The Neuropsychology of Alcoholism

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Neuropsychological Aspects of Substance Use Disorders: Evidence-Based Perspectives

by Daniel N. Allen and Steven Paul Woods

Has great individual chapters on specific substances
Drug and alcohol abuse is a chronic brain disease that causes compulsive substance use despite harmful consequences.

All behavioral experiences lead to physiological changes in the brain, not just psychological change.

Substance abuse leads to physiological brain changes in cognitive, behavioral, and emotional ability.

You ignore these facts at the risk of failure as a clinician.
The most difficult psychiatric patients have neuropsychological deficits, including memory and executive deficits:

- Schizophrenia
- Bipolar Disorder
- Borderline Personality Disorder
- Psychosis
- Severe Depression
- OCD
- Substance Abuse
- ADHD

Example: Cognitive deficits predict functional outcome in Schizophrenia, not psychiatric sxs; containment of the latter does not improve the former
Major 2015 metaanalysis: tested for areas of common gray matter volume increase or decrease across Axis I diagnose

193 studies comprising 15,892 individuals across 6 diverse diagnostic groups (schizophrenia, bipolar disorder, depression, addiction, obsessive-compulsive disorder, and anxiety)

Results: Gray matter loss converged across diagnoses in frontal regions (Anterior cingulate, Insula)

Lower gray matter in this network was associated with poor executive functioning.

Madeleine Goodkind, et al, 2015
Recommendation: Executive Function Groups

- This study reinforces need for all psych. clinics to have a regular Executive Function group for Psychiatric patients.

- Need for teaching behavioral memory techniques, external prosthesis/reminder systems, problem solving strategies.

- All psychologists need to be able to do MoCAs

- Need to do routine MoCAs on psych pts.
Advise to Post Docs: Do not necessarily believe what pt tells you in the hospital; all older patients want to leave hospital and therefore lie.

Many language functions well preserved

Vocabulary continues to increase into old age

But all other abilities may be impaired; only know if you test for deficits

All this applies to patients in clinics with SA issues
Nature of Addiction

- Types of addiction: alcohol, cocaine, heroin, food or porn.
- Are these disorders of **reward**, with drugs hijacking a natural system that is meant to respond to food, sex and friendship.
- They are also a disorder of **learning**, where our brains learn bad **habits** and **responses**.
- Are they a combination of an environmental stimulus and vulnerable **genes**.
- Or perhaps are they an inappropriate response to **stress**, where bad days trigger a relapse to the cigarette, syringe or bottle.
Nature of Addiction

- None of these views are wrong. But none of them are complete, either.
- Addiction is a complex disorder of reward and a disorder of learning.
- It has genetic, epigenetic and environmental influences.
- It is all of that and more.
- Addiction is a display of the brain’s astounding ability to change — neuroplasticity — and it showcases what we know and don’t yet know about how brains adapt to reality. Addiction is example of maladaptive neuroplasticity.
Alcohol

- A central nervous system (CNS) depressant.
- Low doses stimulates neural activity, often increasing social interaction
- Moderate to high doses suppress neural activity
Neuropathology

- **Chronic heavy consumption** leads to **dose-related neurotoxic CNS damage and a pattern of neuropsychological dysfunction**

- There is a **pattern of alcohol-specific damage** known as the **neurotoxicology of essential alcoholism**.

- **Brain atrophy occurs largely due to white matter atrophy**, as well as **neuronal loss in PFC, hypothalamus, and cerebellum in Korsakoff's**

D. Gansler & H. Duncanson, 2014
Risk factors for developing AUD (Alcohol Use Disorder)

- Early adversity
- Genetics (e.g., family history, genotypes): 50% of risk
- Earlier onset of drinking, greater the addiction
- Greater activation of the reward circuit accompanied by blunted executive control systems
- Impulsivity, poor affect regulation, and poor executive function
- Antisocial tendencies

Alcohol Benefits in Normals

- 90+ study: longevity correlated with 1 daily drink

- 2009 prospective study (Anstey): light to moderate drinkers compared with nondrinkers in late life have reduced risk of dementia

  → Protective effect on cardiovascular and cerebrovascular health

- Lothian Scottish Study: reverse causality - bright kids grow up to be bright adults (Etoh: social status effect)

Standard Drink = half oz. of alcohol

Defining the “Standard Drink”

- A standard drink = 14 g ethanol
  - 12 oz of regular beer or cooler (5% alcohol)
  - 5 oz of table wine (12% alcohol)
  - 1.5 oz of hard liquor (40% alcohol, 80 proof)
  - The average person metabolizes about 1 standard drink per hour

BAC of .08 = legally impaired

30% don’t drink;

Next 30%: 1 drink per day;

Top 20%: 2 drinks per day;

Top 10%: 74 alcoholic drinks per week (10 drinks per day) = (70% of the alcohol consumed in any year by everyone)

Median = 3 drinks per week

51% of adults = regular drinkers

Paying the Tab by P. J. Cook, 2007; (NESARC) data
Relative rates of alcohol dependence

Sample average = 3.5%

Hispanics and Blacks have a higher risk for developing alcohol-related liver disease than whites

Moderate & Binge Drinking

- **Moderate drinking** is up to 1 drink per day for women and up to 2 drinks per day for men.

- **Binge Drinking**: (NIAAA) = blood alcohol concentration (BAC) levels to 0.08 g/dL; 4 drinks for women and 5 drinks for men—in about 2 hours.

- **SAMHSA**: 5 or more alcoholic drinks on the same occasion on at least 1 day in the past 30 days.

http://www.samhsa.gov/capt/tools-learning-resources/binge-drinking-terminology-patterns
As you approach .08 to .10 of alcohol in blood, cognitive NP performance significantly decline.

Alcohol Use Disorder (AUD) (Dependence/Abuse)

- Heavy intake – 4 or 5 drinks per day
- Craving
- Social & occupational dysfunction
- Tolerance
- Withdrawal
- Continued use despite negative consequences
Low response to alcohol

- Individuals with low response to alcohol can drink much more alcohol before becoming drunk; low response correlates to becoming alcoholic

- Low response implies you will have more alcohol per session of drinking in your brain; greater chance for long term neuronal damage

- Greater chance of low response with family history of alcoholism
Dependence

Three C’s...

- Compulsion to Drink
- Loss of Control
- Negative Consequences
Neuropathology

- Alcohol does not bind to a specific brain receptor
- It alters functioning of neuronal membranes & of GABA & Glutamate
- Tolerance to CNS effects occurs whether or not an individual meets full criteria for dependence
A. Problematic pattern of alcohol use leading to clinically significant impairment or distress; manifested by at least 2 of 11 symptoms within a 12 month period.
Alcohol Use Disorder

1. Alcohol is often taken in larger amounts or longer period than intended
2. Persistent desire or unsuccessful efforts to cut down or control alcohol use.
3. Great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
4. Craving, or a strong desire or urge to use alcohol.
5. Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home.
Alcohol Use Disorder

6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.

7. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.

8. Recurrent alcohol use in situations in which it is physically hazardous.

9. Alcohol use is continued despite klg of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
Alcohol Use Disorder

10. Tolerance:
   A. Need for markedly increased amounts of alcohol to achieve intoxication or desired effect.
   B. Markedly diminished effect with continued use of same amount of alcohol.

11. Withdrawal:
   A. Characteristic withdrawal syndrome for alcohol
   B. Alcohol (or related substances, such as a benzo) is taken to relieve or avoid withdrawal symptoms
DSM-5: Neurocognitive Disorders

- NCD: The primary clinical deficit is in cognitive function. Only disorders whose core features are cognitive (not Schiz, bipolar)

- Acquired, not developmental: a decline from previous functioning

- Only DSM-5 diagnoses with known pathologies

- “Dementia” is subsumed under major NCD, but not precluded from use in etiological subtypes in which that term is standard
NCD: Cognitive Domains

- **Complex Attention** (Sustained, selective divided)

- **Executive Function** (Planning, decision making, working memory, feedback/error utilization, overriding habits/inhibition, cognitive flexibility)

- **Learning and memory**

- **Language** (expressive, grammar/syntax, receptive)
NCD: Cognitive Domains 2

- **Perceptual-motor** (visual, visoconstructional, perceptual-motor, praxis, gnosis)

- **Social cognition** (recognition of emotions, theory of mind)
DSM-5: Mild Neurocognitive Disorder

1. **Modest Cognitive decline from previous level of performance** in 1 or more cognitive domains
   
   1. Concern of person, informant, or clinician of a mild cognitive decline
   
   2. Modest cognitive impairment on NP testing

2. **Deficits do not interfere with capacity for independence in everyday activities**

3. **Not in context of delirium**

4. **Not explained better by another mental disorder**
DSM-5: Substance/Medication-Induced Major or Mild Neurocognitive Disorder

- A. Criteria met for major or mild NCD
- B. Neurocognitive impairments do not occur during course of a delirium and persist beyond usual duration of intoxication and acute withdrawal
- C. Involved substance or medication & duration and extent of use are capable of producing neurocognitive impairment.
- D. Temporal course of the neurocognitive deficits is consistent with the timing of substance or medication use & abstinence
- E. Not due to another mental disorder.
- Specify if: Persistent (NCD continues to be significant after extended abstinence)
Addictive Drugs in the brain

- All drugs are Trojan horses in the brain

- All addictive chemicals are mimics which look and act like an existing brain neurotransmitter, esp. dopamine (anticipation & reward chemical)

- Increases available amount of dopamine which produces a high; brain responds by reducing dopamine receptors

- Drugs capture reward pathways in the brain which control motivation & compulsion
Substance Abuse: Cognitive Consequences

- Brain damage is a common and potentially severe consequence of long-term, heavy alcohol consumption.

- Even mild-to-moderate drinking can adversely affect cognitive functioning.

- Persistent cognitive impairment, with an established pattern of chronic heavy drinking, can contribute to:
  - poor job performance in adult alcoholics,
  - can interfere with learning and academic achievement in adolescents.

** Cognitive impairment impedes recovery from alcoholism.
Range of Cognitive Impairment

- **Mild (subtle) deficit** = may or may not evidence impairment in daily life
- **Moderate** = more likely show some impairment in daily life
- **Severe** =
  - Wernicke’s Encephalopathy (WE) (acute),
  - Korsakoff’s Disease,
  - Intermediate-duration neurocognitive disorder associated with alcoholism (chronic)
Alcoholism: NP Impairment

- Among 90% of alcoholics without severe impairment,
  
  - 50% have no detectable NP deficits with 3 weeks of abstinence
  
  - other 50% experience mild to moderate NP deficits

- With abstinence:
  
  - mild to moderate deficits resolve in 70-90% of individuals over a 6 month period
  
  - persist in the other 10-30%.

- This persistent condition is known as the intermediate-duration neurocognitive disorder associated with alcoholism (dx = mild NCD)

D. Gansler & H. Duncanson, 2014
Prevalence of NP Impairment

- 33 – 75% of alcoholics entering treatment display neuropsychological deficits, mostly in the mild to moderate range

- Executive Functioning (Problem-solving, Abstract thinking, Concept shifting)
- Learning / Memory (verbal and behavioral)
- Visualspatial ability
- Balance & Gait

Eckardt & Martin, 1986; Meek et al., 1989
Substance Abuse: Neuropsychological Deficits

- Substance abuse causes wide range of neuropsychological deficits
- These NP deficits associated with:
  - Treatment process and outcome.
  - Patient ability to learn and have insight.

- NP results correlate with:
  - Clinician ratings of impairment
  - Therapeutic progress,
  - Relapse
  - Employment status
Importance of Neuropsychological Deficits

- **Neuropsychological Deficits** interfere with **Treatment**:
  - Memory limitations
  - Executive Functional Deficits

- **Psychogenic vs. Neurogenic Interpretation**:
  - Alcoholism and denial are intrinsically linked
  - Denial is *single biggest obstacle in getting treatment for alcoholism*
Deficit blindness (Anosognosia): Need cognitive testing

- Psychological lack of motivation vs. neurological impairment:

- Nature of denial/lack of awareness: **Anosognosia**

- Alcoholics with significant PFC deficits (based on frontal impairment) will exhibit neurogenic denial of deficits (Anosognosia) – they are deficit blind

- As a clinician, the chance of knowing whether a patient has objective cognitive dysfunction based on their verbal report is probably less than 50%, making cognitive assessment based on only an interview worthless. Need a MoCA!
Prevalence of Frontally Based Impairment

NP testing impairments exhibited on:

- Category Test = 50%
  - Abstract thinking
- COWAT = 50%
  - Verbal fluency
- Trails-B = 17%
  - Cognitive flexibility
- Stroop = 12%
  - Cognitive response inhibition

Morgenstern & Bates, 1999
Clinical Recommendations 1

- There is no cure for addiction; it is a brain disorder which involves long term behavioral memory conditioning; but treatment & AA can lead to long-term sobriety

- Need to do some brief cognitive assessment early

- **MoCA**: Montreal Cognitive Assessment

- [http://www.mocatest.org/](http://www.mocatest.org/)

- If significant deficit, may need formal NP testing.
MoCA: Montreal Cognitive Assessment
Neuropsychological testing performance outcome in alcoholics is highly multifactorial in nature.

Alcohol is not the only cause of deficits.
B. Neuropsychiatric Risk Factors:
- Pre-abuse FAS/FAE
- Systemic illnesses and general health
- Head injury
- Psychiatric comorbidity
- Use of other drugs

D. Alcohol History:
- Amount per occasion
- Duration of abusive drinking
- Pattern over lifetime
- Recent amount/duration
- Length of abstinence

C. SES & Education

E. Test Characteristics & Subject Sample

F. Motivation & Expectancies

Neuropsychological Test Performance

A. Age
- Gender
- Family History
- Temperament

Rourke, S. B. and Løberg, T, 1996
Neuromedical Risk Factors

- **Besides dose-related neurotoxic effects of chronic alcohol consumption, NP deficits due to:**
  - Metabolic factors resulting from intoxication and withdrawal syndrome
  - Cerebrovascular disease
  - Hepatic encephalopathy
  - Alcohol-related physical complications (gastritis, esophagitis)
  - Traumatic brain injury (TBI)
Adverse Childhood Experiences Are Common

**Household dysfunction:**

- **Substance abuse** 27%
- Parental sep/divorce 23%
- Mental illness 17%
- Battered mother 13%
- Criminal behavior 6%

**Abuse:**

- Psychological 11%
- Physical 28%
- Sexual 21%

**Neglect:**

- Emotional 15%
- Physical 10%

V. Feletti
Adverse Childhood Experiences vs. Current Smoking
Relationship Between Number of Adverse Childhood Experiences and Smoking Behaviors and Smoking-Related Lung Disease

![Bar chart showing the relationship between ACE score and health problems. The x-axis represents different health conditions: Early smoking initiation, Current smoking, and COPD. The y-axis represents the percent with health problems (%). The chart illustrates the increase in percent with health problems as the ACE score increases from 0 to 4 or more.](image)
ACE Score and Drug Abuse

ACE Score

Percent With Health Problem (%)

0 1 2 3 4 >=5

Ever had a drug problem

Ever addicted to drugs

Ever injected drugs
Newly Dry Brains: Neuropsychological Impairment in Abstinent Alcoholics

**Bad News:** Alcohol is neurotoxic

**Good News:** Abstinence reverses damage

- **Overwhelming evidence:**
  - **Significant pattern of cognitive deficits** in alcoholics/SA: 33-75% with detectable deficits, long before liver, neurological and medical symptoms;
  - **Specific etiology not established**
Predictors of NP Impairment

Age: more important than length of drinking history
Family history, genetics
Age of beginning drinking
Years of drinking, esp. 10 years +
Maintenance drinkers worse than binging
Amount of ETOH per session > frequency
Presence of classic sxss (DTs, blackouts, Sz) are unrelated to NP impairment
Years of ETOH, # of drinks not strongly related to NP impairment
  (.3 to .4 correlation; 20% of variance)

Hawkins, Catalano, & Miller, 1996
ADHD and Chemical Dependency

- Compared with controls, adults with ADHD have a:
  - 3 to 4-fold higher rate of marijuana and cocaine use
  - 3-fold higher rate of alcohol abuse
  - utilize tobacco 40% more

- David Pating, MD, KP CDRP:
  - ADHD Treatment with stimulants reduces CD relapses
Expectations of Adolescents

“It’s sort of unfair to expect (teens) to have adult levels of organizational skills or decision making before their brains are finished being built.

-- Jay Giedd, MD, NIH, 2002
In light of teens’ ongoing development and maturation, some states have introduced graduated driver licensing laws to restrict their actions behind the wheel. Courtesy, with permission: The Allstate Corporation
Drug Use starts early and peaks in Adolescence.

Evidence from surveys:

First Drug Use (number of initiates)

- Infant
- Child
- Teen
- Adult
- Older Adult

The Adolescent Brain, Steve Hanson, OASAS: http://slideplayer.com/slide/5977856/
The Great Pruning, ages 5 to 21: 50% loss of synaptic connections

Brain maturation: from back to front

Images of brain development in healthy youth: ages 5 to 21

Blue represents maturation of brain area

Paul Thompson, PhD, UCLA:
Adolescence Brain Changes

- More vulnerable to neurotoxic events because brain is in last major developmental period
- While overall brain volume stays same after puberty, there are ongoing synaptic refinement & myelination results in reduced gray matter and increased white matter volumes by late adolescence.
- Major synaptic pruning (loss of 50% of synaptic connections in the brain)
- Maturation of frontal and limbic regions
- Increase in myelination (15x faster)
- Dopamine distribution changes (risk taking, reward seeking)
Neuroimaging & NP findings in adolescents

• **Smaller PFC in teens with AUDs** and correlation of smaller volume with greater alcohol consumption

• Hippocampal volume correlate with age at AUD onset (younger age at initiation, smaller volume) and duration (shorter duration, larger volume)

• Both **binge drinkers and marijuana users have poorer white matter integrity**; binge drinkers show even poorer white matter health in several cortical and subcortical regions.

• **Worse NP performance** in attention and information processing, memory, visuospatial functioning, language abilities, and executive functioning compared to nondrinking controls.

Jacobus & Tapert, 2013
Deficit Source

ETOH neurotoxicity:

- Mice with 4 m total access to ETOH—50% HC dendritic loss

- Cerebral atrophy most common finding
  - White matter more affected than gray
  - Frontal and parietal regions most affected

- Disrupts hippocampal connections

- Subcortical atrophy
  - Cerebellum
  - Caudate nucleus
  - Limbic system

JCM Brust, 1993
Deficit Source 2

Nutrition: thiamine deficit

Liver disease: hepatic encephalopathy: lower PIQ

Vascular pathology: reduced Cerebral Blood Flow

Brain trauma

Age of drinking onset: Cloninger’s Type 2 (early onset: symptoms of antisocial personality disorder, more social consequences of drinking and higher sensation-seeking scores, worse prefrontal)

Fetal Alcohol Syndrome

Smoking: COPD
Alcohol and Brain Damage

Alcohol does not kill neurons in adults.

Alcohol damages dendrites and white matter, reducing message traffic between neurons and transmission speed.

Damage is mostly reversible, but neuronal structure is changed in the process.
Chronic Heavy Drinking

- Chronic heavy alcohol consumption is frequently associated with cognitive compromise.

- On average, NP test performance of heavy drinkers falls with the average to low average range.

- Consumption of 3-4 drinks for 7 days a week are associated with reduced cognitive function.

- Heavy drinkers (26 drinks): performance reductions in word-list generations, VS skills, memory & psychomotor speed.
Non-Korsakoff Alcoholism: Mild NCD

- **Non-Korsakoff alcoholism (chronic AUD) cognition**: deficits in problem solving, abstraction, perceptual motor skills

- **Age & maximum quantity & frequency of recent alcohol use predict deficit level**: Mild not Major NCD

- **Younger than 40-45**: better recovery & less severe deficits than older
Non-Korsakoff Alcoholism 2

- No correlation between years of alcoholism and severity of NP deficits

- Quantity and frequency of consumption in 6-month period prior to evaluation are best predictors of NP impairment

- Lower extremity motor deficits are correlated with length of drinking

- 2 systems of brain deficit in alcoholism:
  - Cerebellar-pontine-prefrontal (motoric)
  - Prefrontal-parietal corticocortical (cognitive)
Subacute deficits in non-Korsakoff Alcoholism

- Problem-solving/executive functioning, recent memory, VS ability, and perceptual and motor skills deficits

- Executive function often more resistant to recovery of function.
  - Influenced by family hx of alcoholism and is a predisposing vulnerability to alcoholism
  - Influenced by psychiatric comorbidity,
  - and may be less mediated by dose-effect relationships.

- During intermediate duration phase, recovery in any or all functions, but deficits may be long-standing among 10-30% who display them beyond subacute phase.
Individual differences among Non-Korsakoff Alcoholics

- Alcoholics:
  - uncomplicated types vs
  - those with complex neuromedical risks (hepatic, neurological, nutrition, hypoxemia, alcohol withdrawal, psychiatric)

- Minimal Hepatic encephalopathy (MHE) is:
  - most likely manifestation of cirrhosis from alcoholism
  - most likely to complicate the severity and duration of NP presentation (esp. psychomotor slowing, attention & EF, involving fronto-subcortical networks)
Neuroimaging in “uncomplicated” alcoholics

• Ventricular enlargement, particularly the third ventricle

• Gray and white matter volume loss
  • Prefrontal cortex (PFC), most notably the dorsolateral PFC
  • Hippocampus
  • Cerebellum
  • Corpus callosum

• Associated with longer lifetime alcohol use and poorer NP functioning

• Can partially recover with abstinence through regeneration

• Future relapses had smaller brain volumes than future abstainers in regions of the reward system
Recovery in Non-Korsakoff Alcoholism

- Acute (1-2 weeks), sub-acute (3 weeks to 2 months), and intermediate (2-6 months)

- Recently detoxified: general NP deficits (excluding intact vocabulary) within 1st week or 2 of detoxification

- In general, if deficits are present in non-Korsakoff alcoholism, the same deficits tend to be present and possibly more serious, in Korsakoff alcoholism
Modal Neuropsychological Findings

Big 4 Major Neuropsychological Deficits in AUD:

Executive Functioning
Explicit Memory, esp. nonverbal
Visual Spatial
Motor Ability

Deficits are mild to severe
Executive Functioning

- EF is distinct from Behavioral Memory (more automatic memory processes that have been overlearned by repetition; highly resistant to change).

- EFs allow us to respond flexibly to the environment

- EF is essential for successfully navigating nearly all of our daily activities.

- Impairments in EF thus have serious consequences
Coming Up Next:
Example of Behavioral Memory

- Typewriting skills are behavioral memory
Behavioral Memory
Behavioral Memory:
Remembering how to…

Based on behavioral or motoric practice

- Skills, habits
- Playing a musical instrument, Playing sports
- Riding a bicycle, driving a car
- Playing Chess, bridge
- Interpersonal Skills, psychotherapy
- Recovery, Alcoholics Anonymous
Substance abuse and behavioral memory

- All forms of substance abuse are:
  - heavily based on behavioral memory which is highly resistant to change
  - based on nonconscious learning via repetition of behavior

- Recovery is also primarily based on behavioral memory:
  - Making a behavior “second nature” through constant repetition
  - It is not based on insight (which requires functional EF)
NP in uncomplicated, recently detoxified Alcoholics

- All cognitive domains except IQ are affected.
- Executive dysfunction may be both a determinant and consequence.
- Most recovery occurs during the first month of abstinence;
- 30-80% have cognitive recovery after 1 month of abstinence
- If deficits persist beyond the first month, recovery may take months or years; 20% will not recover fully.
Neuropsychological Deficits

- IQ: At risk for lower performance on NART-estimated IQ

- Lower performance on Perceptual Reasoning & Processing Speed on WAIS4

- Attention: complex attention ↓↓

- Working memory: consistently impaired ↓↓
Neuropsychological Deficits 2

- Processing speed ↓↓: one of the most consistently impaired
- Language: normal
- Visuoperceptual & Visuospatial ↓↓: commonly impaired
- EF dysfunction ↓↓↓↓: categorical thinking, set establishment and maintenance, and decision making deficits
  - flexibility, problem solving, cognitive and behavioral inhibition, search strategy
  - Korsakoff: severe EF deficits
Neuropsychological Deficits 3

**Sensorimotor functions**: ataxia of gait and reduction in manual dexterity are common; fine motor control and grip strength normal;

Sensorimotor: color vision, visual search, **response slowing**, peripheral neuropathy

**Motor Speed**: ↓
Neuropsychological Deficits 4

- **Memory**: common ↓, but not universal; superficial encoding, intrusions, visual memory ↓
  - Recently detoxified: reduced word-list learning & paired associate learning
  - Non-Korsakoff: learning deficits, working memory deficits; subcortical retrieval pattern of memory deficits, with ok retention & recognition
  - Korsakoff: profound encoding & retrieval of episodic memory

- **Procedural/behavioral memory is intact**: AA had it right

- **Abstinence crucial**: 3 weeks to cognitive recovery in younger alcoholics; longer for older
Summary: Neurocognitive Impairments

**Age** is significant variable in reversibility of deficits

**Continuum:** 10% severe; 50-70% some cognitive deficits; 20% normal

- **Specific Deficits; Often Subclinical Levels:**
  - Often significantly inferior to community controls

- Not associated with withdrawal processes

- Equivalent impairment for males/females
Cognitive Reserve: what buffers the impact of brain pathology on cognitive function

- Nun’s Study lead to the concept that some people can tolerate brain damage for a longer time without showing intellectual signs of damage.

- **Cognitive reserve**: difference between amount of brain pathology & actual cognitive function

- CR = more synaptic connections

- CR Benefit: Protective = can have more disease before cognitive decline

- Cost: Once cognitive decline begins, brain decline goes faster (have used up reserve)
Predictors of Cognitive Reserve

- Bigger brain/head circumference
- Higher IQ
- Higher vocabulary level
- Higher education
- Occupational complexity: Work that involves complex thinking and social interaction
- Regular cognitive activity (reading, crossword puzzles)
- Higher literacy
- Social engagement
- Early-age physical activity
Clinical Recommendation 2

- **Dissociation:**
  - Knowledge or ability to describe appropriate behavior does not equal ability to do it appropriately;

- **Implications:** Don’t depend on verbal insight

- Neurologically challenged/impaired, not unmotivated

- Do a MoCA!

- Work on behavioral memory enhancement, a la AA.
State dependent learning / Context dependent memory:
Where is that bottle?

Blackouts = Temporary Amnesia:
BAC level of .2
9 hours to 3 days in length
51% undergraduates = 1x
alcohol suppresses CA1 in hippocampus: encoding deficit

Acute effects on Memory:
Retrieval poorer than Recognition
Retrieval cues aid alcoholics
Alcohol and Neurogenesis

- Moderate alcohol consumption significantly decreases neurogenesis (1400 new stem cells daily) in the adult hippocampus.

- In rats, the number of cells produced in the dentate gyrus of the hippocampus was reduced by nearly 40%.

- Moderate consumption of alcohol for a relatively short period of time can have profound effects on structural plasticity in the adult brain.

Anderson ML et al., 2012
Episodic/factual Memory impaired, esp. visual/nonverbal

Procedural Memory preserved (secret of AA’s success)

Poorer semantic encoding strategies (EF):
  higher intrusions,
  less semantic category usage

Normal forgetting rates

Continuum of memory effects:
  social drinkers to alcoholics (retrieval deficit) to Korsakoff (amnesia), esp. nonverbal memory
Memory and Alcohol: Morris Water Maze

The black, open circle is the old location and the solid red circle is the new location. Control rats (left) rapidly learn to find the new location. Weeks after treatment when alcohol was no longer present, binge treated rats (right) perseverated on the old location. They never found the new location.

Crews, FT, & Nixon, K, 2008
RG: 68 yo, homeless alcoholic

Hx: hosp. s/p seizure, RH-TBI (concave skull)

Score: 17/30*

Executive↓
Language, Attention ↑
Spontaneous Memory ↓: 0**
Normal Recognition: all 5↑**

Conclusion:
Cognitive Disorder due to alcoholism
Clinical Recommendation 3: Memory

- **AA is right**: use *behavioral memory* not insight or talking, esp. in early recovery

- **Behavioral repetition/avoidance** is target

- But also *external reminder systems* to reinforce poor memory

- Download CJV’s Executive Skills & Metacognitive Therapy booklet from website

www.charlesjvellaphd.com
Clinical Recommendation 3: Memory

- **Keep it simple**: Modify written material to make it concise and to the point.
- Paraphrase concepts, use concrete examples, incorporate visual aids, or otherwise present an idea in more than one way.
- If it helps, allow the individual to take notes or at least write down key points for later review and recall.
- **External prosthesis**: Encourage the use of a calendar or planner; if the treatment program includes a daily schedule, make sure a "pocket version" is kept for easy reference.
- Make sure homework assignments are written down.
Clinical Recommendation 3: Memory

- Do not take for granted that something learned in one situation will generalize to another.

- Procedural memory is sensorimotor specific

- Repeat, review, rehearse; repeat, review, rehearse.
Visualspatial Functions

Simple visualspatial functions intact

**Impaired color vision, visual search**

**Visual-spatial organizational functioning impaired**, esp. slowness

**Emotional processing impaired:**
- Emotional cues and facial emotion expression interpretation ↓
Clinical Recommendation 4: EF

- **US Law vs neuroscience:** Knowledge of right & wrong can be present along with total inability to inhibit behavior.

  - **US Law:** no death penalty or life sentence without parole for adolescents (due to lack of EF); but **does not apply to AUD**

- Executive Dysfunction is cause of **neurogenic lack of awareness & loss of inhibition**

- Impairments in Self-Awareness; Introspection

- **Neurologically based Poor Self Monitoring** leads to **inability to understand the consequences of one’s actions.**
Clinical Recommendation 4b: EF

- Caution: when making inferences about motivation based on observed behaviors.

- Non-compliance may not arise from lack of motivation or resistance.

- Unawareness of deficits can be due to executive dysfunction and may not always be due to psychological denial.
Clinical Recommendation 4c: EF

- **Inflexibility** can lead to perseveration i.e. repeating an inappropriate behavior.

- Poor ability to inhibit a response can lead to a broad range of inappropriate behaviors: safety issues; striking out; verbal outbursts; sexual remarks or behaviors; socially inappropriate comments.
Clinical Recommendation 4e: EF

- Let a person know a behavior is inappropriate.

- Do not assume the individual knows and is choosing to do so anyway.

- Provide straightforward feedback about when and where behaviors are appropriate.

- Test prospective memory by having them remember to remind you of something 15 minutes later.
Intoxication: Neurological Condition

Intoxication: alcohol is a CNS depressant with neurocognitive deficits:

- Slurred Speech
- Ataxia
- Neuropsychological Deficits (Memory, Executive deficits)
- Behavioral disinhibition
- Reaction Time ↓

No ability to consent
Stages of Neuropsychological Recovery: **Acute**

Acute deficits: **1 week abstinence:**

Most alcoholics impaired immediately after cessation of drinking

Younger improve over 3-4 weeks to WNL

Older (>40) have slower recovery

Memory and Visual-motor improve markedly

Improvement diminishes or reverses if relapse

Ryan & Butters, 1986
Stages of Neuropsychological Recovery: Short Term

Short term: 2-5 weeks

Younger (<35 age, 5-10 years use) recover to WNL

More chronic history and older age, poorer NP performance:
  Executive, perceptual-motor, LT verbal and nonverbal memory (latter worse), visual-spatial deficits
Stages of Neuropsychological Recovery: **Long Term**

Long Term: 13 months+

**Less conclusive evidence for deficits:**
- Nonverbal learning and memory, esp. Visual memory \downarrow
- Complex memory, esp. encoding strategy \downarrow
- Executive functioning \downarrow

5 years: significant improvement but not as good as controls

Poorer recovery: **TMT-B, Digit Symbol**
Characteristic Behaviors of detoxified Alcoholics: EF

**Executive Functions:**

- impaired judgment
- blunted affect
- poor insight
- social withdrawal
- reduced motivation
- distractibility
- cognitive rigidity
- inattention
- perseveration

**NP test impairment:**

- WCST
- Categories Test
- TMT
- Stroop

**Frontal Lobe Dysfunction**
Characteristic Behaviors of Alcoholics: VS

**Visual Spatial Dysfunction**
- poor sense of direction
- impaired constructional ability
- impaired spatial placement
- impaired drawing ability

**NP test impairment:**
- Rey Complex Figure
- Block Design

**Parietal Lobe Dysfunction**
Visual Impairment on Rey Complex Figure Recall

CJV personal collection
Characteristic Behaviors of Alcoholics: Motor

- impaired timing
- impaired tracking
- impaired balance
- impaired gait
- increased falls

Cerebellar Dysfunction

Gender Differences

Majority of studies: recently detoxified males

Males to Females: 5:1; Men drink 2.5 times more alcohol in lifetime

Women have similar pattern & degree of NP deficits as men

Women metabolize alcohol differently; display NP deficits sooner

Women develop cirrhosis, cardiomyopathy, & peripheral neuropathy sooner

Gender Differences

- Women have less body water than men of similar body weight, so that women achieve higher concentrations of alcohol in the blood after drinking equivalent amounts of alcohol.

- Women have smaller quantities of the enzyme dehydrogenase that breaks down alcohol in the stomach.

- A woman will absorb about 30% more alcohol into her bloodstream than a man of the same weight who has consumed an equal amount.

- More psychiatric comorbidities
Personality Consequences of SA

- **SA produces Disinhibition:**
  - 48-50% of all assaults,
  - 48-65% of homicides,
  - 34% of drownings,
  - 42% of injuries,
  - 47% of young male car crashes;
  - 50% of date rape;
  - 16% of child abuse; burglaries, spousal abuse, suicide attempts

- 50% of successful suicides done under influence of alcohol

- Depression

- Action orientation

- Alexithymia

US Dept Justice: Drug related crime: 1994:
http://www.bjs.gov/content/pub/pdf/DRRC.PDF
Vijayakumar, L; Kumar, MS; Vijayakumar, V, 2011.
Neuroimaging Findings

Recent detoxified: Cerebral atrophy and volume loss, esp. in older Prefrontal sensitive to effects of aging & ETOH

Prolonged abstinence (6 m) causes White Matter increase, decreased 3rd Ventricle volume

PET studies: NP deficits related to decreased glucose metabolism and hypoperfusion in frontal cortex & subcortical structures

SPECT: reduced rCBF

Connectivity networks: Chronic drinking negatively impacts brain connectivity. A positive association between years of drinking and severity of alcohol problems was mediated by reduced Executive Network connectivity.
Neuroimaging

- **Structural MRI:**
  - Smaller cortical gray and white matter volumes & larger ventricles
  - Pronounced in frontal, limbic and cerebellar areas
  - Older show greater volume deficits, esp. frontal
  - Hippocampal volume deficits in adolescent and young adults
  - Younger age of onset and longer duration of use correlates with smaller hippocampal volumes

- **Functional MRI:**
  - Alcoholics use more higher order cognitive processes to do simple tasks

- **Diffusion Tensor Imaging:**
  - Poor WM integrity, esp. corpus callosum
  - Demyelination
Emotional Perception and Alcoholism

- Alcoholic individuals display abnormalities in their perceptions of facial and linguistic emotional stimuli
  - Impairment in emotional facial-expression recognition
  - Deficits in processing affective cues in speech
  - See more fear and less anger in facial expressions
  - Enhanced fear responses
- They perceived the valence of negative facial stimuli to be more neutral as a function of age

Clark, U, et al., Neuropsychology, 2007
Brain in Recovery

- Brain is clearly a dynamic organ

- **Structural brain abnormalities can be partially reversible with sustained sobriety, possibly reflecting remyelination & cellular revoluming:**
  - Increase in glucose utilization post 30 days of sobriety
  - Increase in cortical grey volume, even more WM volume, & hippocampal volume with 30 days
  - Neurogenesis increase with sobriety in rats
  - With 5 years sobriety, increased brain volume
  - Increased frontal and limbic areas with recovery
Wernicke-Korsakoff Syndrome

Carl Wernicke, b Poland 1848
Sergei Korsakoff, b Russia 1853

J.F. Lehmann, Muenchen, in public domain
http://home.kpn.nl/b1beukema/vitaminen.html
Wernicke’s syndrome

In 1881, Carl Wernicke described a neurologic syndrome of acute onset characterized by:
- a global confusional state,
- ataxia,
- ophthalmoplegia (eye muscle weakness),
- nystagmus,
- polyneuropathy in the arms and legs

Thiamine (B1) deficiency + heavy drinking

10% fatal
Korsakoff Syndrome

In 1887 Korsakoff added the characteristic:
- problems in new learning (*anterograde amnesia*)
- as well as the deficits in remembering past events (*retrograde amnesia*),
- and emphasized that these occurred in the *context of clear attention and consciousness*

Some patients tended to *confabulate*, sometimes making up stories or events entirely, but more frequently confusing the *temporal context of actually experienced events*
Wernicke-Korsakoff’s Syndrome

Persistent (Korsakoff’s):

- Normal IQ
- Severe Anterograde and temporally graded retrograde amnesia imposed over typical neurotoxic alcohol effects:
  - defective encoding;
  - severe retrieval deficit;
  - but can recall stores of sexual content
  - better with recognition;
  - but 2 of 3 recalled 9/11/2001 event

Butters & Stuss, 1989
Executive dysfunction; lack of awareness if frontal; blandness, confabulation

Processing speed impaired

28% slight recovery, 26% permanent syndrome
Korsakoff’s Syndrome 1

- Dense Amnesia
- Executive Dysfunction (deficits in planning, decision making, and problem solving; poor WCST, TMT)
- Attention Deficit
- Visuospatial and visual-perceptual deficits are also observed on a variety of concept formation tests that require discrimination and classification of complex visual stimuli
- Abnormally long time to identify visually presented material

Kopelman, 1995
Korsakoff’s Syndrome 2

- **Retrograde amnesia** a typical feature, commonly extending back 25 years or more

- **Memory for autobiographical information as well as knowledge of public events and facts** are affected

- **Memories from childhood and early adulthood** are remembered better than memories from the recent past. (Kopelman 1989; Fama et al 2004)
COGNISTAT: Korsakoff’s

Comparing Korsakoff and non-Korsakoff alcoholics

- **Sudden Onset in Korsakoff’s**

- **Korsakoff patients are impaired on tests of memory, fluency, cognitive flexibility, and perseveration**

- **Non-Korsakoff alcoholics may show some milder frontal system deficits as well**
Korsakoff’s vs. Alcohol induced NCD

- In Korsakoff syndrome patients have a disproportionate disorder of memory.

- No distinct neuropathological basis has been established for alcoholic major NCD.
Neuropsychological and Neurological Deficits Associated with Cirrhosis

- NP deficits are independent of other liver disease symptoms (PSE)
- Deficits esp. in spatial, practic, and perceptual-motor: VS, handwriting, VIQ, memory
- Neuropathology: frontal atrophy, cortical hypoperfusion, neuronal necrosis, demyelination
- EEG: triphasic wave pattern
- Issue of some covariance with alcohol effects
TBI and Alcoholism

- Alcohol is present in:
  - 41% of fatal TBI
  - 7% of falls
  - 60% of MVA
  - >70% of MVA comas
  - 1 person injured every 2 min where alcohol present
Alcohol related Atrophy, esp. Prefrontal

Preexisting EF vulnerabilities in nonalcoholic ACAs

- Nonalcoholic adult offspring of alcoholic individuals showed increased impulsiveness and decreased EF, suggesting weakness of 2 distinct neurobehavioral decision systems.

- Findings support evidence that EF weaknesses may qualify as a suitable endophenotype (genetically based behavior pattern) candidate for alcoholic disease.
Current best theory: Two-systems compromise model

- **Best fits current data**

- **Posits** *alcohol neurotoxic compromise* of
  - Cerebellar-pontine-prefrontal (motoric)
  - Prefrontal-parietal cortico-cortical system (cognitive)
  - producing motor and cognitive compromise, respectively

- **It is based on a pattern of compromise of executive function, visuospatial abilities, and ataxia of gait, along with relative sparing of declarative memory and upper limb speed and strength**
Recovery and White Matter

- **Reversal of atrophy**: abstinence followed by reversal of sulcal and ventricular enlargement

- **Increased cortical grey matter** after 2-4 weeks of sobriety

- **Increase in white matter volume** related to decrease in 3rd ventricular size, 2-7 months
Recovery of Deficits

With longer sobriety, there is significant improvement in brain structure and function.

De-atrophication: dendritic & white matter regrowth

3 weeks as typical recovery

Drinking pre and post age 40

Neuropsychological deficits improve over 4 to 5 years

Neuropsychology of Alcoholism - Parsons et al. 1987
Recovery 2: Reversal of Atrophy

- Cognitive functions and motor coordination improve within 3 or 4 weeks of abstinence; cerebral atrophy reverses after the first few months of sobriety.

- Structural pathology often disappears with long-term abstinence.

- Frontal lobe blood flow continues to increase with abstinence, returning to approximately normal levels within 4 years.

Neuropsychology of Alcoholism - Parsons et al. 1987
Recovery 3: Executive Functioning is slowest to recover

- Skills that require novel, complex, and rapid information processing take longest to recover.

- New verbal learning is among the first to recover.

- Visual-spatial abilities, abstraction, problem solving, and short-term memory, are the slowest to recover.

- May be persistent impairment in these domains, particularly among older alcoholics [over 40]

Neuropsychology of Alcoholism - Parsons et al. 1987
## Chronic substance abuse effects on NP evaluation

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<th>Domain</th>
<th>IQ/Achv</th>
<th>Atten</th>
<th>PSpeed</th>
<th>Lang</th>
<th>Vspatial</th>
<th>Mem</th>
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Vella based on D. Gansler & H. Duncanson, 2014
Increasing evidence that adolescent cannabis use onset results in greater neurocognitive deficits compared with adult onset, especially very early onset.

Regular exposure to exogenous cannabinoids may disrupt healthy neurodevelopment, especially in the PFC and parietal cortices, which underlie higher-order cognitive functioning. This early initiation during the sensitive period of adolescence may place individuals at risk.

Weekly cannabis use before age 18 has been linked with deficits in neuropsychological tasks.
Evidence is building to suggest that regular cannabis use during the teenage or emerging adult years (typically ages 15–25 years) is associated with cognitive deficits.

Two longitudinal studies that followed adolescents with substance use disorders over 8 years found that increased cannabis use during the follow-up period significantly predicted poorer attention and verbal memory.

Early-onset cannabis users exhibit poorer cognitive performance than late-onset users or control subjects.
Cannabis / Marijuana/ THC

- **Intoxication**: widespread Cognitive ↓

- **Acute Deficits** (24 hours):
  - Attention, Executive, immediate recall, RT, time underestimation

- **Long Term Deficits, Chronic Use**:
  - subtle working memory ↓, Digit Symbol ↓, EF ↓,
  - amotivational syndrome?

- **Dependence**: Memory, sustained attention, EF, RT ↓

Schwartz, 1991; I. Grant, 1978
Acute Effects of MJ

- Produces sedation, mood elevation & occasionally hallucinations.

- There is **no dispute** that cannabis produces cognitive impairment during acute intoxication; effects can be shown using driving or flight simulators; esp. memory & learning.

- Primary immediate cognitive effect: adverse effects on learning & memory, etc.

- **Primary adverse effect of acute marijuana use** is diminished psychomotor performance: marijuana has been found to play a significant role in car accidents with 33 percent of drivers arrested at the scene of the accident being positive for marijuana.
Earlier Age of onset: more EF deficits

- **Earlier published reports** using traditional neuropsychological assessment methods typically show a resolution of deficits by 28 days of abstinence.
- Newer neuroimaging research: subtle, long-term effects of cannabis on cognition and brain functioning
- Deficits change as a function of the quantity of cannabis consumed and duration of use.
- Starting between 14–22 years old and stopped by age 22: significantly more cognitive problems at age 27 than their non-using peers
- Users who began smoking before the age of 17 had significant impairments in measures of executive functioning, including abstract reasoning, verbal fluency, and verbal learning and memory

Bolla et al., 2005; Solowij et al., 1995; 2002; Grant et al., 2003; Brook et al., 2008; Pope et al., 2003
MJ and EF: **Acute effects**

**Acute Effects of MJ on EF** (0 to 6 hours after use):

- Disruptions in
  - Attention/concentration ↓↓ in light users
  - Sustained attention ↓↓
  - Learning and memory functions ↓↓
  - Information processing speed ↓↓
  - Planning and decision making ↓↓
  - Inhibition ↓↓
  - Working memory ↓↓ ↓↓

Rebecca D. Crean, et al., 2011
Long-term effects of MJ on EF (3 weeks or longer since last use):

- Normal attention or concentration (28 days to 1 y) vs impaired
- Impaired decision-making and risk-taking
- Impulsivity & inhibition: Normal Stroop, Impaired WCST - intact set shifting and maintenance but impairment in concept formation, planning and sequencing
- Normal Working Memory
- Impaired Verbal Fluency in onset pre-17

Rebecca D. Crean, et al., 2011
Neurologically challenged/Impaired, not unmotivated

Wait 2-3 weeks for cognitive assessment

Neurogenic lack of awareness: Anosognosia - impaired insight into deficits

Don’t depend on insight: insight therapy inappropriate early on, phase appropriate
Clinicians’ misattributions of patients’ behaviors

Verbal skills (previously learned information) remain relatively preserved, so patients appear unimpaired.

Clinicians fail to identify cognitive impairment in at least 40% of patients.

Neuropsychological dysfunction may result in more rule violations in treatment and higher drop out rates.

Low motivation and minimization and denial may be attributable to cognitive deficits.

(Fals-Stewart et al., 1994)
A Neuropsychological Perspective on Treatment Tactics 2

- Think **external prosthetics** for neurological vs. psychological Tx

- Different treatment approaches for cognitively impaired clients: **different levels of care**

- Need for **Mental Status screening**, i.e. MoCA
Clinical Implications of NP perspective

- **Patient – Treatment Matching:**
  - Pre-treatment neuropsychological assessment
  - Target skills to individual patient’s neuropsych profile
  - Reduction in number of skills taught in CBT
  - Behavioral possibly better than cognitive focus
  - Extensive repetition
Cognitively impaired patients might benefit from TBI cognitive rehabilitation in addition to traditional alcohol treatment;

- A la TBI, schizophrenia, cog rehab

Employ errorless learning strategies for simple verbal information (http://www.projectlearnet.org/tutorials/errorless_learning.html)

Encourage elaborative processing (http://adaptivereading.pbworks.com/w/page/3949397/FrontPage)

Provide clear rules for actions and structure tasks/routines
Encourage Physical Exercise: neurogenesis & EF enhancement

Replace use of executive function with procedural memory/prospective memory tactics

Airline Pilot research: Flip procedure books

Implementation Intentions (50%↑): “When situation \( x \) arises, I will perform response \( y \)”

Steps: the how, the when and the where of doing any goal
Emphasize Procedural/behavioral memory:

- AA had it right – walk the walk

Behavioral strategies:

- behavioral repetition
- concrete instructions
- sponsor model
- avoid triggers

Use of reminder strategies

- Calendars, dayminders
- Google calendar (text message reminders)
Evidence-Based Practices Resources:

- NIH:
- SA and MH Services Admin:
  - http://www.nrepp.samhsa.gov/
- National Institute on Alcohol Abuse:
- National Institute on Drug Abuse
- Univ. of Washington Alcohol & Drug Abuse Institute:
  - www.pscyhologicaltreatments.org

Eckardt, MJ & Martin, PR, 1986, Clinical Assessment of Cognition in Alcoholism, Alcoholism: Clinical and Experimental Research, 10-2, 123-127


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