

Emotional Disorders Following Stroke



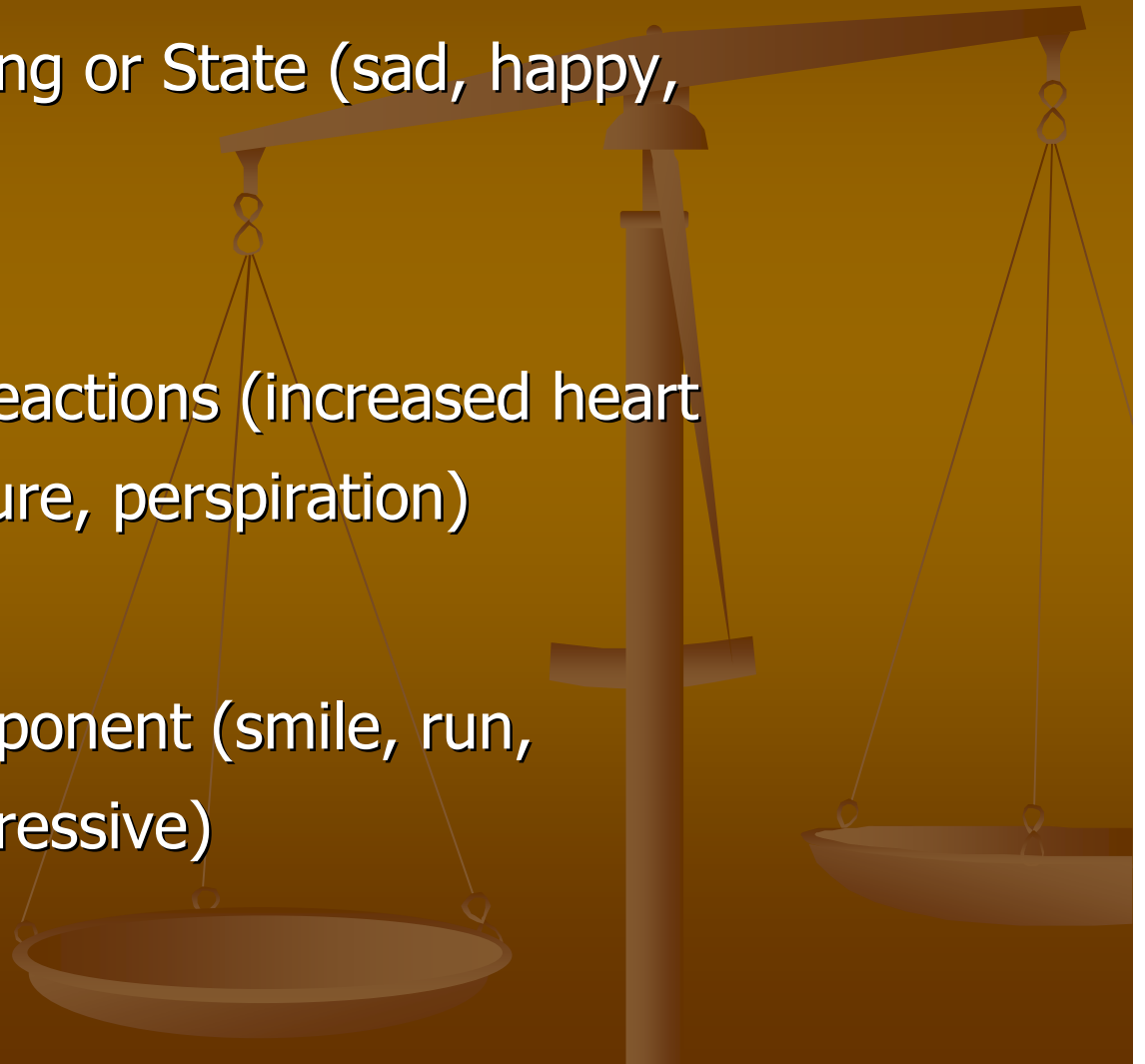
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Emotions

Subjective Feeling or State (sad, happy, afraid)

Physiological Reactions (increased heart rate, blood pressure, perspiration)

Behavioral Component (smile, run, aggressive)



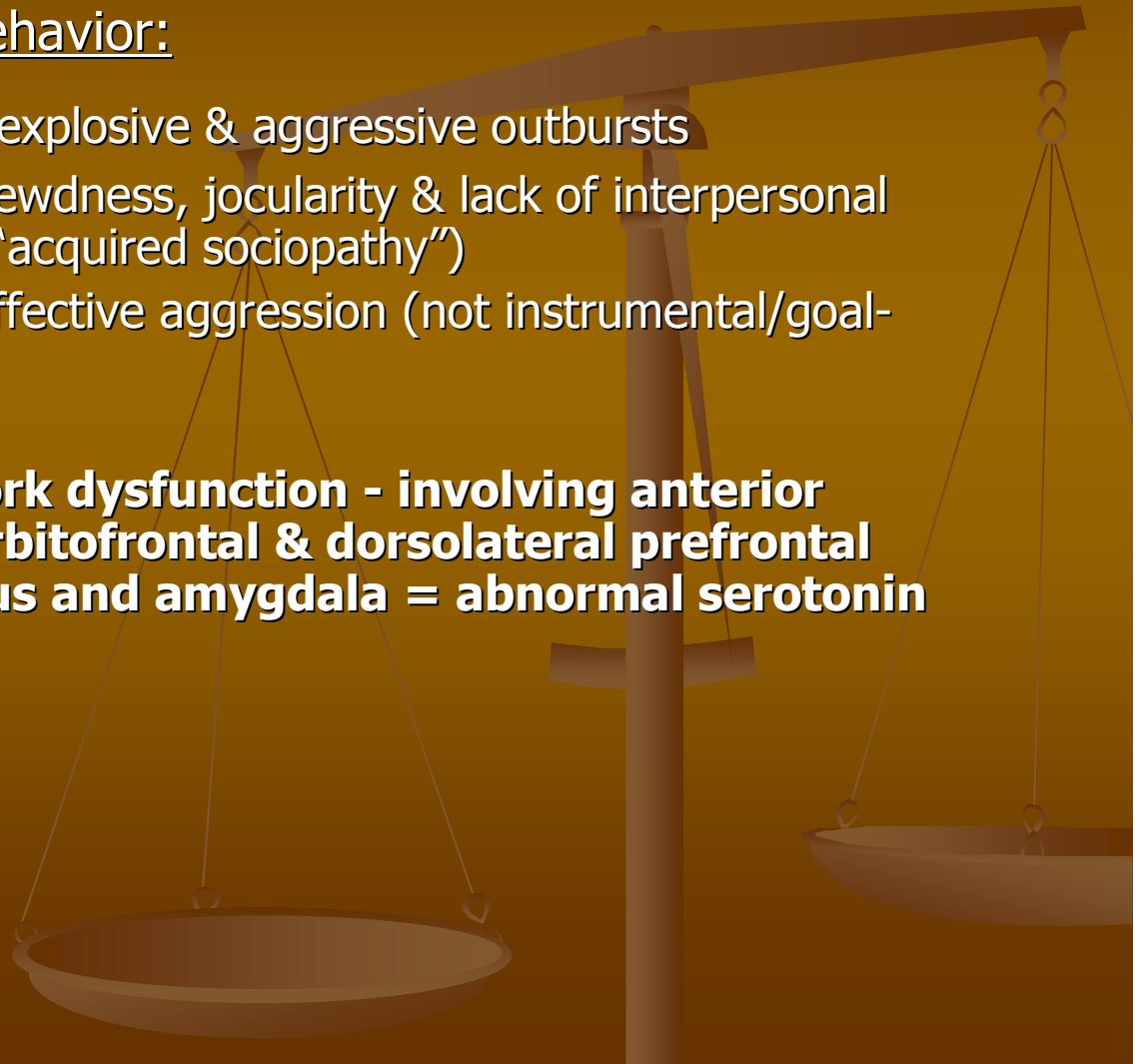
Bilateral Disorders



- Apathy: a lack of feeling, emotion, interest or concern
 - Apathy without depression: 11% of CVA patients
 - **Frontal damage - associated with anterior cerebral artery lesions:** classic cause of apathy
 - Major form = akinetic mutism - associated with **lesions of cingulate gyrus and mesial premotor area**
 - 4 pathological subgroups:
 - **Unilateral cingulate gyrus, suppl motor area & mesial motor areas**
 - **R CVA**
 - **Bilateral lesion of the amygdala** and anterior temporal lobes
 - **Frontal lobe damage**
- Athymormia: “loss of self-psychic activation”: apathetic, aspontaneous and indifferent behavior with motor and affective drive loss, without anxiety or suffering, but adequate activity can be obtained on hetero-stimulation
 - Only **bilateral lesions (pallidal & putamen, caudate, thalamus infarcts)**

Bilateral Disorders

- Violent & Aggressive Behavior:
 - Poor impulse control, explosive & aggressive outbursts
 - Inappropriate verbal lewdness, jocularity & lack of interpersonal sensitivity (Damasio: “acquired sociopathy”)
 - Includes reactive or affective aggression (not instrumental/goal-based)
 - 32% of CVA sample
 - **Frontolimbic network dysfunction - involving anterior cingulate cortex, orbitofrontal & dorsolateral prefrontal cortex, hippocampus and amygdala = abnormal serotonin transmission**



Bilateral Disorders

- Pseudobulbar Palsy (Emotional Lability): uncontrollable laughing, crying (may be unrelated to mood or out of proportion to situation)
 - **Bilateral lesions located in the corticonuclear tracks between the cortex and brainstem**
 - Patients also usually have dysarthria and dysphagia
 - Acute CVA (up to 40% of patients); 6 months post CVA (15-21% of patients)

- Klüver-Bucy Syndrome: at least 3 symptoms present
 - Loss of fear or anxiety
 - Hyperorality (examine objects by mouth)
 - Aberrant sexual behavior (increased activity and/or inappropriate sexual object choices)
 - Hypermetamorphosis (excess visual exploration of environment)
 - Bulimia or loss of alimentary selectivity
 - Psychic blindness (failure to recognize emotional stimuli)
 - Can occur in incomplete form
 - **Bilateral temporal lesions**

Right - Hemisphere Disorders

■ Expressive / Motor Aprosodia:

- Decreased emotional inflection (appears inexpressive or sad)
- Also has analogue in facial (emotional) expressive deficits
- **R frontal lesions**

■ Receptive Aprosodia:

- Indifference to the disease, lack of concern with stroke care
- Also has analogue in facial (emotional) processing deficits
- **R parietal lesions**

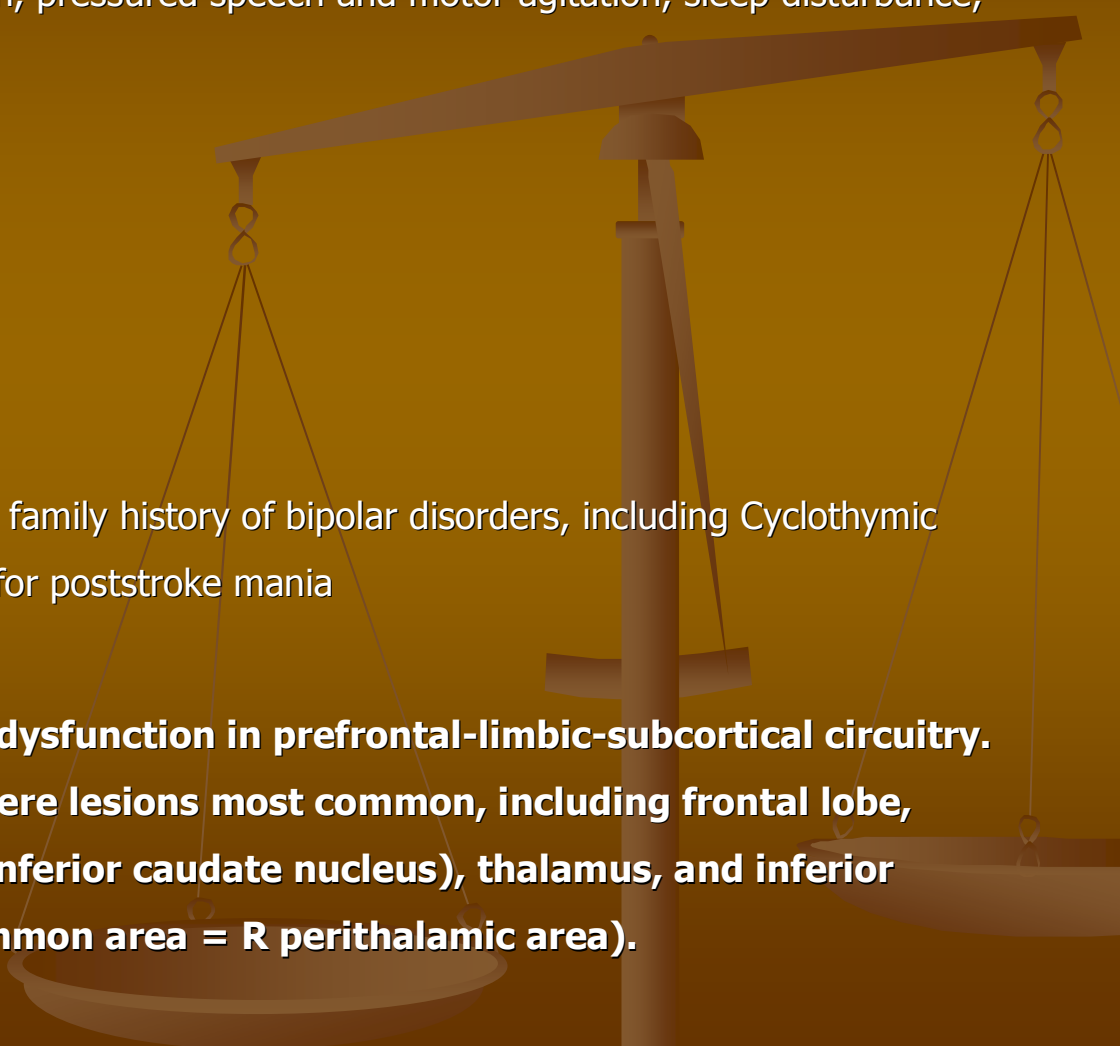
■ Anosognosia:

- Inability to recognize the affective tonality of a sentence → misunderstanding or indifference to others
- **R parietal lesions**



Right - Hemisphere Disorders

■ Poststroke Mania (Secondary Mania):

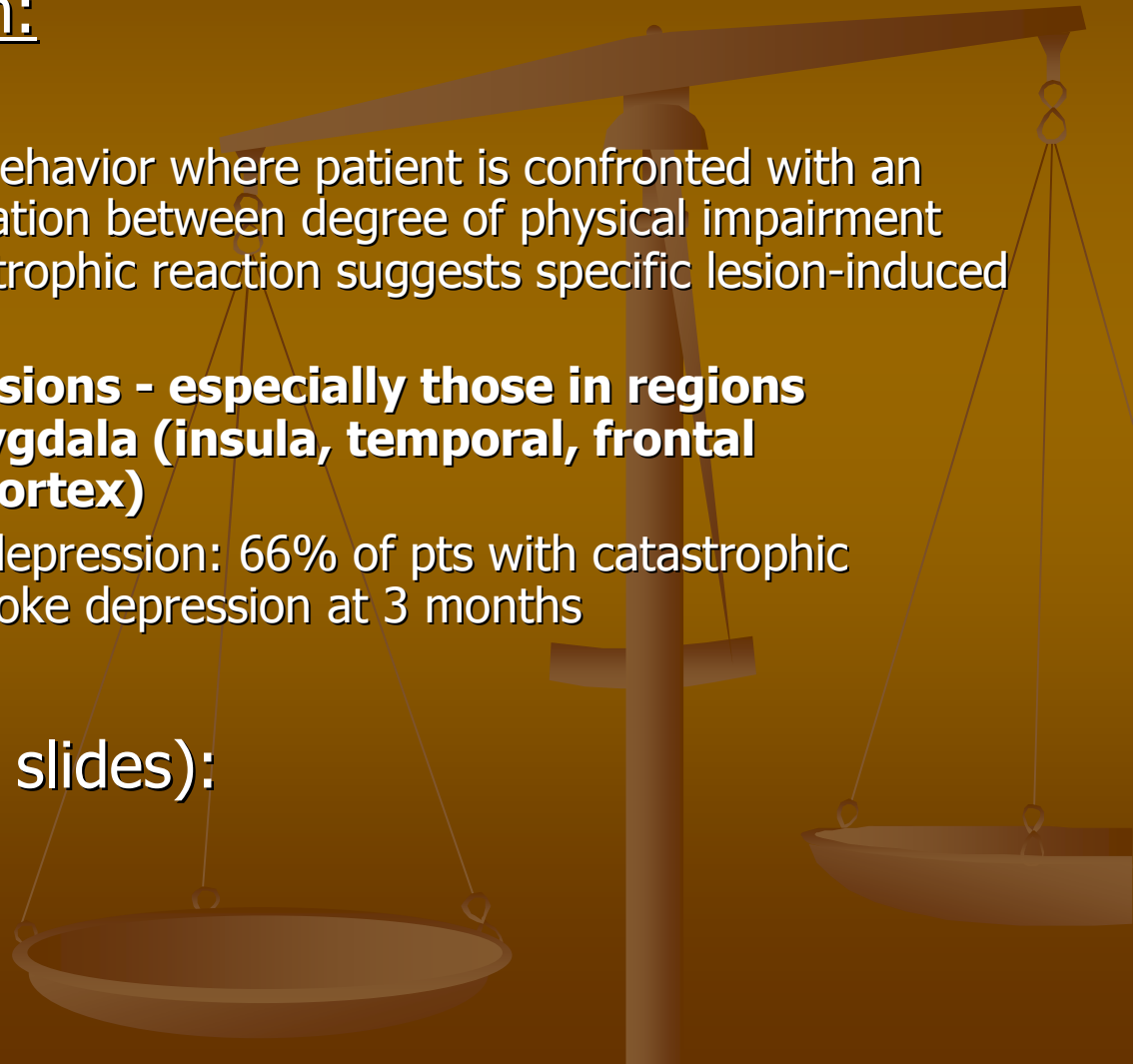
- Symptoms: thought acceleration, pressured speech and motor agitation, sleep disturbance, social & sexual disinhibition
 - Incidence: 1% of CVA patients
 - Duration: 2-4 months
 - Risks: patients with personal or family history of bipolar disorders, including Cyclothymic temperaments could be at risk for poststroke mania
 - **Lesions: bipolar disorder = dysfunction in prefrontal-limbic-subcortical circuitry. In CVA patients: R hemisphere lesions most common, including frontal lobe, basal ganglia (particularly inferior caudate nucleus), thalamus, and inferior temporal regions (most common area = R perithalamic area).**
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Left-Hemisphere Disorders

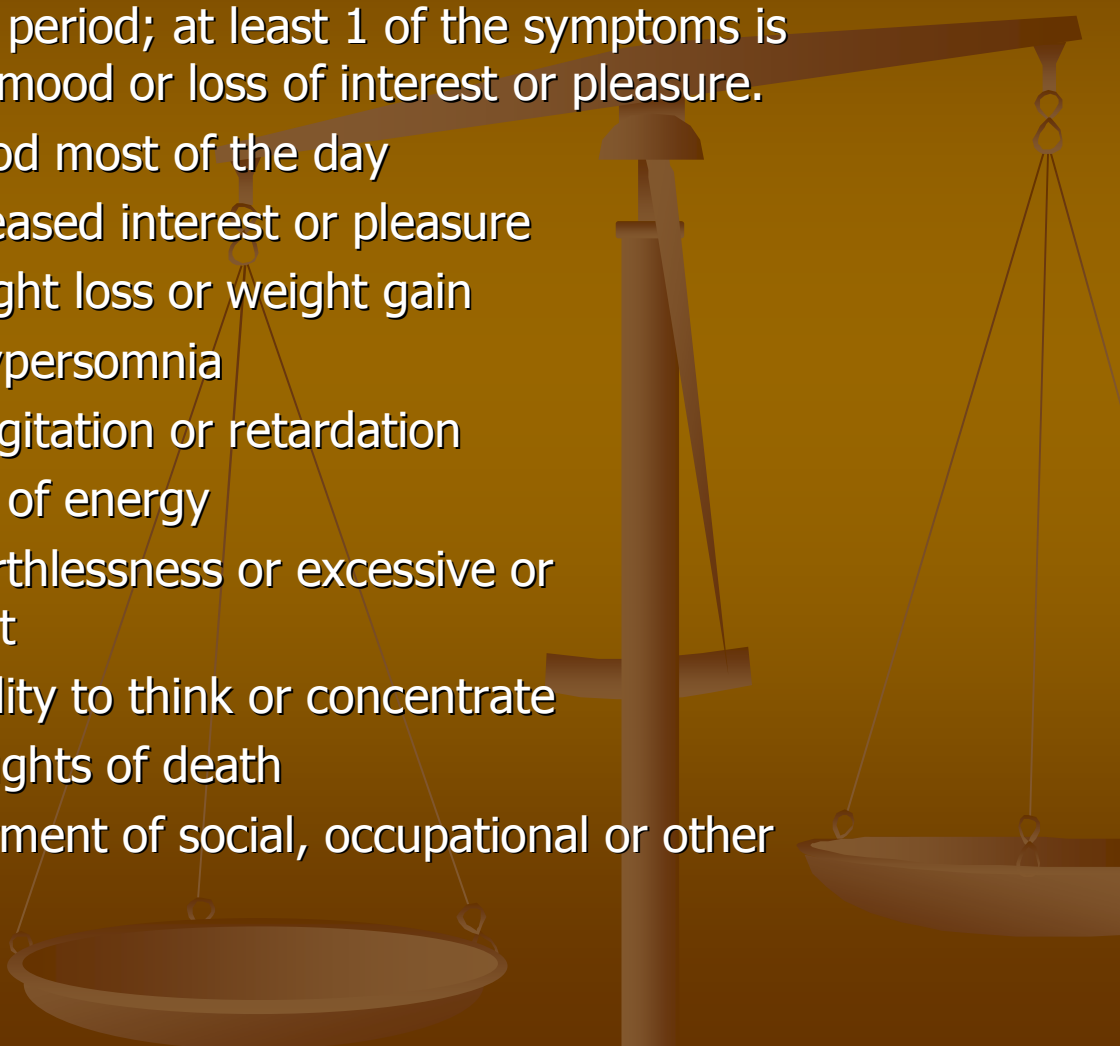
■ Catastrophic Reaction:

- A disruptive emotional behavior where patient is confronted with an unsolvable task. Dissociation between degree of physical impairment and occurrence of catastrophic reaction suggests specific lesion-induced dysfunction.
- **Only L Hemisphere lesions - especially those in regions projecting to the amygdala (insula, temporal, frontal operculum, parietal cortex)**
- Predictor of poststroke depression: 66% of pts with catastrophic reaction develop poststroke depression at 3 months

■ Depression (see next slides):



DSM-IV Criteria For Major Depressive-like Episodes

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- A.
- At least 5 of the following symptoms are present over at least a 2-week period; at least 1 of the symptoms is either depressed mood or loss of interest or pleasure.
1. Depressed mood most of the day
 2. Markedly decreased interest or pleasure
 3. Significant weight loss or weight gain
 4. Insomnia or hypersomnia
 5. Psychomotor agitation or retardation
 6. Fatigue or loss of energy
 7. Feelings of worthlessness or excessive or inappropriate guilt
 8. Diminished ability to think or concentrate
 9. Recurrent thoughts of death
- B. Distress or impairment of social, occupational or other functioning
- C. No bereavement

DSM-IV Criteria For Major Depressive-like Episodes

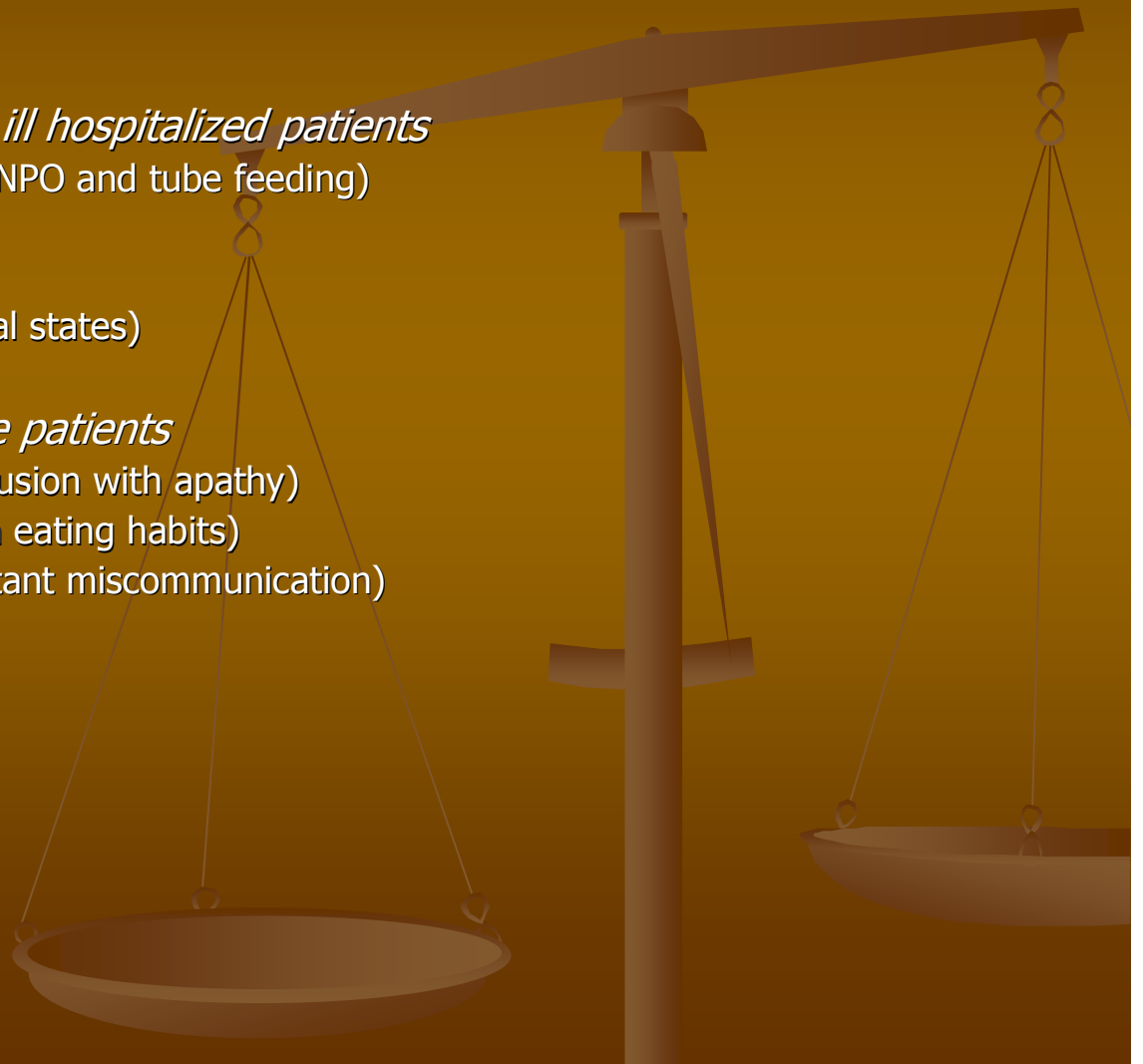
- Persistence of symptoms > 2 weeks: therefore difficult to assess in acute setting
- Overdiagnosis - if too much emphasis given to vegetative signs and cognitive slowing
- Underdiagnosis - if only nonpsychiatric professionals involved
 - '92 study: 50% of patients with questionnaire = post-stroke depression
 - 68% diagnosed by psychiatrists on team
 - 0% diagnosed by team without psychiatrists on team

Diagnostic Confounders of Depression in Stroke

A. INDIRECT

- *Common to many severely ill hospitalized patients*
 - Controlled appetite (e.g., NPO and tube feeding)
 - Frequently awakened
 - Confined to bed
 - Delirium (acute confusional states)

- *Of special concern in stroke patients*
 - Immobility (potential confusion with apathy)
 - Dysphagia (interferes with eating habits)
 - Slurred speech (and resultant miscommunication)
 - Fatigue



Diagnostic Confounders of Depression in Stroke

B. DIRECT

- Aphasia
- Amnesia and cognitive impairment
- Anosognosia
- Aprosody
- Neurological apathy syndromes
 - Isolated abulia/apathia
 - Loss of psychic auto-activation
- Frontal lobe syndrome
 - Klüver-Bucy syndrome
 - Korsakoff's syndrome
- Post-stroke fatigue
- Disorders of emotional expression
- Pseudobulbar Syndrome
- Emotional lability or emotionalism
- Catastrophic reaction
- Dementia



Post-stroke Depression

Incidence

- 0-2 weeks: 6-22%
- 3-4 months: 22-53%
- 1 year: 16-47%
- 2 years: 19%
- 3 years: 9-41%
- 4.9 years: 35%
- 7 years: 19%

- Rehab centers: 30-55%
- Major depression: 56%
- Minor depression: 15%

Risk Factors

- Age: <65
- Gender: female
- History of prior depression
- History of prior stroke
- Psychosocial problems
- High level of education
- Disability
- Living in an institution

Women: High level of education, degree of cognitive impairment, presence of psychiatric antecedents

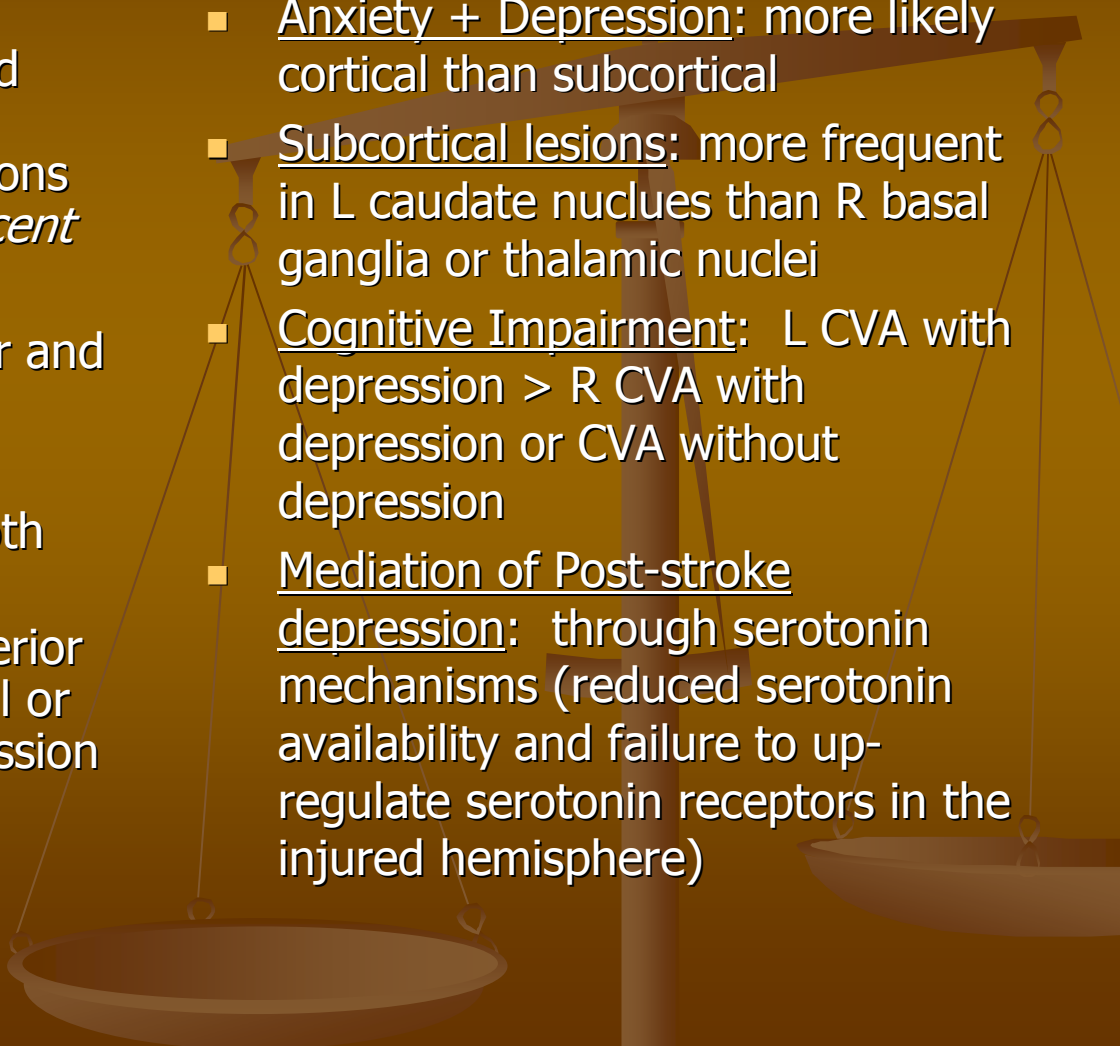
Men: severity of physical impairment

Post-stroke Depression

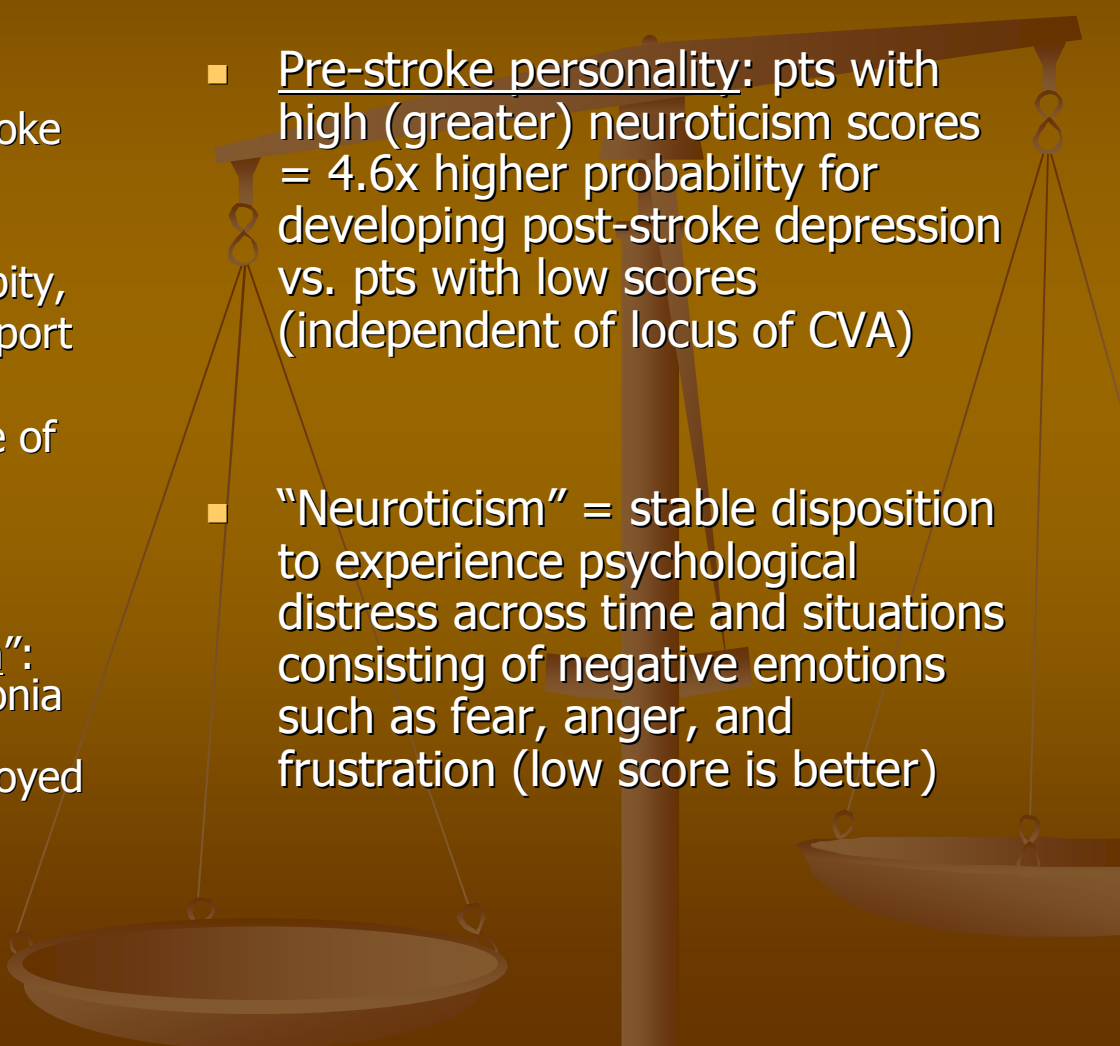


Mortality: patients with post-stroke depression in early phases after a stroke have 3x greater risk of mortality over the following 10 years, in comparison to patients without depression

Neurological Factors in Post-stroke Depression

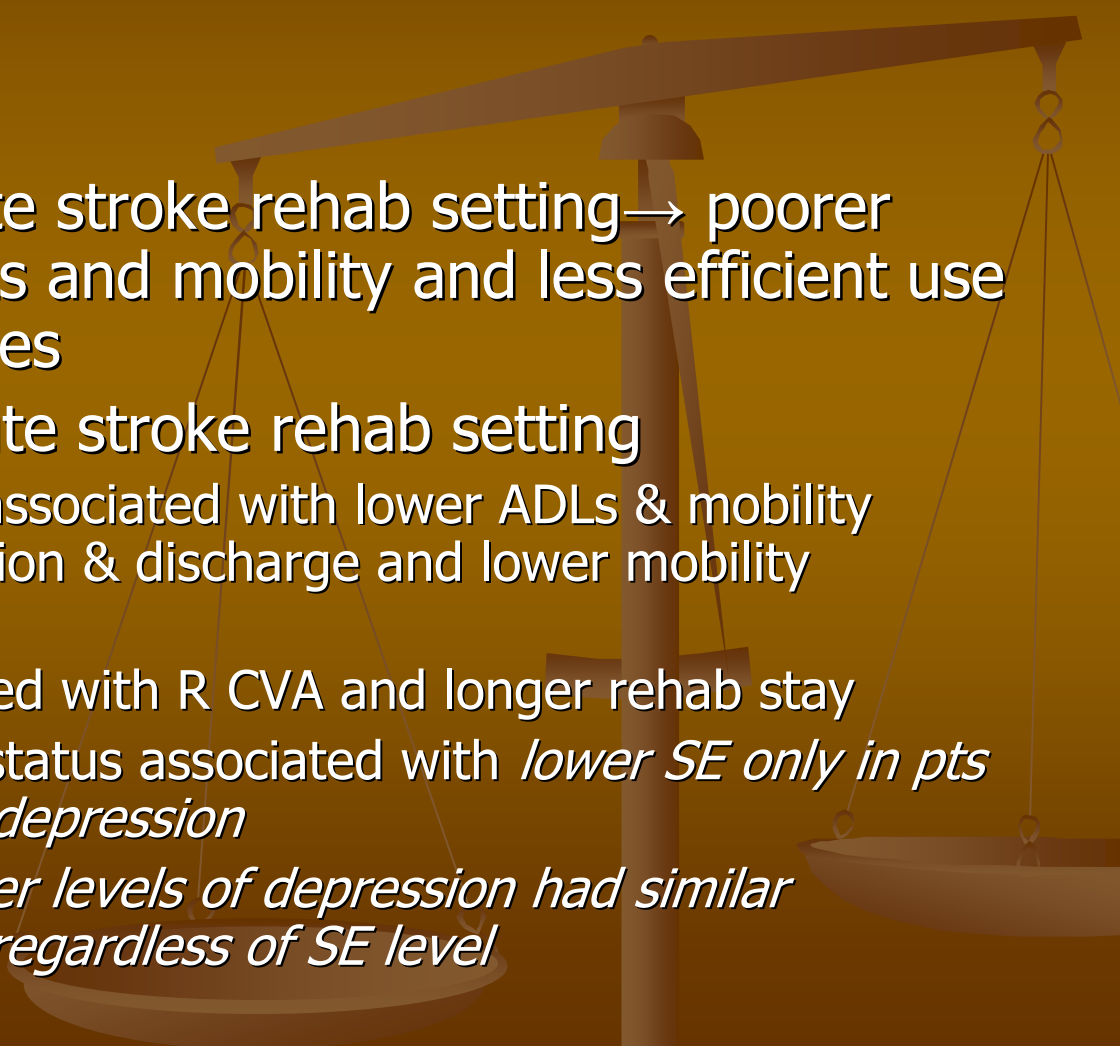
- Robinson's work:
 - frontal, sub-cortical and basal ganglia lesions – especially L frontal lesions (*Note: contested in recent reviews of literature*)
 1. Acute phase: L anterior and basal ganglia lesions
 2. Several months post: anterior location for both hemispheres
 3. 1-2 years post: R posterior lesions (also if personal or family history of depression present)
 - Anxiety + Depression: more likely cortical than subcortical
 - Subcortical lesions: more frequent in L caudate nucleus than R basal ganglia or thalamic nuclei
 - Cognitive Impairment: L CVA with depression > R CVA with depression or CVA without depression
 - Mediation of Post-stroke depression: through serotonin mechanisms (reduced serotonin availability and failure to up-regulate serotonin receptors in the injured hemisphere)
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Psychological Factors in Post-stroke Depression

- “Reactive depression”: adaptive response by stroke (patients complain re: difficulties and the environment, show self-pity, feelings of guilt, often report somatic symptoms that frequently are the source of obsessions & intrusive thoughts)
 - “Non-reactive depression”: main symptom = anhedonia (decreased pleasure or interest in previously enjoyed activities)
 - Pre-stroke personality: pts with high (greater) neuroticism scores = 4.6x higher probability for developing post-stroke depression vs. pts with low scores (independent of locus of CVA)
 - “Neuroticism” = stable disposition to experience psychological distress across time and situations consisting of negative emotions such as fear, anger, and frustration (low score is better)
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Depression & Rehab

■ Rehab Outcomes:

- Depression in acute stroke rehab setting → poorer functioning in ADLs and mobility and less efficient use of therapy resources
 - Self-esteem in acute stroke rehab setting
 - Lower SE ratings associated with lower ADLs & mobility function at admission & discharge and lower mobility efficiency
 - Lower SE associated with R CVA and longer rehab stay
 - Poorer functional status associated with *lower SE only in pts with low levels of depression*
 - Patients with *higher levels of depression had similar functional status, regardless of SE level*
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Depression & Rehab



■ Rehab Treatment:

■ Depression

- Assessment: screening of all stroke patients (NERH has 3 psychologists on inpatient staff); neuropsychological screening as needed for cognition (in collaboration with speech and occupational therapies)
- Treatment: antidepressant (SSRI) meds, education re: CVA, individual & group therapy, cognitive-behavioral therapy (especially in outpatient phase)

■ Self-esteem

- Does poorer functional status result in diminished SE or does diminished SE reduce the capacity to make functional gains?
- Therefore, important to assess and treat SE independent of depression, especially for those patients who are not depressed.

Selected References

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