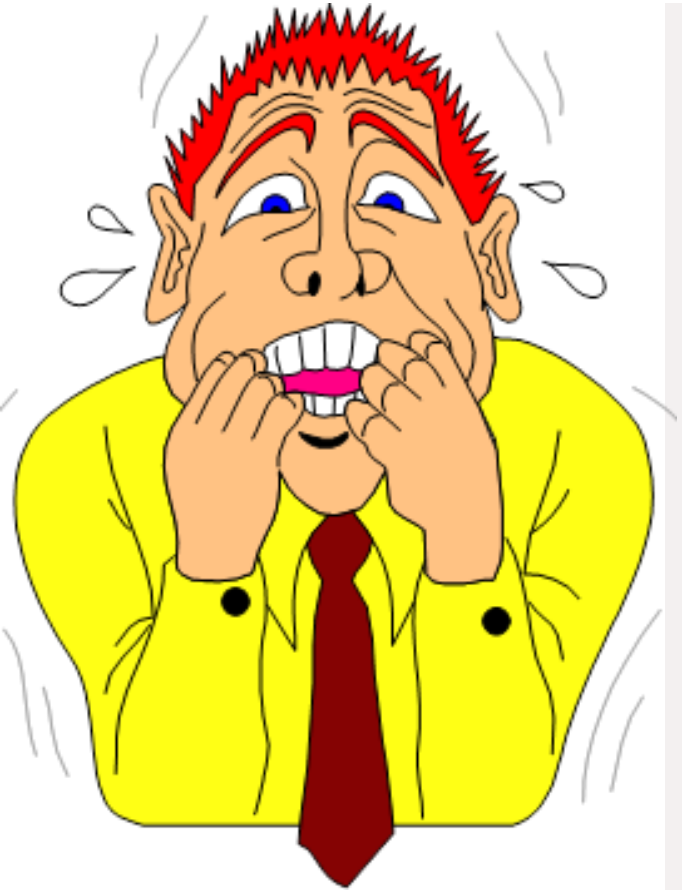
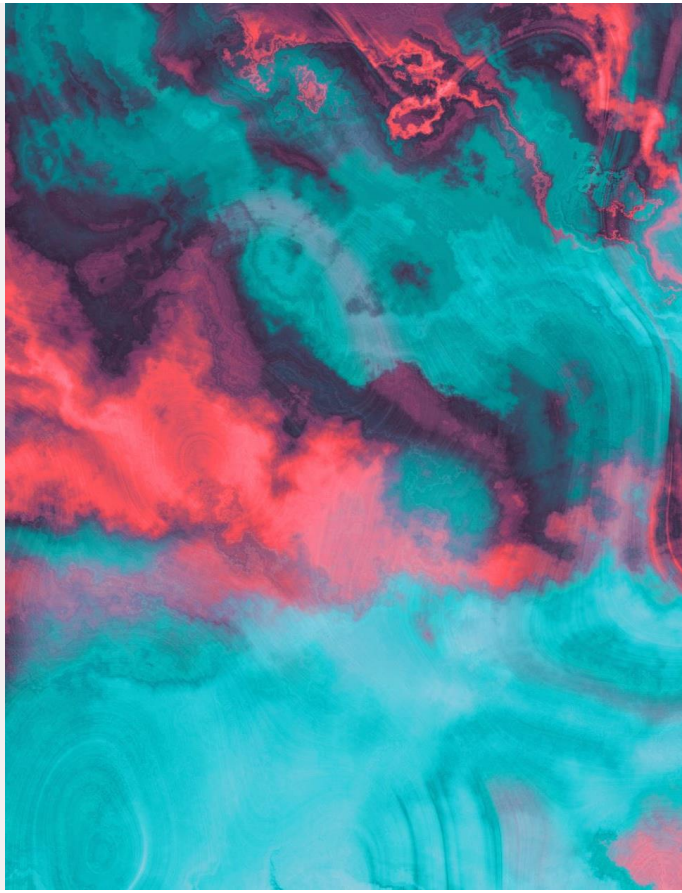


Panic attack- Panic disorder

ANXIETY DISORDERS



Watch clips

- https://youtu.be/_Pw-2gCoEP8?si=hrEQDh_-2eMHtjF4
- https://www.britannica.com/video/221612/Panic-attack

ANXIETY:
SYMPTOM versus DISORDER

Everyone experiences the symptom of anxiety on occasion in response to situations, stress

The symptoms of anxiety can accompany many other primary causes – depression, adjustment disorder, life stress, financial concerns, job difficulties, …

Anxiety DISORDERS are very different !

ANXIETY

Commonly experienced emotion

Common symptom secondary to medical or psychiatric disorders

Normal response to situations

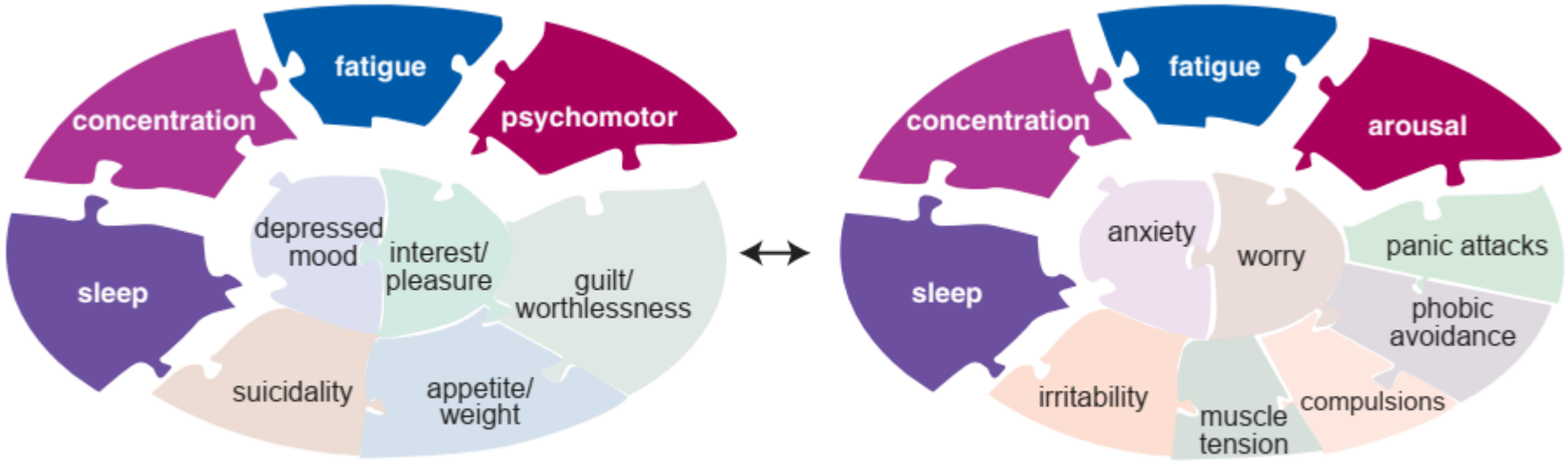
Abnormal response to stressor (ADJUSTMENT DISORDER)

Primary anxiety disorders

Primary Anxiety Disorders

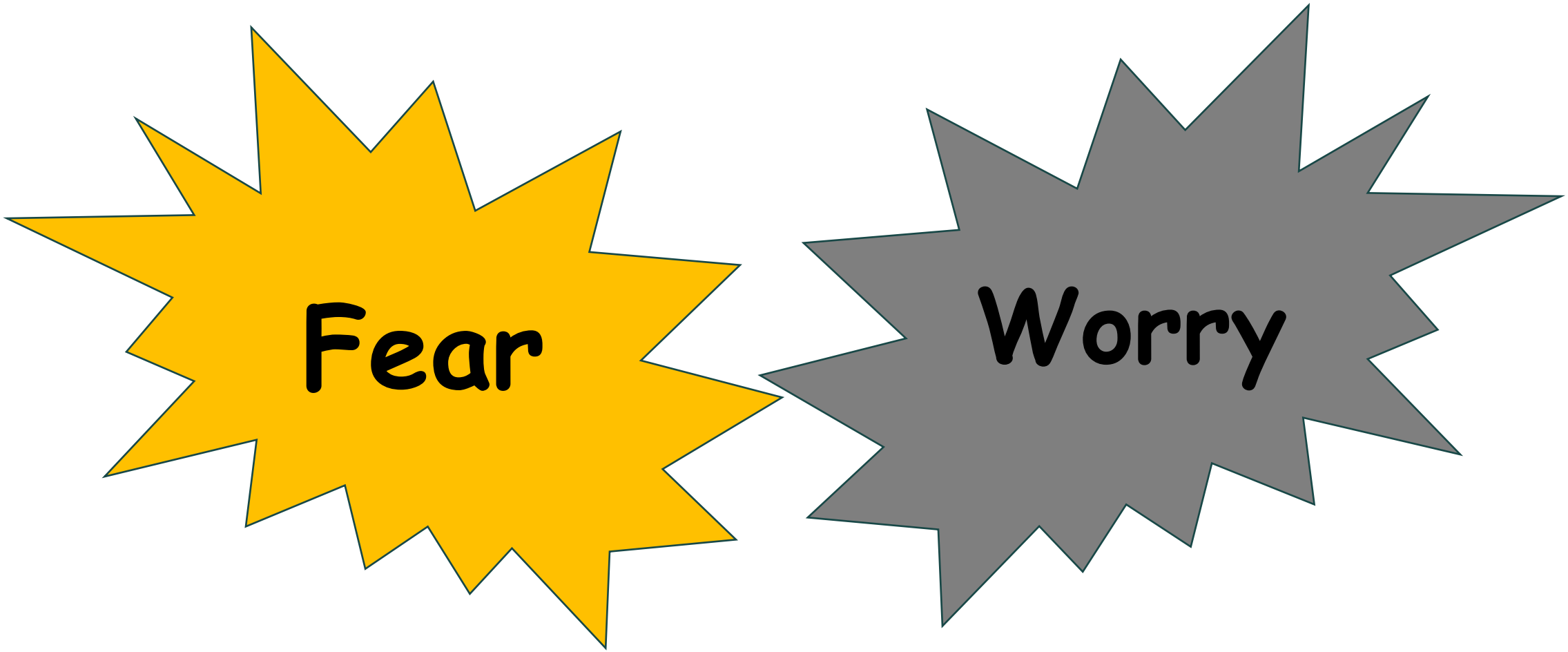
- Generalized anxiety disorder (GAD)
- Panic disorder (PD)
- Specific Phobia
- Social Phobia = social anxiety disorder (SAD)
- Posttraumatic stress disorder (PTSD)
- Obsessive-compulsive disorder (OCD)

Introduction



major depressive disorder

anxiety disorders



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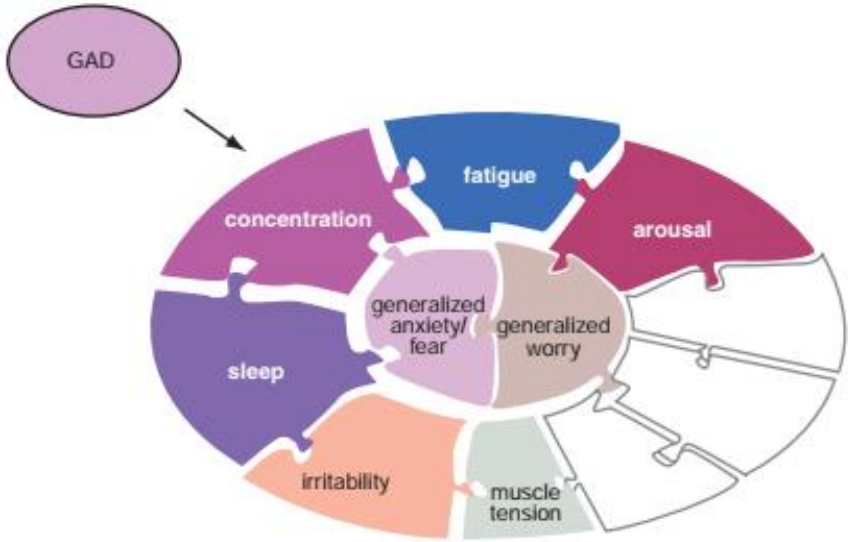
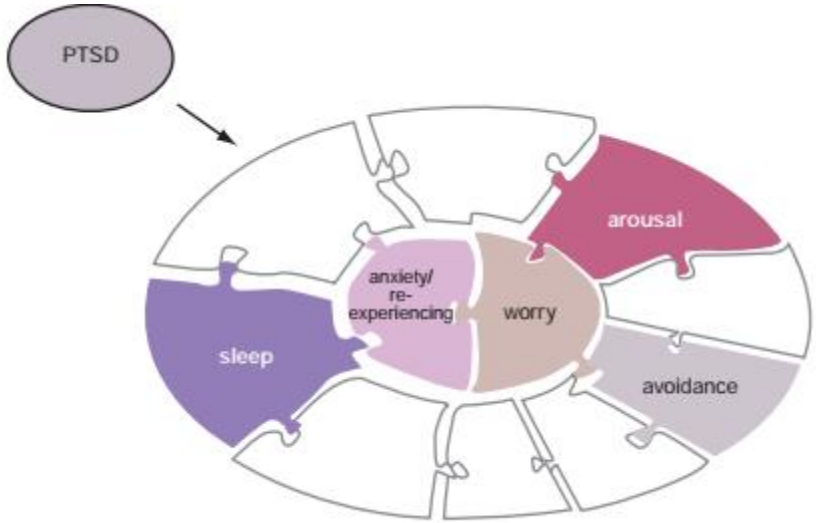
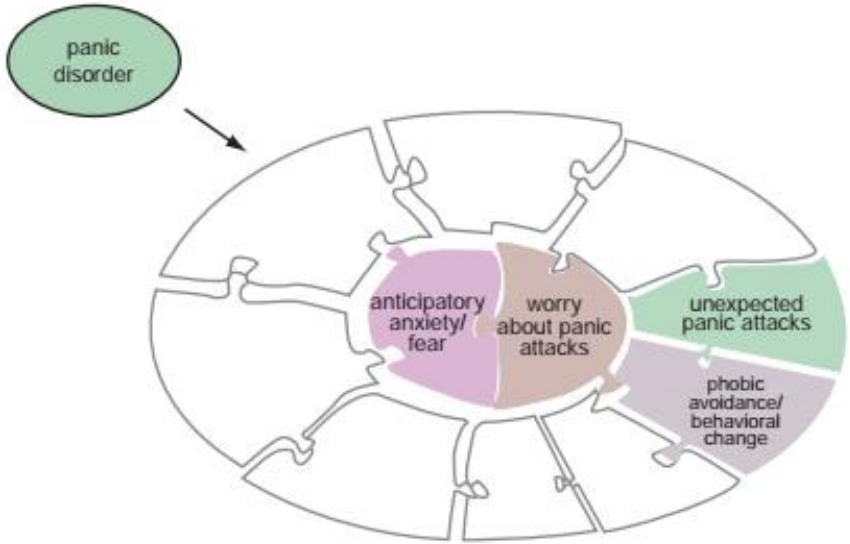
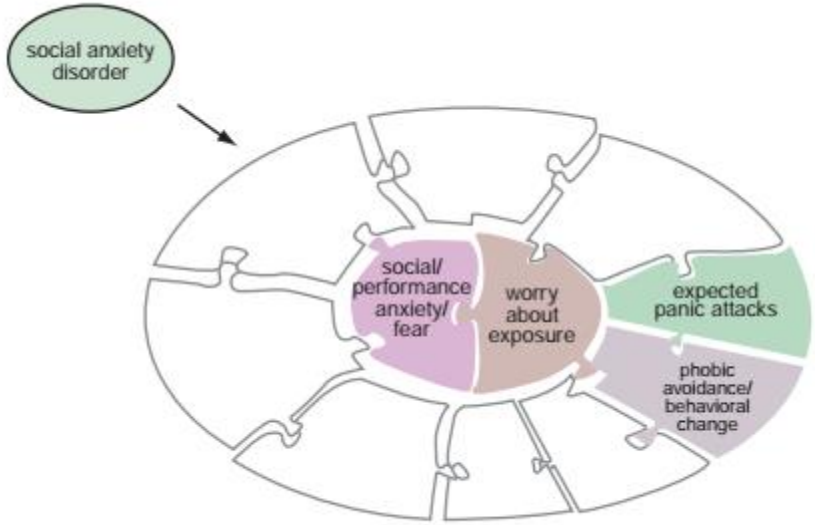

GAD
concentration
fatigue
arousal
sleep
generalized anxiety/ fear
generalized worry
irritability
muscle tension


PTSD
arousal
anxiety/ re-experiencing
worry
sleep
avoidance


panic disorder
anticipatory anxiety/ fear
worry about panic attacks
unexpected panic attacks
phobic avoidance/ behavioral change


social anxiety disorder
social/ performance anxiety/ fear
worry about exposure
expected panic attacks
phobic avoidance/ behavioral change

Pathophysiology

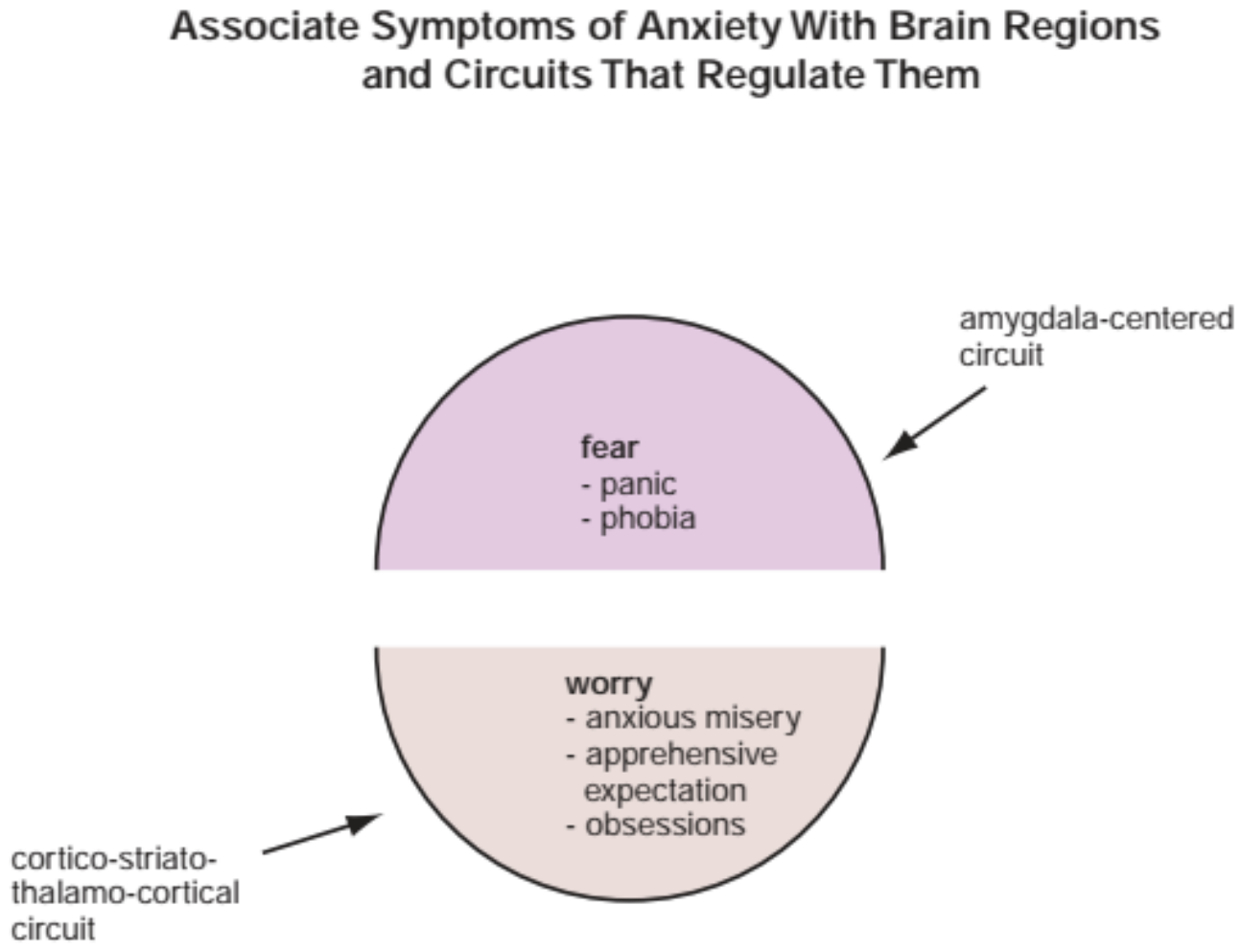
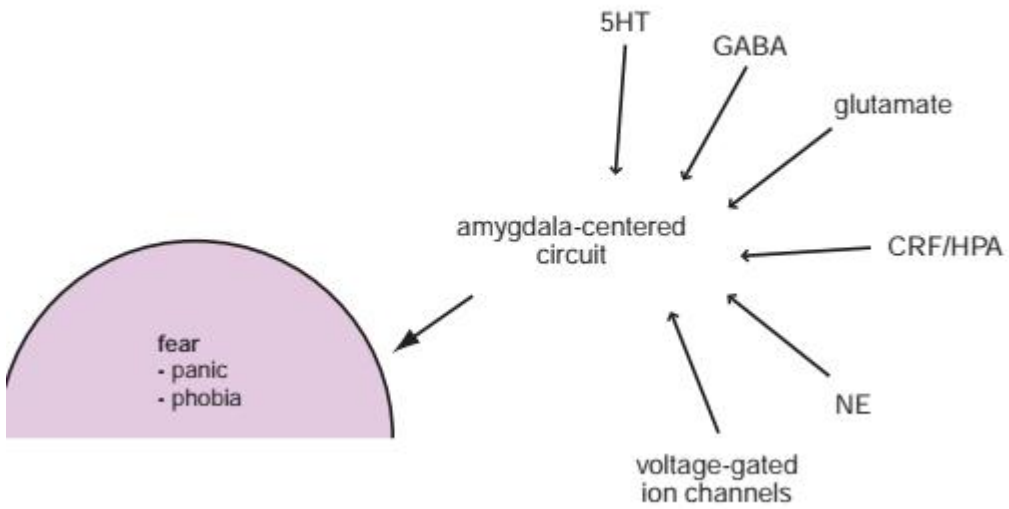
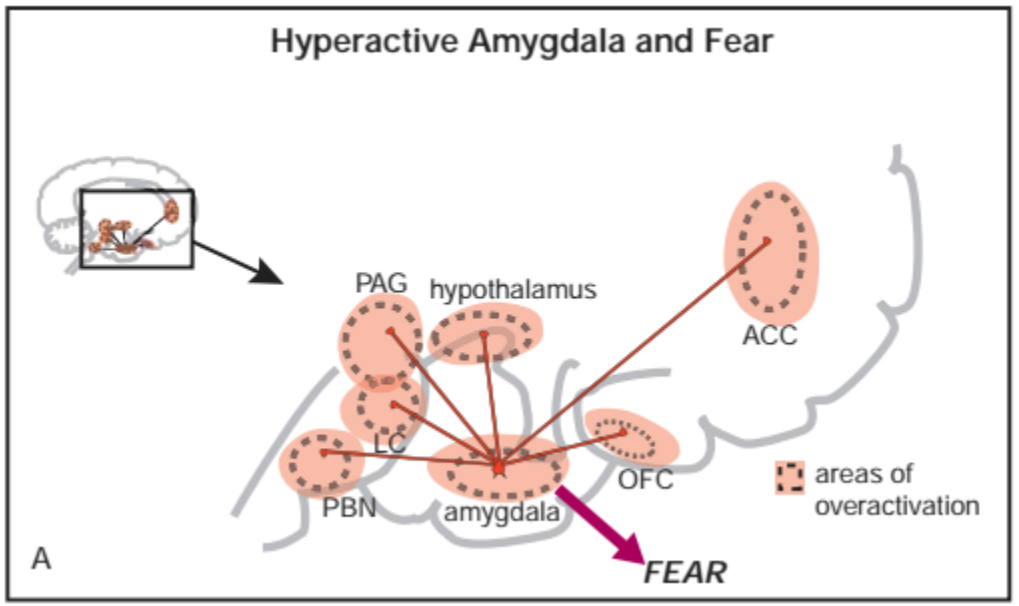
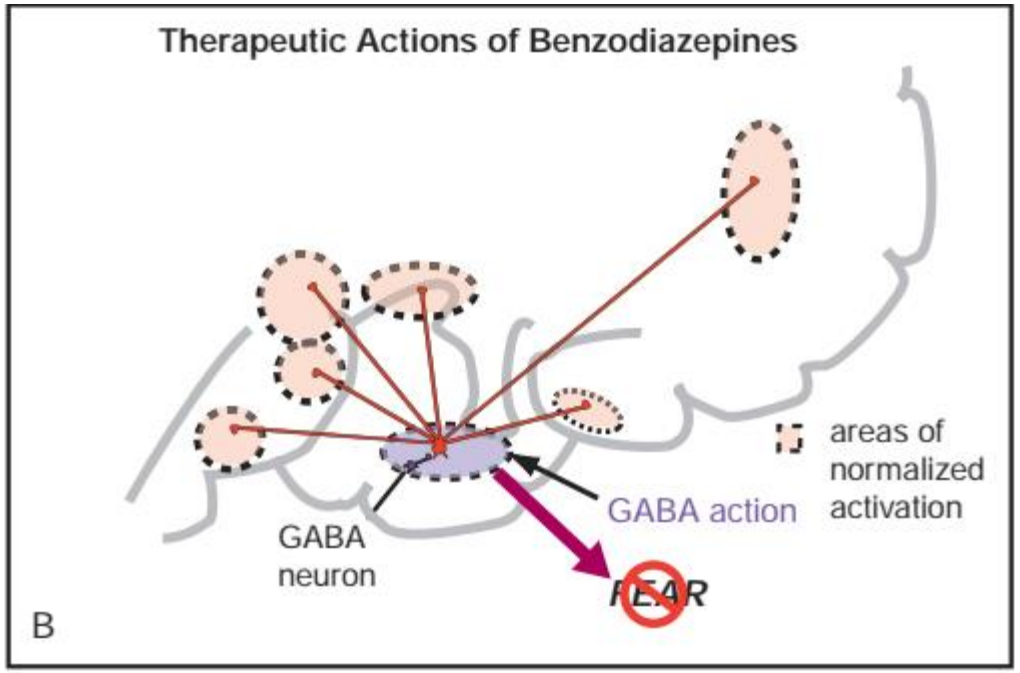
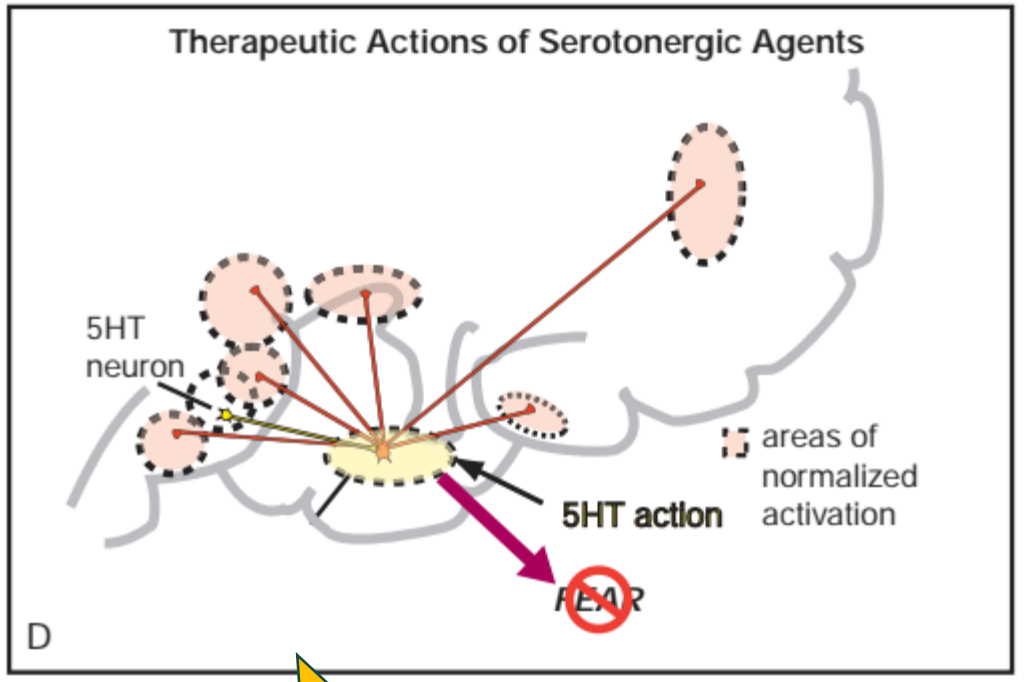


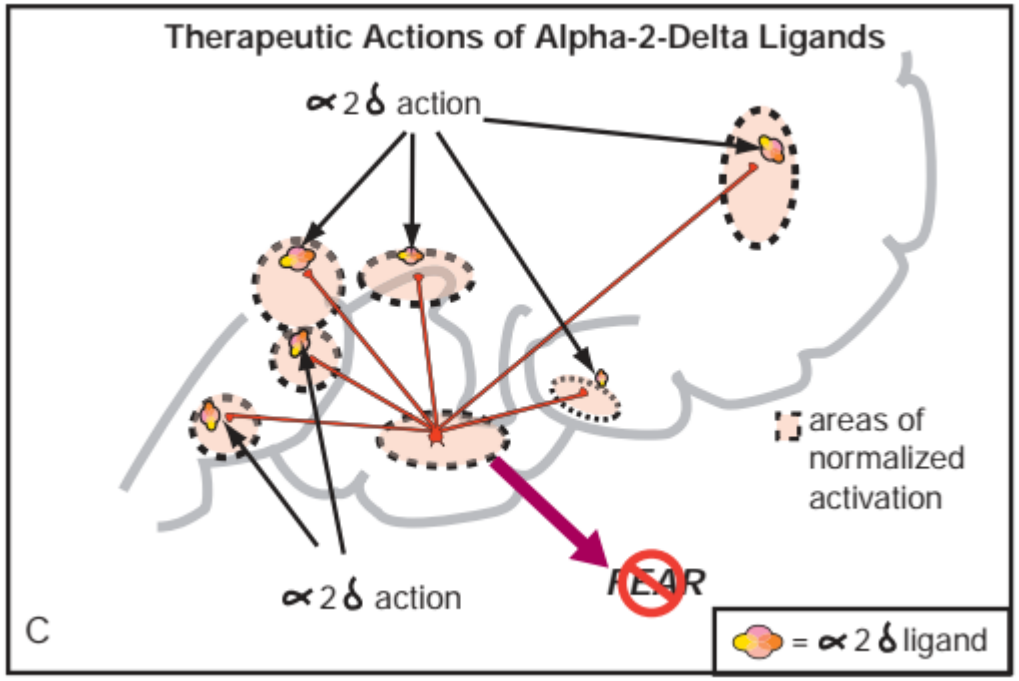
Figure 9-7. Linking anxiety symptoms to circuits. Anxiety and fear symptoms (e.g., panic, phobias) are regulated by an amygdala-centered circuit. Worry, on the other hand, is regulated by a cortico-striato-thalamo-cortical (CSTC) loop. These circuits may be involved in all anxiety disorders, with the different phenotypes reflecting not unique circuitry but rather divergent malfunctioning within those circuits.


5HT
GABA
glutamate
amygdala-centered
circuit
CRF/HPA
fear
- panic
- phobia
NE
voltage-gated
ion channels


Hyperactive Amygdala and Fear
PAG
hypothalamus
ACC
LC
OFC
areas of
overactivation
PBN
amygdala
A
FEAR


Therapeutic Actions of Benzodiazepines
areas of normalized activation
GABA action
GABA neuron
FEAR
B


Therapeutic Actions of Serotonergic Agents
5HT neuron
areas of normalized activation
5HT action
FEAR
D


Therapeutic Actions of Alpha-2-Delta Ligands
∝2δ action
areas of normalized activation
∝2δ action
FEAR
= ∝2δ ligand
C


Fear

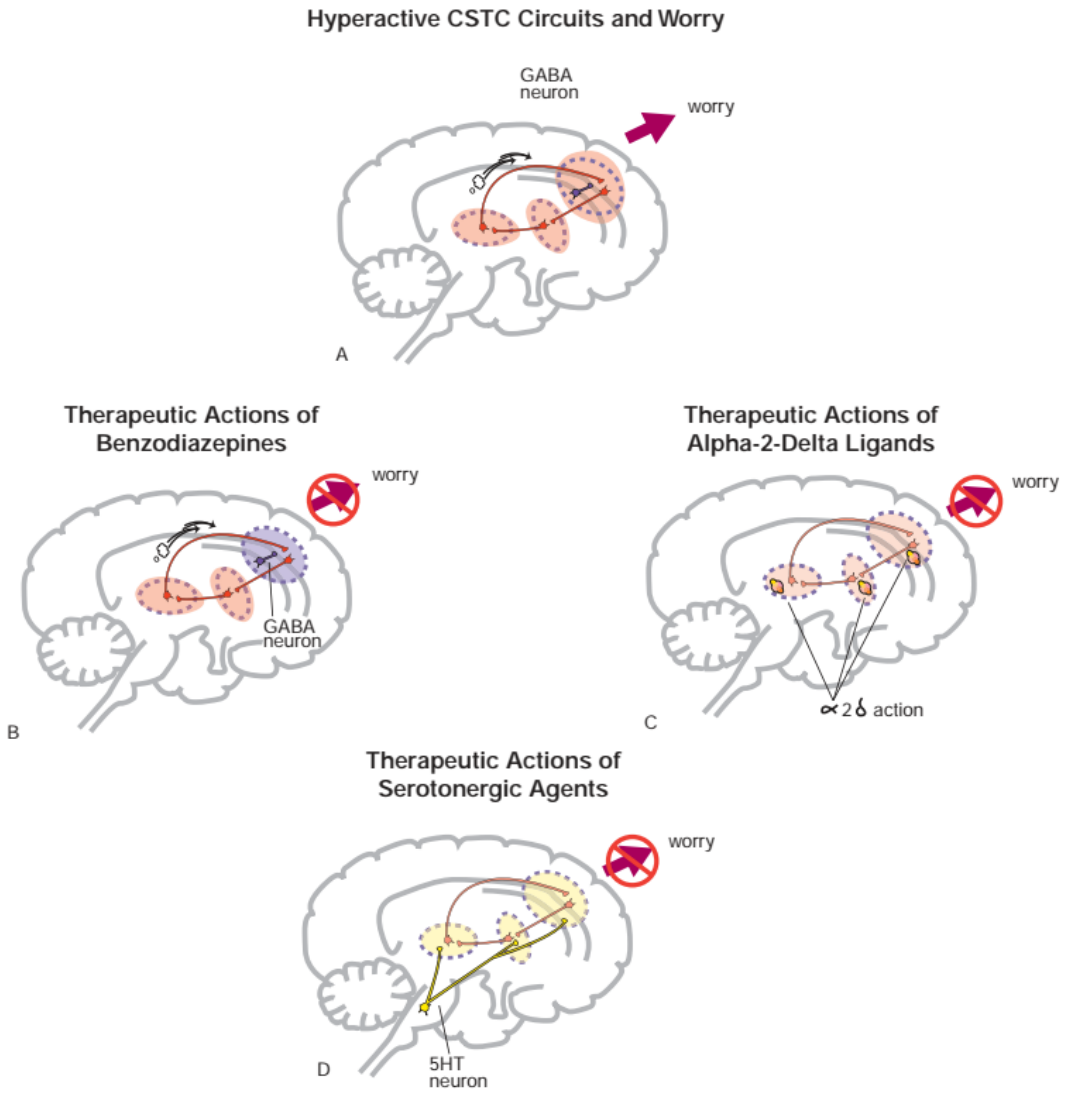
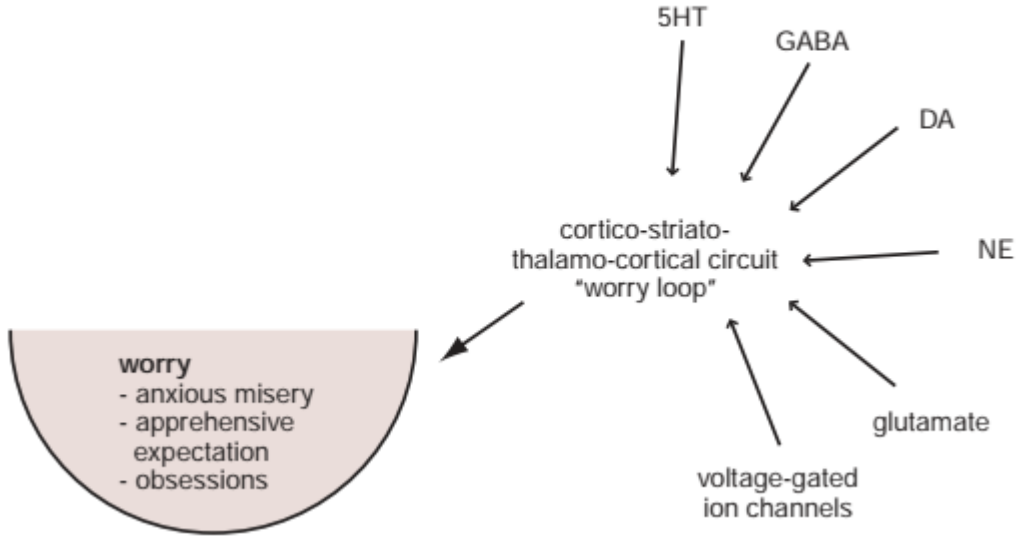


Figure 9-26. Potential therapeutic actions of anxiolytics on worry. (A) Pathological worry may be caused by overactivation of cortico-striato-thalamo-cortical (CSTC) circuits. (B) GABAergic agents such as benzodiazepines may alleviate worry by enhancing the actions of inhibitory GABA interneurons within the prefrontal cortex. (C) Agents that bind to the $\alpha_2\delta$ subunit of presynaptic N and P/Q voltage-sensitive calcium channels can block the excessive release of glutamate in CSTC circuits and thereby reduce the symptoms of worry. (D) The prefrontal cortex, striatum, and thalamus receive input from serotonergic neurons, which can have an inhibitory effect on output. Thus, serotonergic agents may alleviate worry by enhancing serotonin input within CSTC circuits.

Worry



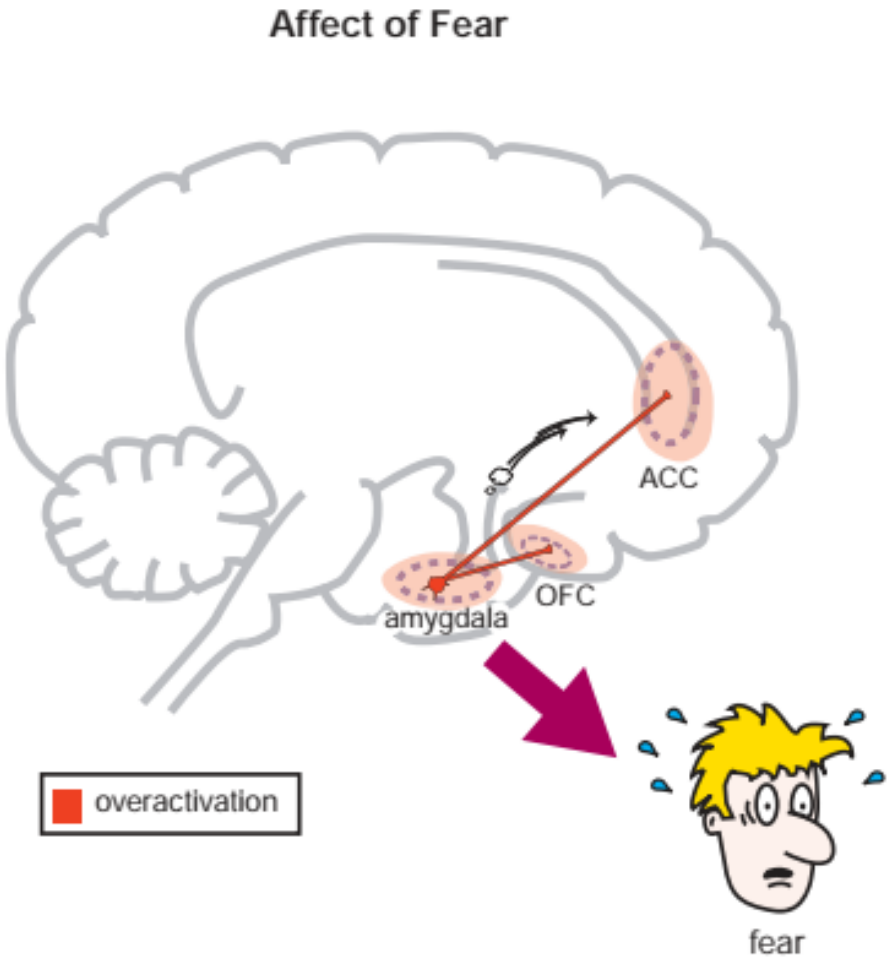


Figure 9-8. Affect of fear. Feelings of fear are regulated by reciprocal connections between the amygdala and the anterior cingulate cortex (ACC) and the amygdala and the orbitofrontal cortex (OFC). Specifically, it may be that overactivation of these circuits produces feelings of fear.

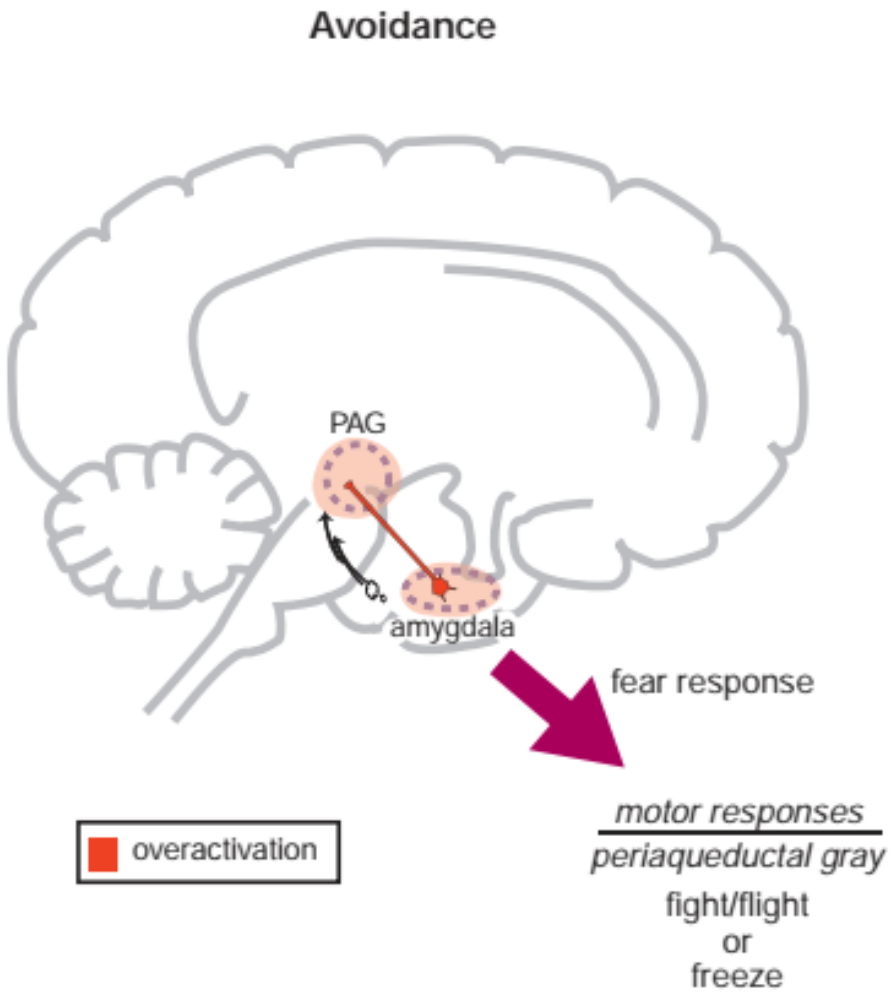


Figure 9-9. Avoidance. Feelings of fear may be expressed through behaviors such as avoidance, which is partly regulated by reciprocal connections between the amygdala and the periaqueductal gray (PAG). Avoidance in this sense is a motor response and may be analogous to freezing under threat. Other motor responses are to fight or to run away (flight) in order to survive threats from the environment.

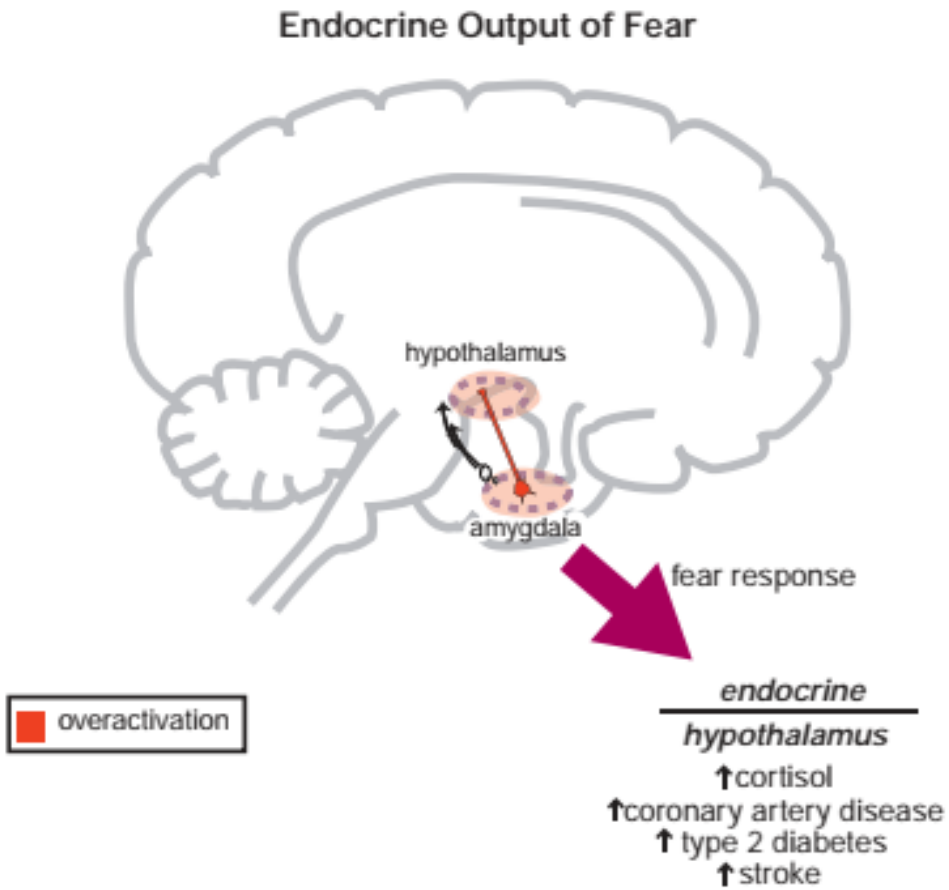


Figure 9-10. Endocrine output of fear. The fear response may be characterized in part by endocrine effects such as increases in cortisol, which occur because of amygdala activation of the hypothalamic–pituitary–adrenal (HPA) axis. Prolonged HPA activation and cortisol release can have significant health implications, such as increased risk of coronary artery disease, type 2 diabetes, and stroke.

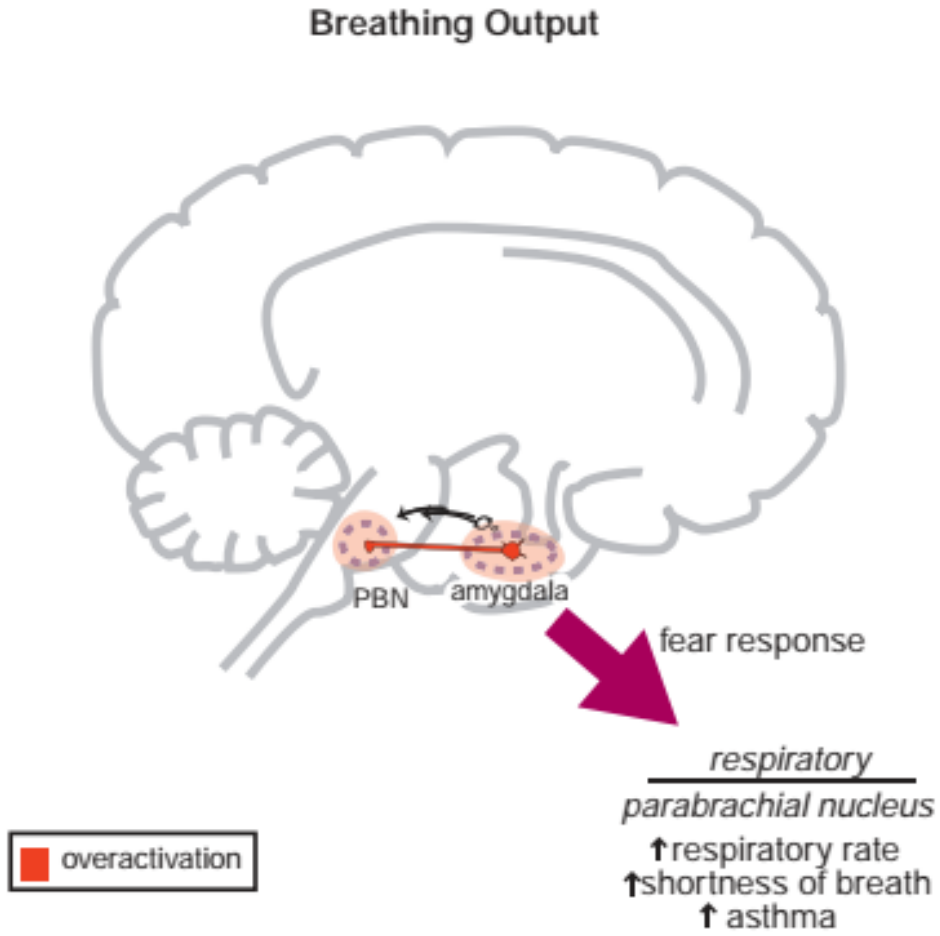


Figure 9-11. Breathing output. Changes in respiration may occur during a fear response; these changes are regulated by activation of the parabrachial nucleus (PBN) via the amygdala. Inappropriate or excessive activation of the PBN can lead not only to increases in the rate of respiration but also to symptoms such as shortness of breath, exacerbation of asthma, or a sense of being smothered.

Autonomic Output of Fear

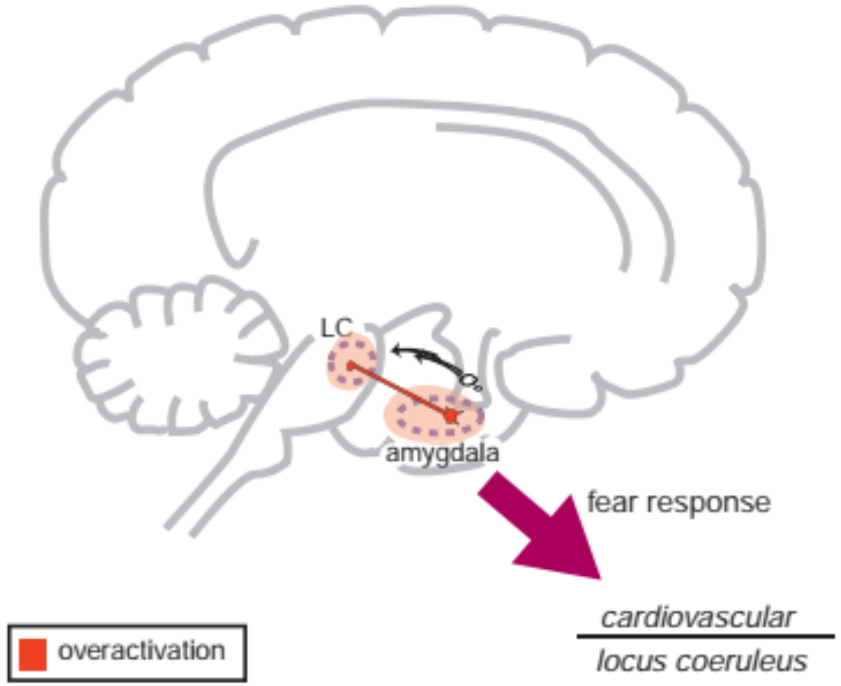


Figure 9-12. Autonomic output of fear. Autonomic responses are typically associated with feelings of fear. These include increases in heart rate (HR) and blood pressure (BP), which are regulated by reciprocal connections between the amygdala and the locus coeruleus (LC). Long-term activation of this circuit may lead to increased risk of atherosclerosis, cardiac ischemia, change in BP, decreased HR variability, myocardial infarction (MI), or even sudden death.

The Hippocampus: An Internal Fearmonger

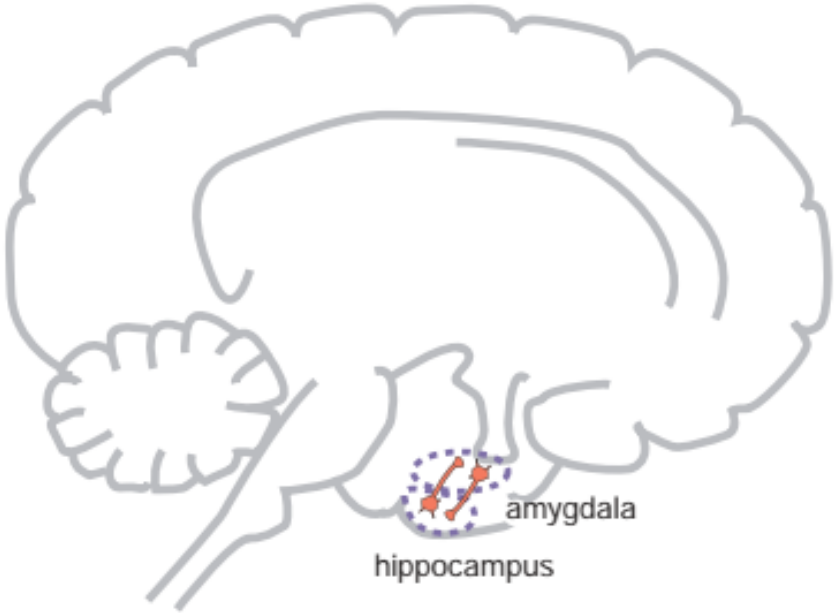


Figure 9-13. The hippocampus and re-experiencing. Anxiety can be triggered not only by an external stimulus but also by an individual's memories. Traumatic memories stored in the hippocampus can activate the amygdala, causing the amygdala, in turn, to activate other brain regions and generate a fear response. This is termed re-experiencing, and it is a particular feature of posttraumatic stress disorder.

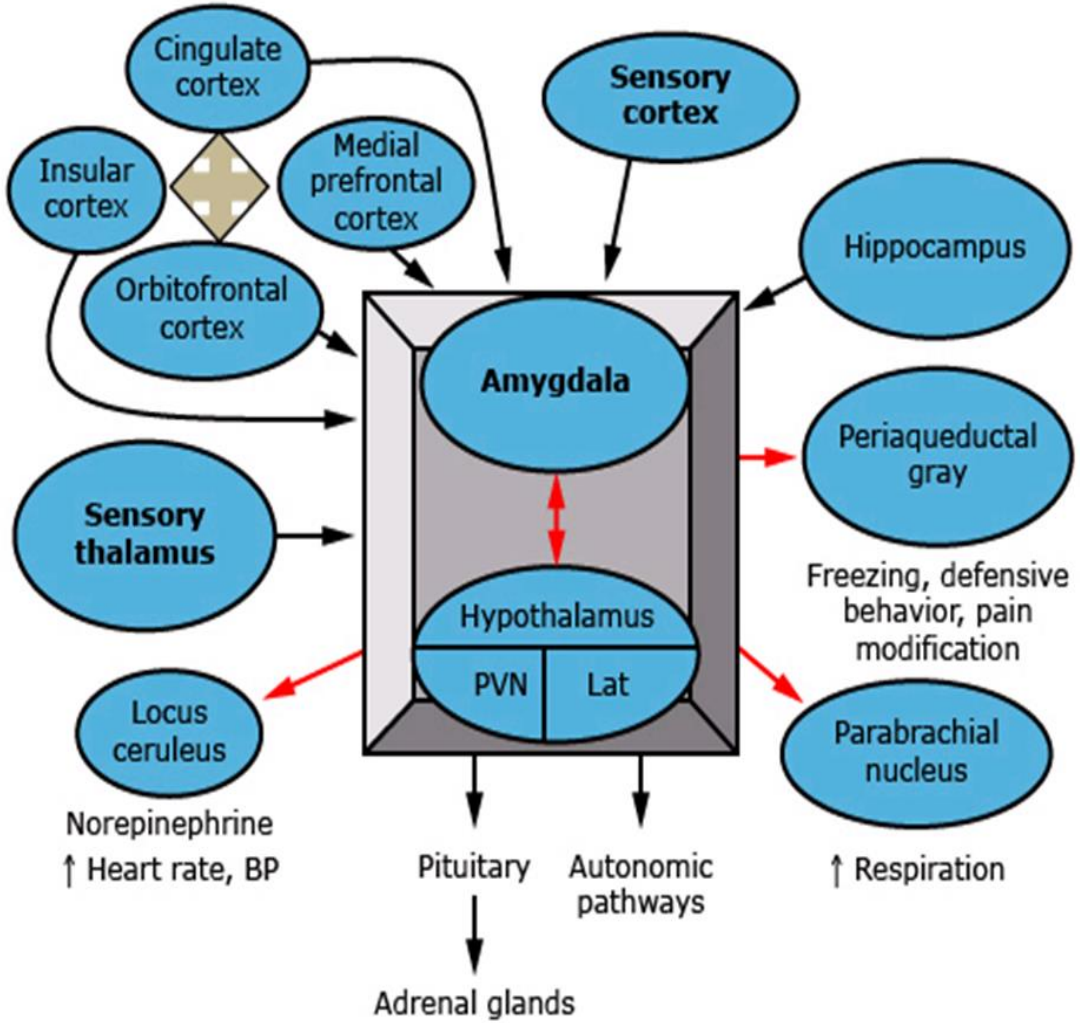
Panic Disorder

- Presence of recurrent, unexpected panic attacks, followed by at least 1 month of persistent concern about having another panic attack, worry about consequences of the panic attacks (e.g., losing control, having a heart attack), or a significant change in behavior related to the attacks
- Also may have agoraphobia (fear of being in places or situations from which escape might be difficult)

Panic Disorder

- Lifetime prevalence between 1.5 and 3.5%
- Age of onset typically between late teens and mid-30s
- Panic disorder is associated with a high rate of ER utilization; most patients think they are having a heart attack; most are given a cardiac workup ending with negative results, sent home with no treatment

Neural circuitry of panic attacks



Original figure modified for this publication. Roy-Byrne, P, Craske, MG, Stein, MB. Panic disorder. Lancet 2006; 368:1023. Illustration used with the permission of Elsevier Inc. All rights reserved.

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Panic Attack

Intense fear or discomfort, in which at least 4 of the following sxs develop abruptly and reached a peak within minutes:

1) Tachycardia
2) Sweating
3) trembling or shaking
4) difficulty breathing
5) feeling of choking
6) chest pain
7) nausea or abdominal distress
8) feeling dizzy or faint
9) derealization or depersonalization
10) fear of losing control or going crazy
11) fear of dying
12) paresthesias (numbness or tingling sensations)
13) chills or hot flushes

Panic disorder : DSM-V

A. Recurrent unexpected panic attacks

B. At least one of the attacks has been followed by a month or more of one or both of the following:

1. Persistent concern or worry about additional panic attacks or their consequences (eg, losing control, having a heart attack, "going crazy").

2. A significant maladaptive change in behavior related to the attacks (eg, behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).

Panic disorder: DSM V

C. The disturbance is not attributable to the physiological effects of a substance (eg, medication or illicit drug) or another medical condition (eg, hyperthyroidism, cardiopulmonary disorders).

D. The disturbance is not better explained by another mental disorder. As examples, the panic attacks do not occur only in response to

- Feared social situations, as in social anxiety disorder
- Circumscribed phobic objects or situations, as in specific phobia;
- Obsessions, as in obsessive-compulsive disorder
- Reminders of traumatic events, as in posttraumatic stress disorder
- Separation from attachment figures, as in separation anxiety disorder

Somatic features

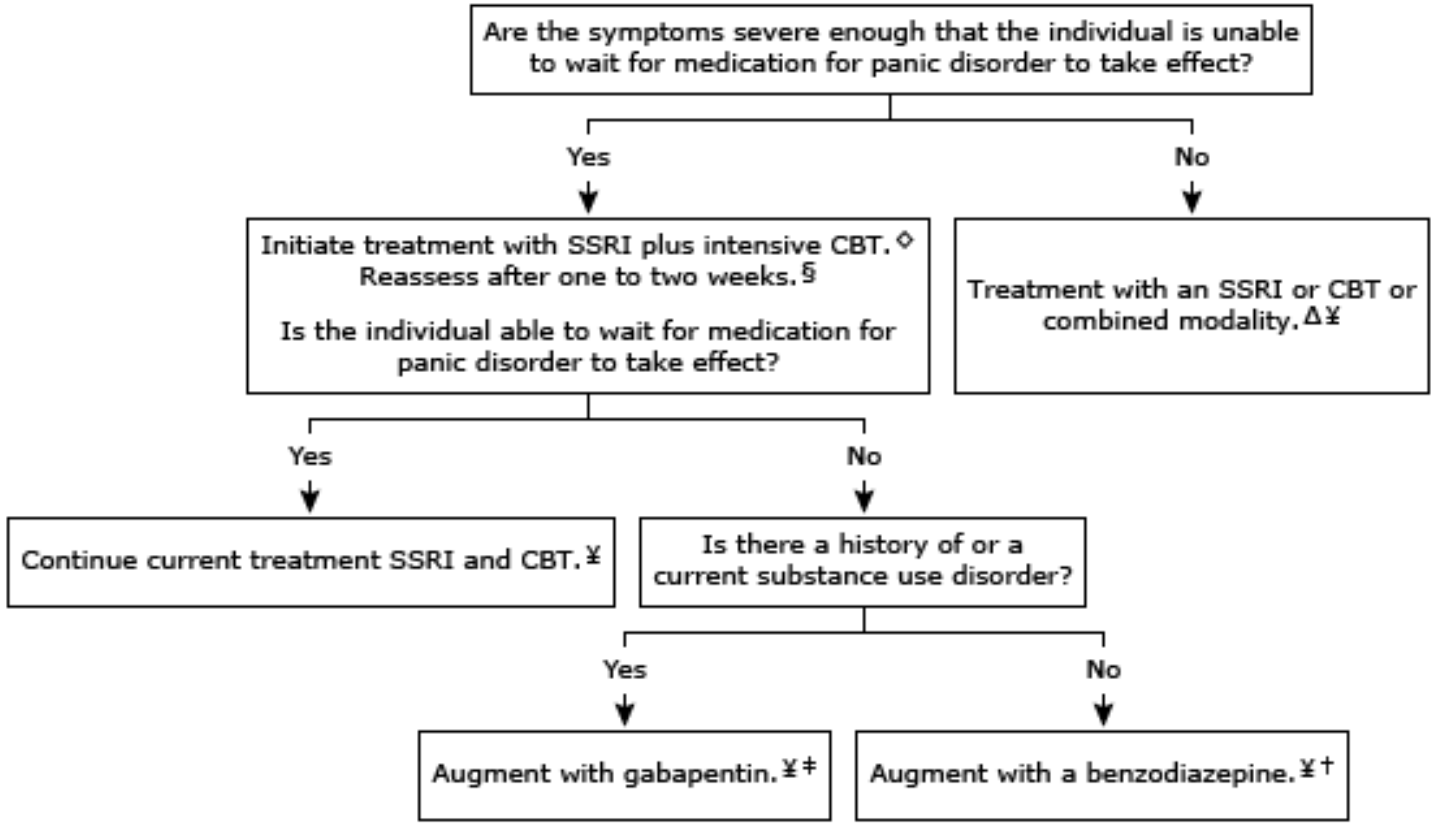
- Cardiac — 39 % (chest pain in 22 %, tachycardia in 25 %)
- Neurologic — 44 % (headaches in 20 %, dizziness in 18 %, faintness and pseudoseizures in 9 %)
- Gastrointestinal - 33 % (epigastric pain in 15 %)

Main objectives of treatment

- minimize attack frequency, anticipatory anxiety, and phobic avoidance thereby improving functioning.

PD treatment

Choosing initial treatment for individuals with panic disorder with or without agoraphobia*¶Δ



SSRI: selective serotonin reuptake inhibitor; CBT: cognitive-behavioral therapy.

* This algorithm assumes that the individual does not have active suicidal ideation, plan, or intent.

¶ In some cases, symptoms of panic disorder do not cause significant psychosocial distress or interfere with functioning. These individuals may reasonably elect to forego treatment and monitor symptoms.

Δ We treat all individuals with comorbid disorders (eg, major depression, posttraumatic stress disorder, anxiety) with an SSRI plus CBT. We take into account patient preference in all treatment decisions.

◇ In cases with extreme stress, where the individual is unable to wait for medications to take effect, we treat with intensive CBT (eg, twice weekly or more) providing psychoeducation and support.

§ In cases when the individual is unable to wait one to two weeks (eg, due to severe symptoms), we move on to the next treatment decision more rapidly.

¥ Refer to UpToDate content (and associated algorithm) for subsequent treatment.

‡ We prefer gabapentin over pregabalin due to its lower likelihood for misuse than pregabalin. Mirtazapine is a reasonable alternative agent for individuals with prominent insomnia. Refer to UpToDate content for dose and titration of each of these agents.

† Our preference among benzodiazepines is clonazepam (refer to UpToDate content for administration and dosing of clonazepam).

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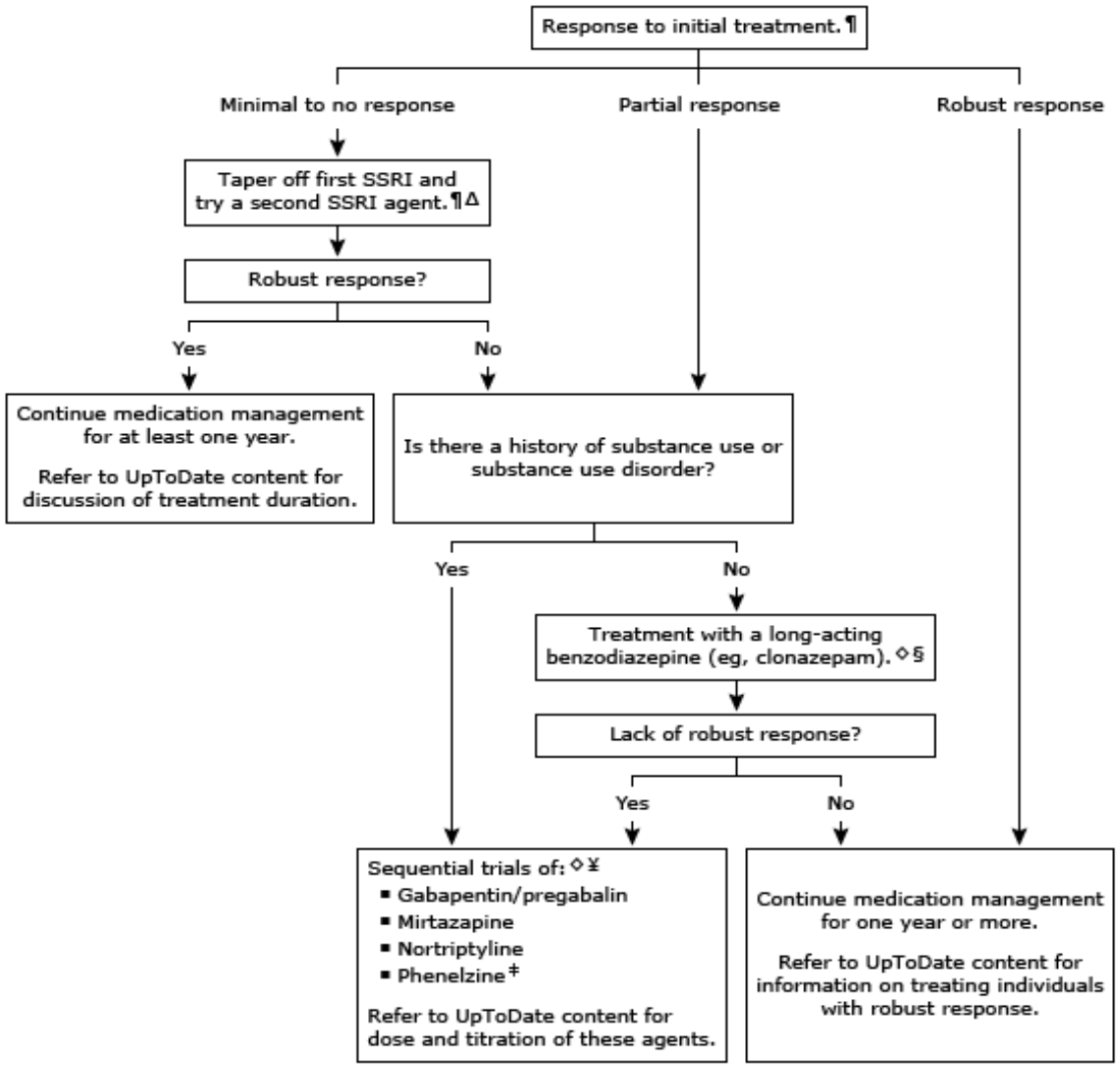
Subsequent pharmacologic management of panic disorder*



* This algorithm reviews the approach to choosing pharmacologic management of panic disorder after initial treatment (with an SSRI and initial stabilization of severe symptoms). Augmentation with cognitive-behavioral therapy is a reasonable option at any point (if not already done).

¶ We consider an adequate therapeutic trial to be four to six weeks at the maximum tolerated dose within the dose range. Refer to UpToDate content for information on medication starting doses and target dose range.

Δ Our preference is to try sequential trials of two SSRI followed by one SNRI (ie venlafaxine) for those with minimal to no response to adequate medication trials. Refer to UpToDate content for discussion of dose and titration of SSRIs and SNRIs.

◇ We typically treat with sequential augmentation trials in individuals with partial response. We typically switch medications sequentially in individuals with minimal to no response to medications.

§ Our preference is to use the long-acting agent clonazepam. Acceptable alternative option is lorazepam. Refer to UpToDate content for dose and titration of benzodiazepines.

¥ Limited data support these agents in the treatment of panic disorder.

‡ Individuals must be able to adhere to medication and dietary restrictions. MAOIs cannot be used concurrently with SSRIs/SNRIs/tricyclic antidepressants. Adequate wash-out period prior to beginning MAOI is based on half-life of medication discontinuing. Refer to UpToDate content for more information on prescribing MAOI.

Treatment of Panic Disorder (CBT/SSRIs)

SSRIs: titrate 2-6 wk usual dose
- Fluoxetine 20 to 40 mg
- Paroxetine 20 to 40 mg
- Sertraline 100 to 200 mg
- Citalopram 20 to 40 mg
- Escitalopram 10 to 20 mg

SNRI:
venlafaxine ER 75-150 mg / d

reduces all three core components of panic disorder (attack frequency, anticipatory anxiety and phobic avoidance)

BZDs:
- clonazepam 2-4 mg/d
- alprazolam 3-10 mg/d

TCAs:
- imipramine 150-300 mg/d
- clomipramine 50-150 mg/d

AEDs
Gabapentin
Pregabalin

Treatment of Panic Disorder

Onset of action fast with BZDs (w/in 1 week)

TCAs and SSRIs take 3-5 weeks for initial response, usually > 10 weeks for maximum response

Drugs can eliminate panic attacks, but not the agoraphobia (a learned behavior)

Duration of treatment panic disorder at least 1 year