

Disclosure

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Summaries, recommendations, and claims made hereafter represent the personal opinion of the presenter as based upon an assessment of the salient literature. Unless otherwise explicitly stated, the contents of this presentation do not represent the opinion of either the Veteran's Health Administration or the University of South Florida.

There are no relevant financial relationships to disclose

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Objectives

- Summarize the most common clinical presentation of delirium
- Identify both common and rare contributors to the development of delirium
- Describe the role of structured delirium screening instruments in PALTC
- Review pharmacologic and non-pharmacologic management of delirium

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"...an acute neuropsychiatric disorder... of impaired consciousness... characterized by generalized impairment of cognition, with inattention as its cardinal feature, but also involves a range of noncognitive symptoms affecting motor behavior, sleep-wake cycle, thinking, language, perception, and affect." 1

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Other terms used

- ► Acute...
 - ...brain failure
 - ...brain syndrome
 - ...dementia
 - ...organic psychosis
 - ...organic reaction
 - ...organic syndrome
 - ...reversible psychosis ...secondary psychosis
- ► Cerebral insufficiency
- ► Confusional state
- ► Dysergastic reaction
- ► Encephalopathy
- ► Exogenous psychosis
- ► Infective-exhaustive psychosis
- ► ICU psychosis
- ► Metabolic encephalopathy
- ▶ Oneiric state

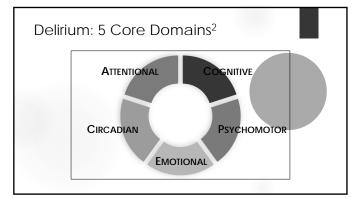
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DSM-5 Diagnostic Criteria

- A disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).
- The disturbance develops over a short period of time (usually hours to a few days), represents a change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.
- An additional disturbance in cognition (e.g., memory deficit, language, visuospatial ability, or perception).
- The disturbances in Criteria A and C are not better explained by another preexisting, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma.
- ▶ There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal (i.e., due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies.

Phenomenology (DRS-98)³ ▶ Core deficits: Attention 97-100% Orientation 76-96% S1 memory 88-92% LT memory 89-96% Sleep-wake 92-97% Thought proc. 54-79% Visuospatial 87-96% Motor 24-94% Language 57-67% ▶ Non-core Perception Delusion 21-31% Affect 43-86%

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Motor subtypes: 1

- ► Hyperactive (Greek phrenitis)
 - ▶ DSM-5: *...hyperactive level of psychomotor activity that may be accompanied by mood lability, agitation, and/or refusal to cooperate with medical care.*
 - $\,\blacktriangleright\,$ Excited catatonia sometimes thought to be an extreme variant
- ► Hypoactive (Greek lethargicus)
 - DSM-5: "...hypoactive level of psychomotor activity that may be accompanied by sluggishness and lethargy that approaches stupor."
 - ▶ Retarded catatonia sometimes thought to be an extreme variant
- Mixed
- ▶ Identified as a subtype in the 1990s, so no fancy Greek phrase
- ▶ DSM-5: *...normal level of psychomotor activity even though attention and awareness are disturbed. Also includes individuals whose activity level rapidly fluctuates."

Consequences in Acute Care ²

- ► Mortality rates 4-65% during the index hospitalization
- ▶ Number of delirious days predicts mortality
- ▶ Implications of persistent cognitive difficulties

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Consequences in PALTC ⁶

- ▶ Delirium may be present in up to 70% of LTC patients
- ► Mean duration of delirious episodes 1.5 +- 1.4 weeks
- ▶ Increased mortality rate
- ▶ Increased fall rates
- ▶ Increased polypharmacy for management
- ▶ Rehospitalization and extended PA LOS

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Risk Factors in PALTC: Cognition 7 MMSE 10+ MMSE < 10 3.4% Baseline Prevalence 33.3% Incidence per 100 person-weeks 6.9

Causes: Acute Care

I Infection / latrogenic

Withdrawal

Acute metabolic (acidosis, alkalosis, electrolytes)

Trauma (closed head injury, CVA)

CNS pathology (seizure, stroke, encephalitis)

H Hypoxia, Hypercarbia

D Deficiencies (thiamine, niacin, B12, folate)

Endocrinopathies (thyroid, glucose, adrenal)

Acute vascular (HTN crisis, arrhythmia)

Toxins/drugs

Heavy metals

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Causes: PALTC



Eyes, ears, other sensory deficits

Low O2 states (MI, CVA, PE)

Infection

R Retention (urine, stool)

Ictal state

U Under-hydration / under-nutrition

М Metabolic (DM, Electrolyte derangement)

Subdural Hematoma

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SIHS: Neuronal Aging



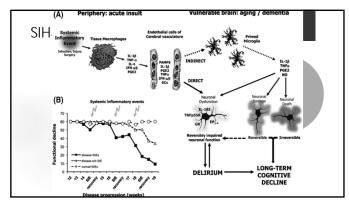
► Changes in proportion of stress-regulating neurotransmitters

► Relative changes in brain blood flow, vascular density

▶ Blood-brain barrier permeability disruption

▶ Neuronal loss





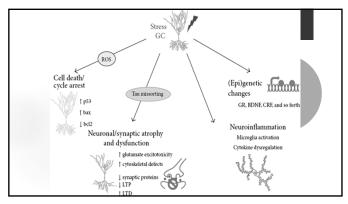
SIHS: Oxidative Stress

- ▶ "Clinical expression of a cerebral metabolic defect"
- Reactive oxygen and nitrogen species cause damage to cell structures, proteins, and DNA
- ► Brain is highly susceptible due to high lipid content (myelin sheaths), high rates of oxidative metabolism, and low antioxidant capacity
- ► May reduce ionic gradients thus resulting in cortical spreading depression
- Influx of sodium, calcium; efflux of potassium results in a dramatic release of glutamate and dopamine

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SIHS: Neuroendocrine

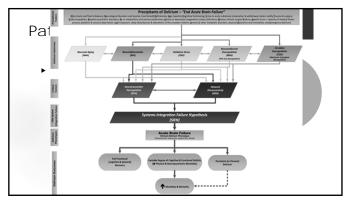
- Glucocorticoids (GC) in the normal stress response mobilize energy stores and suppress nonvital functions in an effort to facilitate adaptation and maintain homeostasis
- Dysregulation of the limbic hypothalamic-pituitaryadrenal axis may cause chronic activation of GC receptors
- ➤ Highest concentration of GC receptors in the brain is in the hippocampus
- Also creates reactive oxygen species and tau hyperphosphorylation

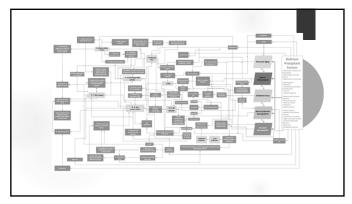


SIHS: Circadian dysregulation

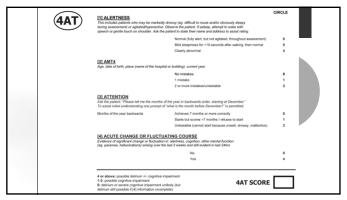
- ▶ Melatonin production impaired by inflammation
- ▶ Deprivation increases both homeostatic and allostatic loads
 - ► Homeostatic: Buildup of metabolic byproducts
 - ► Allostatic: Increased sympathetic tone
- Decreased levels of melatonin metabolites found in hyperactive delirium, elevated levels in hypoactive

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Clinical examination

- ▶ Physical examination
- ▶ Neurologic examination
 - ▶ Includes sensory examination
 - ► Assessing for focal neurologic injury
 - ▶ Prospective trial in 2011 proposed that the presence of >1 primitive release sign pre- and post-operatively may suggest a higher likelihood of progressing to delirium
 - ▶ Myoclonus
 - ▶ Positive: muscular contraction
 - ▶ Negative: interruption of muscular activity

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Management: Environmental



- ▶ Prominent role of ambulation
- ▶ Social engagement
- ► Environmental reorientation
- ▶ Responding to



Management: Pharmacologic

- ▶ Every class associated with benefits and risks
 - ▶ Sedative-hypnotics
 - ▶Benzodiazepines
 - ▶Non-benzodiazepine sleep aids
 - ▶ Antipsychotics

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New Strategies in Pharmacologic Management?

► Alpha 2 agonism

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Prevention of Delirium in PALTC

- ▶ Recognition of delirium on admission
- ▶ Identification of risk factors at every stage
- ▶ Systematic approaches to proactive screening
- ▶ Structured responses to medical evaluation
- ▶ Proactive support to prevent decompensation

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Prehabilitation Expanded

- ► Automated identification algorithms
- ► Structured protocol responses
- ► Hospital Elder Life Program LTC



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Thank you!

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