Changing Faces

Clinical Approach to Personality Change Due to Another Medical Condition

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ACADEMY OF CONSULTATION-LIAISON PSYCHIATRY

Psychiatrists Providing Collaborative Care Bridging Physical and Mental Health

Goals of this symposium

- Introduce medical personality change
- Discuss evaluation of medical personality change
- Describe 3 conditions & treatment options
  - Traumatic brain injury
  - Huntington disease
  - Brain tumors

Medical Personality Change

What it is and how to assess for it

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(Proactive Integration of Mental Healthcare in Medicine)

MEDICINE *of* THE HIGHEST ORDER

UNIVERSITY *of* ROCHESTER MEDICAL CENTER

CLP 2019

Disclosure: Mark Oldham, MD

With respect to the following presentation, there has been no relevant (direct or indirect) financial relationship between the party listed above (and/or spouse/partner) and any for-profit company which could be considered a conflict of interest.

Audience participation

- *Raise your hand if you knew before this session that medical personality change is a billable condition.*
- *Keep it raised if you have ever diagnosed it.*
- *Keep it raised if you have diagnosed it this month.*

Phineas Gage

- https://news.harvard.edu/gazette/story/2015/10/lessons-of-the-brain-the-phineas-gage-story/

(plan to embed this 2-min video)

Outline

1. **Personality vs personality change**
2. Why it matters
3. A clinical approach

What personality is

An “enduring pattern of inner experience and behavior”

1. **Cognitive** appraisal of self, others, & events
2. **Emotional** thermostat
3. **Behavioral** regulation, including impulsivity
4. **Social** functioning

The “big 5” personality traits (OCEAN or CANOE)

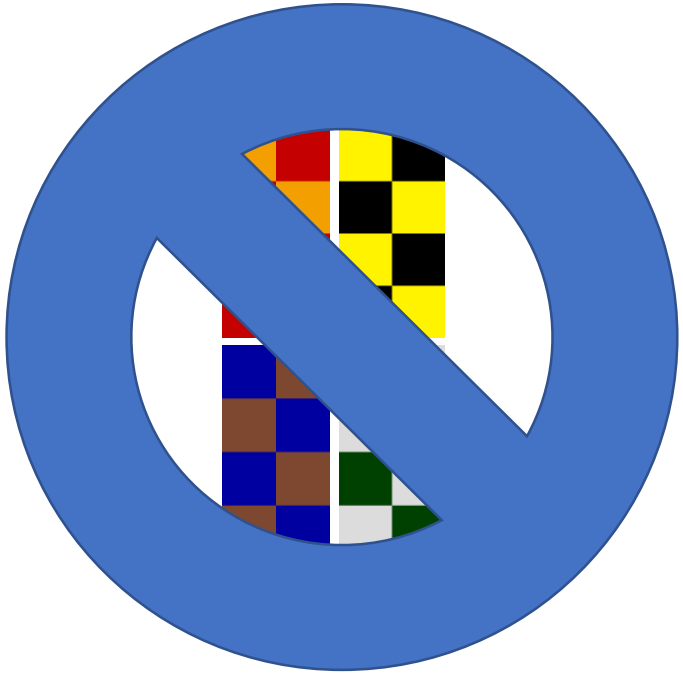
What personality is not

Temperament

Myers-Briggs Type Indicator®

Reflected in Hogwarts houses

- Gryffindor (bravery)
- Hufflepuff (loyalty)
- Ravenclaw (wit)
- Slytherin (cunning)



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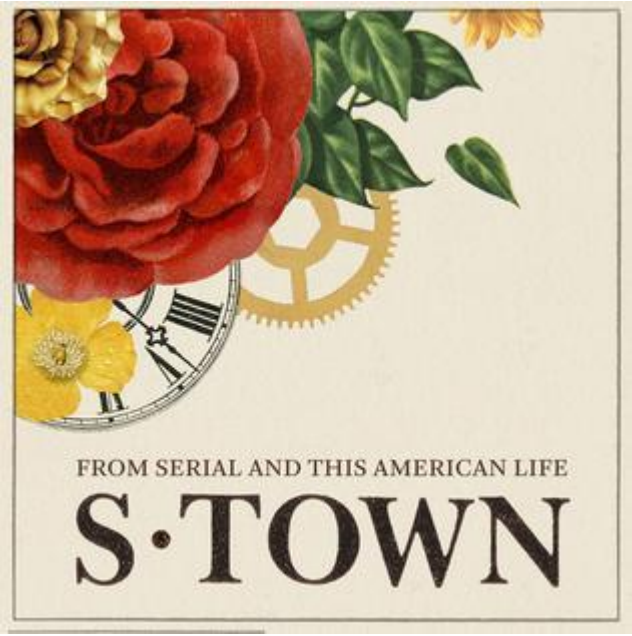
Personality Change Due to Another Medical Condition

- “Persistent” personality disturbance
- Physiologically caused by a medical condition
- Not better explained by another condition
  - Neurocognitive, mood, anxiety, psychotic disorder
  - Psychological adjustment or sequelae of trauma
  - Physiological effects of psychoactive substances
- Not delirium
- Clinically significant distress or impairment

DSM-5 subtypes

- Disinhibited type
- Labile type
- Apathetic type
- Paranoid type
- Aggressive type
- Other type
- Combined type
- Unspecified type

S-Town



- The story about John B. McLemore, a horologist who became increasingly erratic and disinhibited and ultimately ended his own life.
- He practiced fire gilding
  - Mercury & gold are melted into a slurry
  - Spread on & then torched to vaporize mercury

Outline

1. Personality vs. personality change
2. **Why it matters**
3. A clinical approach

Why it matters

- Dysdecorum & poor judgment
- Frequently overlooked *vs* other syndromes
- Patient's often-unwitting mis-reporting of symptoms
- Collateral may not report due to…
  - Embarrassment
  - Difficulty articulating
  - Uncertainty about how to address

The value in diagnosis

- Clarity to patients & families
- Language to understand their experience
- Guide work-up regarding medical conditions
- Suggest treatment options

Outline

1. Personality vs. personality change
2. Why it matters
3. **A clinical approach**

Clinical approach

- Focus on **specific behaviors or events**
  - “Give me an example”
  - “What happens”
- Establish change from baseline
- Ask about domains of highest achievement
  - *e.g.*, work, education

Clinical instruments to consider

Informant-based interviews

- Mild Behavioral Impairment Checklist
- Frontal Behavioral Inventory
- Neuropsychiatric Inventory Questionnaire

Neuropsych assessment

- Frontal Assessment Battery
- Executive function testing
  - *i.e.*, sequencing, organizing, abstracting, planning

Categories of personality change

- **Strategic injuries** (*e.g.*, **TBI**, CVA, FTD)
  - Hypofrontality
  - Cerebellar cognitive-affective syndrome
  - Non-dominant parietal lesions (“right brain syndromes”)
  - Temporal lobes (“viscous personality”)
  - Amygdalar damage (Klüver-Bucy syndrome)
- **Conditions with characteristic CNS involvement**
  - Genetic: **Huntington disease**, Wilson disease
  - Infectious: HIV disease, syphilis
  - Endocrine: thyroid disease
  - Inflammatory: encephalitis (acutely or post-event), rheumatic conditions, MS
  - Toxins: heavy metals, chemotherapy, radiation therapy
  - Post-hypoxic injury
- **Space-occupying lesions** (*e.g.*, neoplasms, NPH, hematoma, hygroma)

Personality Change Due to Traumatic Brain Injury (TBI)

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This presentation will include discussion of off-label or investigational use of products: psychotropic medications in patients after TBI.

Personality change due to TBI

DSM 5 : Neurocognitive disorder due to TBI with a behavioral disturbance classified as Major or Mild.

Characterized by cognitive decline from a previous level of performance in one or more cognitive domains (complex memory, attention, language and perceptual motor) that is due to a medical condition.

Personality change due to TBI

- A persistent personality disturbance that represents a change from the individual's previous characteristic personality pattern.
- Clinically significant change from baseline personality and functioning in presence of a traumatic brain injury
- With descriptive subtypes as discussed earlier

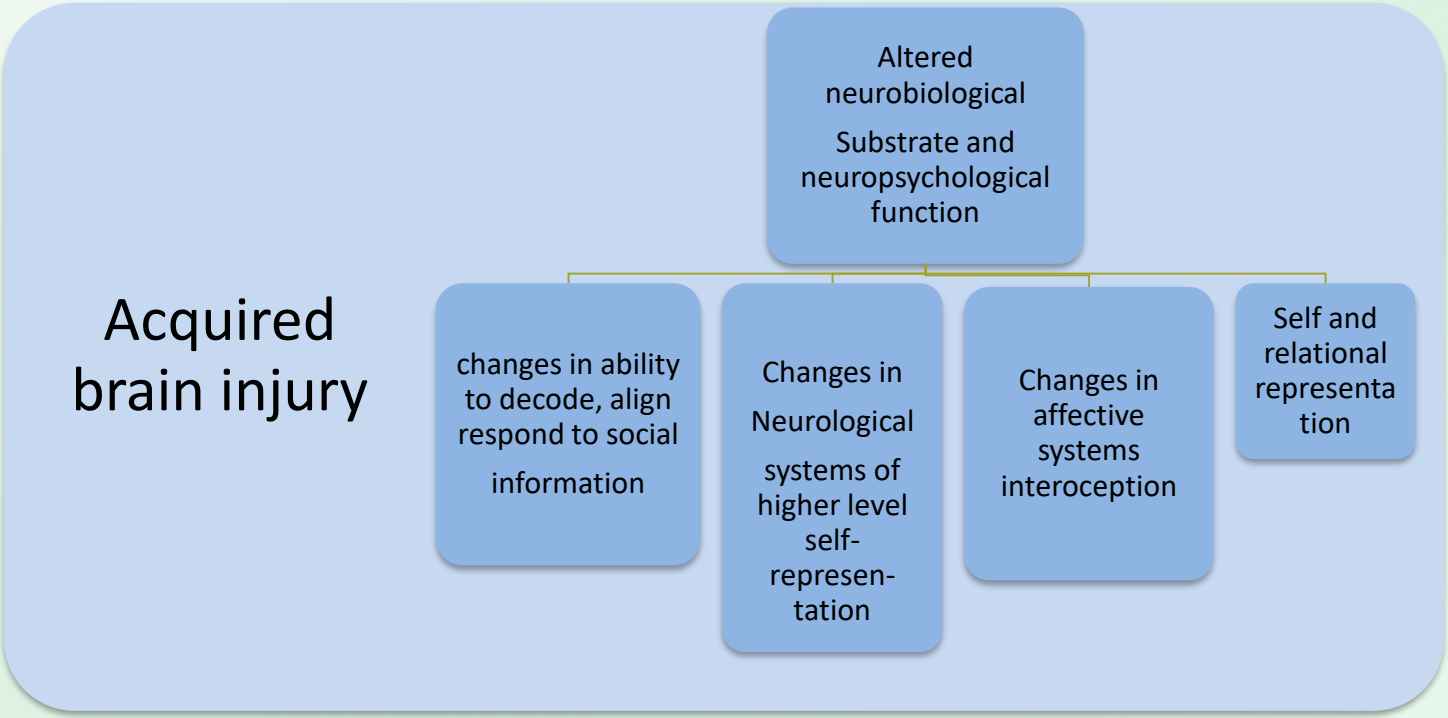
NBC video

https://www.nbcnews.com/video/brain-injury-takes-away-intrinsic-qualities-like-motivation-47909443781

Personality change due to TBI

- Frequency of self reported PC varied from 20–48% in individuals after TBI (Weddell & Wood Brain Inj 2016)
- Commonly associated with frontotemporal injuries
- Time course post-injury (Rao and Lyketsos Psychosomatics 2003)
  - 49% at 3 months
  - 80% at 2 years
  - 65% at 10 years

Personality change due to TBI



Adapted from Biopsychosocial deconstruction of personality traits. Noel Yates, Fergus and Mcgrath 2008

Classification of TBI

Classification	LOC Duration	GCS	PTA Duration
Mild	< 30 min with Normal CT/MRI	13-15	< 24 hr
Moderate	LOC < 6 hr Abnormal CT/MRI	9-12	< 7 days
Severe	> 6 hr with Abnormal CT/MRI	< 9	> 7 days

PTA: Post Traumatic Amnesia.
GCS: Glasgow Coma scale
LOC: loss of consciousness Brain injury Medicine 2nd edition (Zasler)

Posttraumatic amnesia (PTA)

- PTA is the interval from injury until the patient is oriented and able to form and recall new memories (consistently)
- Important index of TBI severity and outcome
- PTA duration in severe TBI may be followed by PC up to 18 m post-injury (Wedell et al 2016; Diaz et al 2014)

Adapted from ref 111: Arciniegas DB, Beresford TP. Neuropsychiatry: an Introductory Approach. Cambridge, UK: Cambridge University Press; 2001

Personality change due to TBI

Dorsal PFC (Dysexecutive)	Orbitofrontal (Disinhibited)	Mesial frontal (Apathetic)
Impaired judgment, poor planning, reduced insight	Stimulus-driven behavior	Diminished spontaneity
Cognitive impersistence	Reduced social insight	Reduced verbal output
Motor programming deficits	Distractibility	Increased response latency
Poor self-care	Emotionally intense	

Ref: Adapted from " The Frontal lobes and neuropsychiatric illness; Pg 15 Salloway and Duffy"

Personality change due to TBI

- **Affectively labile** (rapid, dramatic and pathological shifts in mood) – most common
- **Aggressive**
- **Disinhibited**
- Apathetic
- Paranoid

Biological factors associated with personality change

- Injury severity (including PTA)
- Orbitofrontal location of lesion (ventromedial cortex)
- Lesions of olfactory bulbs and cortices
- Olfactory impairment in TBI may be associated with social & emotional difficulties (Neuman *et al*. *Archives of PMR* 2012)
- Lesions of the orbitofrontal cortex:
  - Deficient emotional recognition, apathy
  - Impulsivity, antisocial behavior
  - Reduced empathy, poor frustration tolerance

Psychosocial factors associated with personality change

- Pre-injury adaptive function
- Emotional distress
- Self-reported depression
- Executive deficit

Max J Neuropsych and Cl Neurosc 2006

Personality change due to TBI

In moderate to severe TBI, degree of self-reported PC is correlated with:
(Weddell & Wood *Brain Inj* 2016)

- General cognitive function
- Executive function
- Olfaction (hyposmia: an index of ventral/frontal damage)
  - *May present as dysgeusia or ageusia*
  - *Post traumatic gustatory loss (frontobasal brain damage)*

  (Ch 23: The influence of head injury on olfactory and gustatory function, from *Handbook of Clinical Neurology* 2019)
- Social emotional behavior

Treating personality change

Non-pharmacological considered first line

- Cognitive and behavioral rehabilitation
  - Education both to patients and to family members
- Mindfulness
- Restorative training: attempts to restore a specific cognitive deficit
- Compensatory training use of memory, assisted devices to compensate for a particular deficit

(Rao, Fann, *et al*. *Psychosomatics* 2009)

Pharmacological management

No medication is FDA-approved to treat TBI-related neurobehavioral symptoms.

Pharmacological management

Amantadine (Ter Mors *Brain Inj* 2019)

- May reduce irritability, aggression and apathy
- Improves initiation, concentration, processing speed
- Dose up to 200 mg/day
- SE: Irritability, ataxia, nausea, seizures, rigidity

Psychostimulants (Plenger et al *Archives of PMR* 1996; Kraus et al *J Clinic Neuropsychiatry* 1997)

- Methylphenidate or mixed amphetamine salts
- Can be useful for managing impulsivity, apathy, abulia, and inattention

Pharmacological management (cont'd)

- **Beta-blockers**
  - Small RCTs: reduces intensity of aggression (Brooke 1992; Greendyke 1986)
- **Carbamazepine** and **oxcarbazepine** (OXC)
  - Open label trials (*e.g.*, Azzouvi et al 1999)
  - RCT of OXC: reduced irritability & aggression (Mattes 2005)
- **Lithium**
  - Promise in *preclinical* studies post-TBI
  - Beneficial effects on neuroinflammation and may protect neurons and enhance functional recovery (Leeds 2014)
- **Gabapentin** and **lamotrigine**
  - Very limited evidence

Pharmacological management (cont'd)

- **Antipsychotics** may affect psychomotor function
  - Typical antipsychotics and benzodiazepines best AVOIDED
  - Delay neurobehavioral recovery, neuroplasticity and repair post TBI (Arciniegas and Silver 2006)
- **Haloperidol**
  - Slows cognitive and motor recovery in animal studies
  - Prolongs duration of post traumatic amnesia
  - Human studies

Antipsychotics after brain injury

- Single vs multiple doses of haloperidol & risperidone studied on functional outcomes post experimental brain trauma
  - Motor/cognitive tasks unaffected after a single dose ($p > 0.05$)
  - Motor/cognitive deficits persisted after daily use of risperidone (4.5 mg/kg) and haloperidol ($p < 0.05$) even after wash out period (Kline, *Neurological Critical Care* 2007)

- Daily administration of haloperidol for 19d impeded cortical recovery but intermittent dosing did not

  (Bao, Zimmerman et al, *J of Neurotrauma May 2019*)

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Clinical Approach to Personality Change Due to Huntington's Disease

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HD and Personality Change

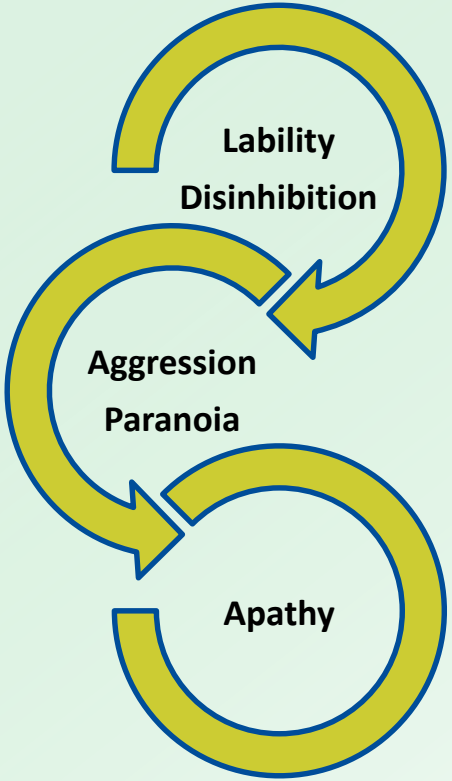
- Famous folk singer Woody Guthrie suffered from HD and experienced significant personality change

- “In it, it has elements of schizophrenia, psychopathy and a psychoneurotic anxiety state, not to mention the mental and personality changes occurring in Huntington’s chorea”

- Led to the creation of the HDSA (Huntington Disease Society of America)

Intro to HD

- Autosomal dominant expansion of CAG
- Symptoms develop around age 30-50
  - First sign in almost 20% of patients (Bates, 2014)
- Why HD?
  - Personality change is common
  - Neurobiological pathways hypothesized
  - Illustrative of other neurocognitive disorders
  - Symptom progression



Nance, M., Paulsen, J. S., Rosenblatt, A., & Wheelock, V. (2011). A Physician's Guide to the Management of Huntington's Disease. *Huntington's Disease Society of America.*

Neuropsychiatric Inventory

Cummings, J. L. (1997). The Neuropsychiatric Inventory: Assessing psychopathology in dementia patients. *Neurology*, 48, S10-S16.

Neuropsychiatric Inventory

Paulsen JS, Ready RE, Hamilton JM, et al. Neuropsychiatric aspects of Huntington's disease. Journal of Neurology, Neurosurgery & Psychiatry 2001;71:310-314.

Mild Behavioral Impairment (MBI)

- Behavioral equivalent of Mild Cognitive Impairment (MCI)
- MBI Checklist tool for assessment
- Facilitates early discovery
- Goal of preventing progression

I CAMP at Sundown

Impulsivity

Comportment Δ

Affective Δ

Motivational deficit

Perceptual Δ/Paranoia

Sundowning

Oldham *et al*. *Crit Care Clinics* 2017.

MBI and MCI

- MBI is twice as likely to progress to Major Neurocognitive Disorder compared to MCI (70% vs 34%)
  (Taragano et al, 2009)
- MBI is a precursor specifically to FTD

Taragano, F. E., et al. (2009). Mild behavioral impairment and risk of dementia. *The Journal of clinical psychiatry*, *70*(4), 584.

Lability and Disinhibition – Nora Belle Tanner

- “The whole town knew about her. She got careless with her appearance. She let herself run down. She walked around over the town, looking and thinking and crying…Us kids would stand around in the house lost in silence, not saying a word for hours.”
- “She’d throw all of our furniture and all of our fixings, our chairs and our tables and our beds and our bookcases and our dressers all around over our whole house while she had one of her bad spells”

Lability

- Irritability among more ubiquitous symptoms
- Disruption of serotonergic innervation
- Nonpharmacological management:
  - Interview caregivers privately
  - Minimize triggers
  - Psychoeducation and psychotherapy
- Pharmacotherapy:
  - Higher dose SSRIs than for depression
  - Can augment with buspirone, mirtazapine, propranolol, or nabilone *(double blind, placebo controlled study looking at NPI as endpoint– Curtis, 2009)*
  - Severe cases may require neuroleptics and long-acting benzodiazepines
  - Elevated risk of sedation and bradykinesia

Disinhibition

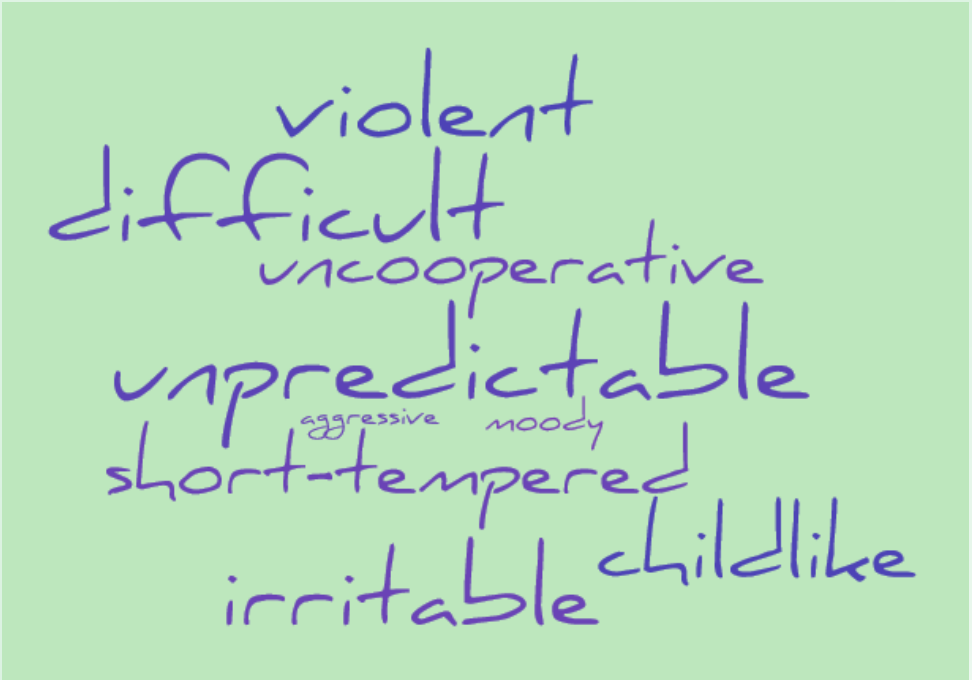
- Hypofrontal syndrome
- Sexual disinhibition especially distressing for caregivers
  - Inappropriate behaviors
  - Perseveration
  - Broadening of attention
- Loss of sympathy/empathy and social dyscomportment
- Manage with antiepileptics and atypical antipsychotics
- Benzodiazepines worsen disinhibition

Aggression and Paranoia – Woody Guthrie

- “Behaving very strangely at times. One afternoon he lost his temper and came charging at Marjorie with a kitchen knife. She screamed, “Woody!” which shocked him back to his senses, after which he apologized profusely. . . but the incident stayed in her mind.”
- “…it turns paranoid, with Woody accusing Marjorie of being an FBI spy simply because she tries to figure out what pains and symptoms he is actually hiding.”
- “Woody’s connections to the Communist Party in America attracted the attention of the FBI.”

Aggression

- Fluctuating jealousy can lead to aggression against spouse
- Subcortical aggression often more severe than in Alzheimer's
- Some evidence that antipsychotics are helpful although literature is inconclusive



Adapted from Shiwach R. Psychopathology in Huntington's disease patients. Acta Psychiatr Scand 1994: 90: 241-246.

Paranoia

- Less common than other personality changes
- Progression from labile personalities
- Need to rule out comorbidities
- Antipsychotics display mixed efficacy
- Minimize changes to the environment and avoid direct confrontation whenever possible

Apathy

- Mid-to-late symptom in 50% of patients with HD in large cohort studies
- Impaired connectivity between prefrontal cortex and basal ganglia
- Nonpharmacologic interventions
  - External prompting; Structured environment/schedule; Finding enjoyable activities
- Pharmacotherapy
  - Often exacerbated by medications
  - Acetyl cholinesterase inhibitors and stimulants demonstrated effectiveness in Alzheimer's and other dementias
  - Avoid atomoxetine or bromocriptine
  - Case series supporting use of amantadine or bupropion
  - Taper neuroleptics or dopamine depleters when possible

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Personality change due to Brain Tumors

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Location, Histology and Epidemiology of Brain Tumors

- **Glioma** – > 50% of adult primary brain tumors, onset ages 40–60
  - Mostly in frontal lobe (supratentorial)
- **Meningioma** – > 20% adult primary brain tumors, women > men
- **Children** more likely to have infratentorial tumors
- **Metastasis** – **10x** more common than primary tumor
  - Cancers commonly metastatic to the brain: melanoma, kidney, breast, lung
  - In areas of rich blood supply (MCA, ACA)

General Considerations about Personality Change due to Brain Tumors

- **Rate of tumor growth** often determines its symptoms
  - Slow growing tumors typically cause **cognitive** problems rather than personality changes (*Lishman WA: Organic Psychiatry: 1997*)
  - Fast-growing tumors tend to cause **personality** changes
  - Very fast growing tumors cause other symptoms including seizures, headaches and other symptoms of increased ICP (diplopia)

Personality change due to Brain Tumors

Location	Common Personality Changes	Explanation of Personality Changes
Frontal Lobe Tumors	Abulia, apathy, depression, psychomotor retardation, increased sleep, disinhibition, irritability, anger	Damage to the frontal lobe affects the following circuits: 1. Dorsolateral circuits (executive function) 2. Orbital frontal circuits (inhibition) 3. Medial frontal circuits (motivation)
Temporal lobe tumors	Memory impairment, seizures, psychotic symptoms possible (visual and auditory hallucinations)	Temporolimibic structures are destroyed including Papez circuit disrupted causing memory impairment, mesial temporal lobe damage causes seizure focus
Parietal lobe tumors/metastases	Silent tumors (although there is right left confusion and dyslexia)	Much of the parietal lobe function is duplicated in other brain regions

Specific Tumors and Classic Syndromes

- Olfactory groove meningiomas represent 10% of intracranial meningiomas.
- They are located in the frontal lobe.
- Psychiatric symptoms include cause depression, apathy, anosmia, psychomotor retardation and incontinence (late stage).
- The treatment is resection.

Specific Tumors and Classic Syndromes (2)

- Crainopharyngiomas affect mostly children from 5 to 15 years old.
- They grow near the pituitary gland.
- Common psychiatric symptoms include hyperphagia and hypersomnia.
- The treatment is resection.

If you are evaluating a patient for depression/personality change, when do you need to think about a brain tumor?

- Change in smell (anosmia) – tumor affecting frontal lobe and olfactory bulb
- Incontinence – also frontal lobe sign
- Seizures and diplopia – often due to increased intracranial pressure
- Age 50 – gliomas most often present in patients 40–60
- Extreme fatigability – may sleep >12 hours/day
- Focal neurological deficits

Case Presentation

- 55-year-old actress previously healthy, presented to the ED after fainting at home
- For the past year, she exhibited the following:
  - Very irritable and “edgy”
  - Weight gain of about 50 lbs
  - Sleeping about 12–18 hours per day
  - Stopped brushing her teeth
  - “Always late”
  - Stopped opening or paying bills
  - Lost 3 jobs (prior to which she had never lost a job)

Case Presentation: Pre-surgical MRI

- **Right frontal lobe meningioma** – meningiomas typically grow slowly but her brain tumor grew over the course of 1–2 years

Combined Type Personality Change due to Brain Tumor

- **Apathetic Type**
  - Psychomotor retardation
  - Emotional dulling
  - Loss of initiative
  - Reduced capacity for mental tasks
- **Labile Type**
  - Jumping from topic to topic
  - Irritable
  - Not organized
  - Use profanities

Case Presentation: Post-surgical MRI

- **MRI shows residual edema and significant reduction in tumor burden**
- Patient no longer endorses depressive symptoms
- She is auditioning again
- She is brushing her teeth and attending to her hygiene
- But she is still irritable per her family

Treatment of Medical Personality Change due to Brain Tumors

- **Corticosteroids**
  - Commonly used to reduced cerebral edema
  - May induce personality changes (mania/psychosis) in a dose-dependent fashion
- **Dopaminergic/activating agents** (*e.g.*, levo-dopa, bupropion, tranylcypromine, stimulants)
  - May be useful for frontal lobe syndromes of apathy/abulia
- **AED mood stabilizers** (e.g., valproic acid, carbamazepine, oxcarbazepine)
  - Can manage aggressive outbursts, especially when there is a seizure focus
  - Many neurosurgeons tend to use levetiracitam first line (Vecht, 2014)
- **Electroconvulsive therapy**
  - Risk of status epilepticus or recurrent seizures with ECT and brain tumors (Fink 1979)
  - May consider seizure prophylaxis (*e.g.* phenytoin in case by Fried & Mann 1988)

Cognitive Dysfunction in Cancer Patients
"Chemo Brain" and "Radiation Brain Injury"

- **Chemotherapy**: although most agents do not cross blood brain barrier patients may still experience *subjective* cognitive impairment
  - Brain cancer chemotherapy has been associated with an apparent 10–20 IQ point drop
  - Intrathecal methotrexate can cause confusion, learning problems and permanent intellectual disability
  - Cognitive impairment most pronounced during treatment with a third experiencing persistent impairment (Vannorsdall 2017)
- **Whole brain radiation**: causes necrosis of cerebral arteries and a series of small, stroke-like infarctions and/or radiation necrosis that has effects on cognitive function
  - These side effects typically happen 6–18 months after treatment

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