape mostly women, combat mostly men

B. Elderly
1. Lang & Stein (1993): is most common psychiatric sx of older adults (esp GAD), underdxed
   a. medical problems, financial, losses, cognitive impairment play a role
   b. generally recommend CBT or SSRIIs (less side effects than benzodiazepines)
III. Etiology

Reading: Stein, Jang & Livesley (1999); Rivaz-Vazquez (2001); Chambless & Gillis (1993); Maxmen Ch 11; DSM-IV

A. Biological

1. Genetics: anxiety dis run in families- esp panic, OCD, but research suggests may be a nonspecific vulnerability
   a. Watson et al. (1994) negative affect (NA) is a nonspecific risk for anxiety, depression – is heritable
   b. Marks (1986): family studies: increased anxiety disorders in 1st degree relatives of pts with anxiety disorders
   c. Kendler et al. (1992): 30-40% heritability for phobias
   d. Beidel & Turner (1997): genetic basis: anxiety proneness not specific anxiety disorders; also increased risk
      with low SES (role of environment)
   e. Andrews et al. (1990): MZ>DZ twins in concordance for anxiety dis (not necessarily the same disorder)- suggests nonspecific genetic vulnerability
   f. Stein, Jang & Livesley (1999): twin study of over 300 pts
      1) anxiety sensitivity heritability = 45%: is more of a "neurotic" trait - a risk factor for anxiety dis
      2) However, additive effect of genetics with environment
      3) childhood sexual/physical abuse also a risk factor; role of traumatic conditioning experiences

2. Biochemical
   a. some evidence serotonin, norepinephrine or GABA (gamma amino butyric acid) involved in anxiety disorders
   b. evidence: response to antidepressants (increase availability of serotonin, norepinephrine); family hx depression
      or mood disorders in pts with anxiety disorders
   c. Nesse et al. (1994): found support for higher NE in panic and agoraphobia > controls; nonspecific - is elevated
      with mania, other dis (how does this conflict with findings that anxiety dis pts respond to antidepressants?)
   d. Rivaz-Vazquez (2001):
      1) role of amygdala, LC, RN, serotonin and norepinephrine in anxiety disorders
         a) amygdala: in limbic system, role in emotional significance of stimuli, sympathetic/parasympathetic
            NS, hypervigilance, hyperventilation
         b) locus cerulus (LC): in pons, primary NE structure in brain, associated with mood and anxiety,
            increased arousal, vigilance; higher levels of NE associated with anxiety-like physiological sx
         c) raphe nuclei (RN): also in pons, primary site production of 5-HT, with projections to amygdala
      2) low 5-HT results in dysregulation of LC and production of NE: increased firing in LC and increased
         production of NE lead to anxiety
   e. panic disorder: increased sensitivity to CO2 receptors; challenge with CO2, induces panic; some evidence this
      increased sensitivity is genetic (cognitive aspect too: interpretation of sensations)

3. Other
   a. predisposing medical conditions: e.g., mitral valve prolapse predisposes to panic (misinterpret sx of MVP as
      anxiety -> panic attack): again, cognitions play important role (providing info about MVP can eliminate panic)
   b. Rosen & Schulkin (1998): hyperexcitability of fear circuits in amygdala (in temporal lobe - is collection of
nuclei that relates to other parts of the brain responsible for emotion, motivation) through kindling process (hyperexcitable -> fire more -> become more sensitive, lower threshold)

1) is an interactive theory: psychosocial stress-> release of hormones that change brain fear circuits
2) hyperexcitability one set of nuclei (bed nucleus of stria terminalis) associated with nonspecific fear (e.g., GAD)
3) hyperexcitability another nucleus in the amygdala associated with cue-specific fear (e.g., phobias)

B. Psychological
1. Psychoanalytic: defense mechanisms against anxiety fail: forbidden thoughts, impulses surface; lack of evidence

2. Behavioral: empirical support, interacts with biological factors and predisposition, and with cognitions:
   a. social learning/modeling: learn to be fearful by imitating others (e.g., parent with snake phobia)
   b. classical conditioning (traumatic experience): Stemberger et al. found support for specific social phobia
   c. operant conditioning: avoidance of anxiety provoking situations -> anxiety reduction, is very reinforcing
   d. stimulus generalization: leads to more situations, stimuli becoming associated with anxiety

3. Cognitive: appraisal and interpretation of physiological sx plays an important role; also maladaptive cognitions
      1) biological dysregulation and negative life events lead to false alarms (internal and external stimuli inappropriately interpreted as danger)
      2) sensitivity to life events could be biological or learned, conditioned
      3) false alarms associated with interoceptive cues person finds aversive, and autonomic NS sx
   b. Information processing theories:
      1) person fears physiological sensations associated with normal arousal (exercise, hormones, adrenalin, etc)
      2) interprets sensations as evidence of physiological danger (heart attack, death)
      3) person may not be aware of interpretation;
      4) results in increased attention to body, physiological sensations, monitoring for future attacks
      5) relates to theory of predisposing medical conditions like MVP - misinterpretation of sx
   c. Maladaptive schemas and automatic thoughts:
      1) Beck (and others) have shown anxiety disorders associated with certain schemas; certain automatic, maladaptive thoughts
      2) Clark, Beck & Beck (1994): threat-related cognitions (worry about future events) associated with anxiety

C. Other and Interactive
1. Most biological (genetic, neurotransmitters) theories are actually interactive theories that include role of cognitive interpretation, environmental conditioning

2. stress and behavioral and cognitive theories recognize role of predisposing biological vulnerabilities

3. Gray's theory: dysfunction in the behavioral inhibition system
   a) hypervigilance when discordance between actual and expected stimuli, when stimuli suggests danger, or are aversive (interaction of cognitive with biological systems)
   b) results in motor behaviors seen with anxiety (hypervigilance, freezing behavior)

a) social phobics have decreased social cooperation, see situations as more competitive, have lower dominance, more submission;
b) defense system: dominance/submission hierarchy, avoidance of harm; focus of social phobics
c) safety system: newer (primates); cooperation and approach

III. Treatment
Reading: Rivaz-Vazquez (2001); Chambless & Gillis (1993); Maxmen Ch 11; DSM-IV

A. Biological

1. Rivaz-Vazquez (2001): SSRIs have become the first line of treatment for anxiety disorders

a. TCAs: 1st antidepressants approved for anxiety
   1) still a lot of evidence TCAs effective for some anxiety disorders but high number of side effects, drop out rates
   2) Anafranil (clomipramine): 1st TCA approved for tx of anxiety dis: approved for tx OCD; (perhaps more than SSRIs but more side effects)

b. Specific SSRIs approved for tx anxiety disorders:
   1) Prozac (fluoxetine): FDA approved for OCD
   2) Luvox: (fluvoxamine): FDA approved for OCD
   3) Zoloft (sertraline): FDA approved for OCD, panic, PTSD
   4) Paxil (paroxetine): FDA approved for OCD, panic, social phobia

c. New and atypical antidepressants that affect 5-HT and NE:
   1) Effexor XR (venlafaxine): FDA approved for GAD
   2) Serzone (nefazodone) and Remeron (mirtazapine): newer but some research support;
   3) Serzone is SSRI + 5-HT2 antagonist
   4) Remeron is tetracyclic that increases release of 5-HT and NE (not a re-uptake inhibitor)

d. MAOIs used in past but like TCAs problems with side effects and drop out

e. benzodiazepines: (diazepam-Valium; chlordiazepoxide-Librium; lorazepam-Ativan; Alprazolam-Xanax): panic, Social/specific phobia, GAD: some rapid relief but generally not recommended
   1) some effectiveness but may habituate to them; also no lasting effect when DC
   2) problems: side effects; rebound effects; dependency/abuse, cognitive and motor impairment

f. SSRIs have equal or better efficacy than TCAs, MAOIs, benzos, with better profile of side effects; however, have their own side effects: nausea, wt gain, sedation, headache, insomnia, sexual dysfunction
   1) because side effects may resemble anxiety may need to start at lower doses than for major depression
   2) however, therapeutic dose range for anxiety disorders somewhat higher than for major depression

2. Other medications
a. Buspar (buspirone): non-sedating anxiolytic, no dependency or abuse because slower acting, better than benzodiazepines
b. beta-blockers: (propranolol-Inderal; atenolol-Tenormin); especially for social phobia, blocks physiological sx;
B. Psychological: cognitive-behavioral: considerable evidence for their effectiveness short term and long term

1. 3 key elements of behavior therapy:
   a) exposure->extinction anxiety (in vivo, imagination)
   b) anxiety management: relaxation; pair relaxation (incompatible w/ anxiety) with anxiety-provoking stimuli (classical conditioning)
   c) response prevention: (operant conditioning: eliminate the reinforcement for avoidance, or compulsion)

2. cognitive therapy:
   a) identify, challenge maladaptive schemas and automatic thoughts; replace with more functional
   b) correct misperception of sensations

3. specific techniques of CBT: relaxation, systematic desensitization, flooding, response prevention, thought stopping, exposure - imagery, in vivo

4. Chambless & Gillis (1993): review of use of CBT for GAD, panic w/ or w/out agoraphobia, social phobia
   a) CBT> waiting list control, mixed results for CBT compared to behavioral
   b) however, cognitive change was the best predictor of tx outcome
   c) CBT may also reduce associated depression

5. Gould et al. (1997) meta-analysis of controlled trials of meds vs therapy:
   a) CBT=pharmacotherapy for GAD but CBT helped w/ depression, increased maintenance, effects of meds diminished when DC'ed; also meds: high side effects, drop out rates
   b) Panic: relapse after DC meds (poss GAD, too)
   c) OCD: clomipramine (Anafranil) >SSRIs: but Qs about the method (Also, as Rivaz-Vazquez noted, more side effects, drop outs)
   d) Survey of physicians found that the most common meds used were benzodiazepines (diazepam), followed by buspirone: may differ now (probably would be using SSRIs)
IV. Specific Disorders

Reading: Stein & Kean (2000); Rivaz-Vazquez (2001); Chambless & Gillis (1993); Maxmen Ch 11; DSM-IV

A. Panic disorder and agoraphobia:
1. panic disorder: 4 attacks/4 weeks; or 1 attack + 1 mo of worrying about attack
2. agoraphobia: fear of places escape difficult; some can go out if with "safe" person
3. relationship between the two (panic attacks-> agoraphobia; rarely see alone)
4. epidemiology: increased in women (2 or 3:1)
5. comorbidity with per dis, up to 50% with mood dis
6. increased risk w/ certain medical conditions (mitral valve prolapse)
7. runs in families: genetics play role in proneness (20% in 1st degree relatives; concordance MZ:DZ twins 5:1
8. role of caffeine, stimulants (sub-induced); self-medication
9. Treatment:
   a) meds: Rivaz-Vazquez (2001):
      1) Paxil 1st FDA approved med, one study: 90% improved; about equal to clomipramine but faster
      2) Zoloft 2nd FDA approved
      3) not approved but research suggests Prozac, Luvox, Serzone are effective
   b) behavioral and cognitive approaches:
      1) main tx=exposure, have moved from graded exposure to self-directed approaches; 65-75% effective, but 20% not improve or residual sx; use exposure to panic-provoking situations
      2) cognitive: address interpretation of bodily sensations;
   c) Ballenger (1999): both meds and therapy effective; SSRIs are first line of tx but less relapse with CBT; exposure based tx for those with panic + agoraphobia

B. Social phobia:
1. irrational fear of social sits where exposed to the scrutiny of others
2. originally thought did not lack the skills (while avoidant did) but this distinction blurred w/ generalized type
3. comorbidity with GAD, major dep, OC pd and avoidant pd
   a) generalized more severe, earlier onset than specific type
   b) specific social phobia: may be more related to traumatic conditioning experiences
   c) generalized more related to neurosis, childhood shyness
   d) not found support for role of family/genetics
5. Stravynski & Greenberg (1989):
   a) 2 dimensions of sx: anxiety (low-high) and social skills deficit (low-high)
   b) exposure necessary component in tx, if social skills deficit may need social skills training
   a) one of most common anxiety disorders (5-8%)
   b) this study: 6.7% in community survey looking at DSM dx and impairment
   c) increased rate of comorbid depression
   d) found those w/ social phobia impaired across a variety of measures of functioning – education, occupation, interpersonal
7. Treatment:
   a) Rivaz-Vazquez (2001): all SSRIs acute efficacy; only paroxetine (Paxil) FDA approved – has most support; Serzone or Effexor may be useful alternatives - need more research
   
b) Gould et al. (1997): meta-analysis of research on cog-beh and med tx social phobia
      1) found cog-beh=meds overall; best cog-beh=exposure; best med=SSRI (less side effects, drop out)
      2) concluded cog-beh best due to lower costs
      3) need more research: follow-up studies, examine comorbidity - esp with avoidant (poorer prognosis), generalized vs specific subtypes, effectiveness of tx components, combined txs

c) Ballenger (1999): both meds and therapy effective: Paxil or other SSRIs; CBT may reduce relapse

d) Other tx: beta blockers before speech (Q efficacy), self talk and examination of cognitive distortions

C. Specific phobia
   1. irrational fear specific sit, object
   2. common in childhood, most disappear
   3. many not seek tx; cog and beh tx recommended (graduated exposure; desenistization); meds not effective

D. Obsessive compulsive disorder
   1. obsessions: recurrent, intrusive, repulsive thoughts; increase anxiety
   2. compulsions: stereotyped behaviors compelled to perform; reduce anxiety, person recognizes as senseless
   3. examples: thoughts of contamination, doubt; compulsive checking, handwashing
   4. early onset, chronic, relatively rarer; equal sex ratio; high comorbidity with depression (up to 80%)
   5. self-medication common
   6. genetic evidence: concordance twins: MZ=70%, DZ=50%; 25% in 1st degree relatives
   7. some evidence abnormality in frontal lobes, basal ganglia, serotonergic dysregulation (why SSRIs help)
   8. serotonergic meds helpful: SSRIs, serotonergic tricyclics: clomipramine (Anafranil)
   9. Treatment:
      b) beh therapy (exposure in vivo, imagination, satiation, thought stopping) effective in reducing rituals, less effective with obsessive thought
      c) exposure + response prevention 50-70% effective in reducing rituals; exposure in vivo> antidepressants
      d) cost effectiveness of self-exposure homework assignments
      e) Rivaz-Vazquez (2001):
         1) clomipramine (Anafranil) and SSRIs (fluoxetine - Prozac; sertraline - Zoloft; paroxetine - Paxil; fluvoxamine - Luvox) considered anti-obsessional medications
         2) Anafranil may be slightly more effective but more side effects
         3) research show all SSRIs about equal but some pts respond better to certain meds than others
      f) Ballenger (1999): all SSRIs effective but may relapse when DC; prolonged exposure (in vivo) plus response prevention may be preferred

D. Generalized anxiety disorder
   1. tension, arousal, worry
   2. gradual onset, young, more common in women
   3. more common than panic, OCD, social phobia: 8.5% lifetime; female: male ratio 2:1
   4. high overlap with mood dis(>50% comorbid dep)
   5. Rivaz-Vazquez (2001): is a heterogeneous, multidimensional disorder, often mis(under)diagnosed due to comorbidity with major depression
6. Treatment

a) **Rivaz-Vazquez (2001):** recent approval of extended release venlafaxine (Effexor XR) - 1st antidepressant approved for GAD; paroxetine (Paxil) and TCAs are better than benzodiazepines; some suggestion Serzone and Remeron may help

b) behavior therapy: relaxation: Marks (1989): combination cognitive and behavioral best

c) Ballenger (1999):
   1) CBT, applied relaxation, behavior therapy all work
   2) benzodiazepines work (probs with abuse, relapse when DC, need to taper off); buspirone (works but slow acting); Effexor or other SSRIs (or tricyclics) may be preferred

E. Post-traumatic stress disorder and acute stress disorder:

1. significant trauma; intrusive sx/re-experiencing of trauma; numbing/avoidance; increased arousal
2. acute stress: shorter duration (< 1 mo), dissociative sx
3. PTSD: > 1 mo; acute: < 3 mos; chronic: > 3 mos; delayed onset (onset > 6 mos after trauma): worse prognosis, higher comorbidity
4. High comorbidity:
   a) Davidson & Foa (1991): dep most common, followed by other anxiety dis, pers dis, alc; also schiz, schizophreniform, somatization
   b) has been Q where fit: anxiety disorder, dissociative, special chapter for stress-related dis
   c) Green et al. (1989): Vietnam vets: 29% PTSD - increased risk: if on special assignment, grotesque sits - high rate dep, alc, other disorders (3.6% PTSD only); 11% comorbid antisocial pd
5. Q of pre-disposing factors such as pers dis; family hx (Maxmen: 95% have pre-existing psychiatric disorder)

6. Treatment:
   a) debriefing after trauma may help prevent PTSD: Critical incident stress debriefing; recent research suggest not prevent development PTSD
   b) **Rivaz-Vazquez (2001):** is a complex disorder, with varied sx; medication tx:
      2) sertraline (Zoloft) 1st medication FDA approved for tx PTSD
      3) not FDA approved, but a number of studies suggest Prozac, Paxil, Luvox work
      4) also some case reports for Effexor XR, Serzone and Remeron
   c) other meds: MAOI phenelzine, imipramine, adrenergic blockers (propranolol, clonidine), benzodiazepines: all less desirable because of side effects, not as effective as SSRIs
   d) cognitive and beh approaches effective: individually or in group format
   e) 2 basic approaches to therapy:
      1) work through trauma - thoughts, feelings
      2) deal with here and now, sx management
      1) divides PTSD sx into 2 types sx: phobic and generalized anxiety
      2) use exposure first (systematic desensitization): deals with phobic type sx
      3) then anxiety management techniques (relaxation, also stress inoculation, distraction, breathing retraining, deep muscle relax, social skills training) - address generalized sx of anxiety
   g) Foa: recent work in 1990s, 2000s: PTSD results from failure to adequately process the trauma; her recent tx
approach focuses on narratives and pt's understanding of the trauma – how they fit it into the story of their life

h) EMDR: Eye Movement Desensitization and Reprocessing
   1) developed by Shapiro (1989; 1991): generate rhythmic eye movements, use imagery, generation of neg/pos cogs/beliefs; claims can "cure" in as few as one session; DK mechanism effect
   2) has restricted use of EMDR to those who have received special training and restricted others from teaching EMDR or using it in research (claims may be dangerous if not adequately trained)
   3) DeBell & Jones (1997) review of the literature review of 7 studies of EMDR: results of their review mixed: not as + as Shapiro's; may help phobias and PTSD; need research: dismantling studies and looking at mech of action; no evidence EMDR more dangerous than other txs